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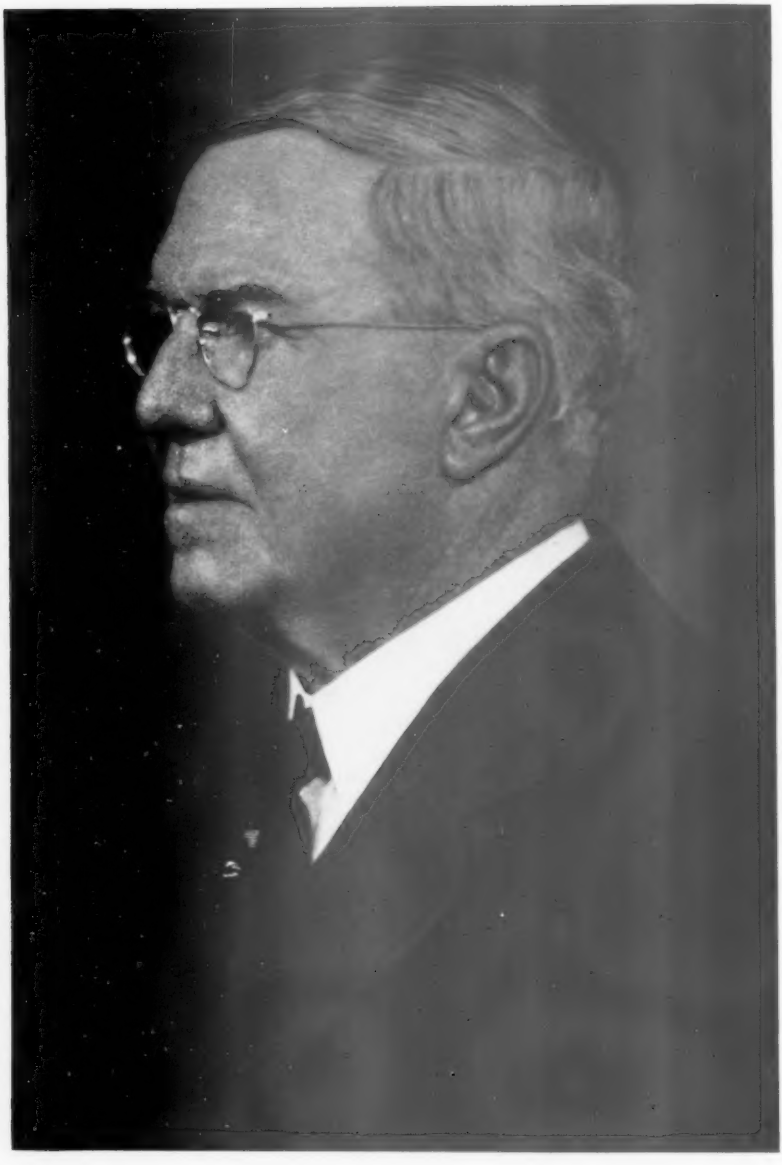
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Richard H. Hutchings

THE PRESIDENT'S ADDRESS.*

By RICHARD H. HUTCHINGS, M. D.

An annual address by the president to inaugurate the proceedings of The American Psychiatric Association is a custom that has become almost a tradition. Last year it came about that I was chosen by you to preside over the activities of the Association as president and so, at the annual convention, it becomes my duty and privilege to present the presidential address. The honor of having been made your president for the twelve-month climaxed a career of more than 40 years in psychiatry in a way that no other distinction could equal. I am conscious of an obligation which cannot be liquidated by any act of mine but must remain an obligation and I your debtor.

We are now about to take part in a parliament of mental hygiene, in a congress of psychiatrists, whose deliberations are fraught with implications of the greatest significance to the public welfare. It is a stimulating thought that throughout the year, in hospitals, laboratories and clinics, earnest workers have been occupied in quiet places with studies, experiments and investigations, the purpose of which was human betterment and the advancement of learning. They are our colleagues who are now assembled to give report of their labors and to invite the comment and appraisal of their peers.

In contrast to these beneficent aims and peaceful labors stands out in bold relief the unsettled state of affairs throughout the world. The wars and threats of wars bring home to us the conviction that the need for mental hygiene in the management of public affairs, for the guidance of those who are charged with responsibility for the conduct of nations, is now as great as it ever was. The evils which afflict the deviated personalities set in high places soon are found to have permeated the body politic and make evident the thin veneer which separates modern man from the tom-tom beating savage of an earlier day. The schizoid mystic who retires to the

* Delivered at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

mountain-top to commune with the elemental forces within his own personality, returns upon occasion with renewed cathexes of anal-sadistic impulse, to bring about reactions which afflict whole nationalities and alter the form and direction of cultural progress.

We cannot, however, with good grace point an accusing finger at our neighbors across the seas until something more is done about the obvious deficiencies in crime prevention and law enforcement in our own large cities. It is being recognized each year that the contribution which psychiatry would be able to make to prevention of delinquency and crime has not been sufficiently tested. Only a beginning has been made; only a few clinics in connection with criminal and children's courts and courts of domestic relations have been organized. The undertaking has not received public support as widely as it deserves. Financial backing is likewise inadequate.

To bring about a better appreciation of the value of the contribution psychiatry is prepared to furnish in this direction, mental hygiene should be stressed to a greater degree in the colleges and law schools. Adequate appropriations will not be forthcoming to finance such clinics until the benefits are more generally realized. It is to the younger people that our efforts at enlightenment should be directed. It is a matter of education, of planning for the future. The conservatism of later years, if it does not prompt overt hostility to newer ideas which aim to modify established forms and practices, at any rate finds it agreeable to continue in the old ways.

The Association has an active and effective committee on "Psychiatry in Medical Education"; I have wondered why the limiting adjective was inserted in this title; I have wondered why it should not be simply psychiatry in education. Perhaps a useful undertaking would be to encourage the assimilation of mental hygiene in its more practical aspects in courses in sociology and psychology wherever those subjects are taught. If courtesy did not prompt you to remain silent I would hear a chorus declare: "It is already being done!" I have only praise for those enlightened instructors who are doing this job to the best of their ability but they are relatively few. They bring their classes to nearby state hospitals for clinical talks and psychiatrists are enabled to give the students the viewpoint we like to have them take into their communities.

When due credit has been given, the number of collegiate and secondary schools giving courses in mental hygiene is relatively small; the number giving acceptable courses is smaller still and those in which the course is taught by a psychiatrist are negligible.

A great deal has been accomplished in the last two decades to promote more extensive and systematic teaching in state hospitals. It should be regarded as one of the prime functions of the institutions. Each, depending on its size, could afford opportunities for several residencies in psychiatry for young men and women who have completed a rotating interneship in a general hospital who would be an asset to the medical staff and able to assume some responsibility in return for the opportunity and maintenance provided; and affiliating or post-graduate courses for nurses and affiliating courses in cooperation with recognized schools of social work. There is a demand for these clinical opportunities that could be adequately met. Every state hospital, even those located in rural districts remote from centers of learning could make this much contribution to the national welfare for the material is there and the personnel for instructors.

For a long time it has seemed to me that as hospital officials and as psychiatrists, a main duty and purpose should be the endeavor to instruct and inform. The more information is given out, the better cooperation will be received from the public. Good will of the people should be deserved and cultivated and must be based upon mutual confidence and understanding.

We must take care that the movement goes on constructively; there is danger that our institutions become exploited for purposes other than that for which they were established; they are threatened by a redundancy of their own growth which in time will impair their standards and promises to bring them down to the level of almshouses.

The number of patients entering the public mental hospitals is increasing more rapidly than provisions are made for their care. This circumstance has given rise to apprehension for the future. Well informed people, however, recognize that the mental hospitals are suffering mainly from over-popularity; they share in the appreciation which has come to general hospitals since the advent of modern surgery and the appearance of the trained nurse. The

prejudice against them is rapidly passing. In states where voluntary applications are authorized, it is found that from 10 to 20 per cent of admissions are accounted for as being in that group. Psychoneuroses and mild depressions constitute the greater number—types that could not have been detained when the legal definition of "insanity" governed the choice of cases to be received. There is another factor, however, of greater relative importance than all others combined.

The age distribution of first admissions is not as it was even 25 years ago. In 1912, in New York State, the number of first admissions to mental hospitals, of senile dementia and psychosis with cerebral arteriosclerosis was 762. Twenty-five years later, 1937, the number had increased to 3,516. I have selected these two categories because they represent disorders which cannot be materially benefited by hospital care and represent psychoses characteristic of advanced age. Of the two, cerebral arteriosclerosis shows a far greater increase among first admissions. In that period of time, senile dementia among first admissions practically doubled while the arteriosclerotic group multiplied more than 14 times. If for purposes of comparison, we eliminate these two groups and give consideration to the other categories of psychoses, it would be seen that the increase in hospital population during the 25-year period is hardly more than can be accounted for by the growing population of the state and voluntary admissions. These figures refer to the State of New York but are representative of the trend. Our institutions promise to become in time vast infirmaries with relatively small departments for younger patients and curable disorders. What can be done with the old people, who are now crowding into our mental hospitals in such numbers as to become a serious administrative problem? It is one of the important developments for the immediate future and we should prepare ourselves to deal with it. The great majority of them are harmless and tractable and need only custodial oversight.

History has given us an example which it may be profitable to study. It pertains to the care of another group, orphan children. Long ago when conditions of life were cruder than now, orphans were "bound out" to be cared for until adulthood was reached. They were in that way provided homes, foster homes of a sort. Some of them may have been good homes but many were not. In

too many instances children were exploited and abuses of various kinds occurred so that the system fell into disrepute.

In an effort to change this situation for the better, there came the era of the orphan asylums, places of refuge from cruelty and neglect. When the term asylum became obnoxious, the name was changed to home but they were still institutions for children supported by charity. Some were better than others, but certain evils are adherent in the system and none offered the environment equal to well ordered family life. The latest phase of orphans' care is that known as selective adoption and we find again the orphan where we saw him first, in a private home. Progress in human affairs seems to advance in great circles. The plan or system that was discarded yesterday as outworn and useless, is tomorrow revived and improved, and seems to be new and desirable. The orphan is again in a private home but one vastly different from that in which he was bound out a century ago.

There is a lesson to be learned from this which may be applied to the mental patient. He, too, has passed through comparable phases in America. From beggary and jails he went to the shelter of the almshouse. It was better than the hunger and cold of the wayside. As culture and humanity advanced the almshouse came into disrepute. Evils connected with the local institutions made them intolerable to an awakened consciousness. Then the states were called upon to assume responsibility and vast hospital systems were brought about.

So well did the states perform this service and so popular did the state hospitals become that with other factors operating, the task is becoming almost too great to be adequately dealt with under the present plan. There is a limit to numbers and congestion beyond which even a great humanitarian undertaking as a state hospital, becomes unworkable. I shall not elaborate upon the reasons for the increasing numbers of dotards in state hospitals. They are known to you as the lengthened span of life, the shifting of population from rural to urban areas, the demand for hospital care as against home care for all kinds of illnesses, and there are yet others.

The movement will go on inevitably, for it is a result of economic changes that are moving irresistibly. We must prepare to meet the situation and deal with it constructively. The tendency for

communities and families to pass on the burden and expense of the aged and unwanted, threatens to convert the state hospitals into vast infirmaries for dotards. The patients needing active treatment will in time occupy a relatively small portion of the huge establishments, and all this will at last result in a burden of expense which may impair the standards now with some difficulty maintained.

Fortunately a way out of this dilemma may be found in resort to selected and supervised boarding homes for harmless, ambulant seniles. Experience in a few states and in foreign countries indicates that such a plan is feasible for a considerable percentage of harmless patients. Where limitations of numbers seem to have been reached, it would be found not to be anything more than limitations in the personnel available for supervision. If one considers the cost of the erection of expensive hospital buildings, their maintenance and repair, heating, lighting and other overhead expenses, it would be evident that boarding patients at a cost of five or six or even seven or eight dollars per week would be greater economy than appears upon first glance.

For a large proportion of state hospital inmates of this class, the need for sequestration does not exist: it may have been necessary for a while but later when they might leave the institution, no one will receive them. The boarding home movement has demonstrated that patients who cannot live in peace and harmony in their own homes or neighborhoods will many times be found adapted to live comfortably in a selected and supervised foster home in another locality.

Successful for centuries in Belgium the boarding out plan was tried in America particularly in Massachusetts, but never with notable success until recently. Its revival is characteristic of what has just been said. Its success in Gheel was due to religious fervor and long established custom. When attempts were made to establish something like it here, the venture was not satisfactory; something was lacking. The Great War permitted us to salvage an adjunct that gives great promise for our aims and methods—the trained psychiatric social worker. It required the stress and anguish of war to bring about the innovation and the schools for the training of a helper who would have been useful in our plans long

before. When the idea of family care was revived after the war, a factor that had been wanting in the early trials was now at hand.

With well trained social workers to select the foster home and the patient who will best fit that *milieu;* who will visit that home and interpret the patient to the family and the family to the patient, a surprisingly large number may live comfortably supervised by a single social worker with psychiatric training. Psychiatric insight and understanding are essential; without them errors of omission and commission would be perpetrated and vexatious situations likely will arise. It is my earnest wish that some way may be found to establish good working relationships between the Association and the national organization of psychiatric social workers for mutual benefit and understanding.

Social workers to be designated as psychiatric, should have met reasonable standards of education and training on a plan approved by The American Psychiatric Association. Until recently the demand for social workers in this field exceeded the number of students graduating from accredited colleges and there was for a time an unfilled want which gave an opportunity for institutions with no psychiatric facilities or properly equipped teachers, to offer courses in psychiatric social work for which they were indifferently prepared. The Association's committee is alert to the evils of this situation and has presented a report to the Council which deserves careful reading and membership cooperation. It is my earnest hope that the standards of education and practical training of psychiatric social workers may speedily become as much a concern of the Association as we have made that of psychiatric nurses.

It is difficult at this time to interpret the significance of newer methods of treatment of schizophrenia. What has been done in it is to be regarded as experimental. Consensus of opinion has not been even approximated. Its advocates seem to be unduly sanguine and there are some others who have not been impressed by the net results of treatment. This divergence of view is wholesome so long as it is not intolerant; the truth will eventually prevail. But regardless of the final issue, in this controversy, we are already in a position to see that some of our preformed opinions of neurophysiology and pathology must be revised; that insulin in massive doses brings about phenomena that will, when interpreted, illumine that obscure boundary which occupied the attention of Storch a

few years ago. That hazy region which is neither psychic nor somatic yet both in one; that region where the physical processes express themselves in terms of emotion, and perhaps in terms of intellect as well, may be available to experimentation. We warmly welcome the opportunity offered by this new approach to mental physiology.

Such is my attempt to interpret prevailing trends in psychiatry in America, but I am one of those who also believes that the future should be considered in relationship or in contrast to what has gone before. I shall, therefore, devote a few moments to the consideration of certain historical aspects which I hope will interest you.

I have thought that the recognition of general paresis was more a misfortune for the future of psychiatry than a benefit for the reason that it came prematurely. Science of that day was not prepared to make any use of its recognition. Eighty years were to pass before its nature was understood or any effective treatment devised. In the meanwhile, it was taken as a paradigm of mental disease but deductions derived from it pointed in the wrong direction for advancement.

About that time and for several decades later an acrimonious dispute was carried on over the question of, to which authority should the care of mental patients be entrusted. There were many who held that the individuals who were recognized as lunatics in the light of the knowledge of that day, were at least many of them strong and able-bodied and their illness was not of the flesh. Therefore, the trouble, whatever its nature, did not fall within the domain of the physician. Some there were who held that because ideas of sin and self-reproach often were expressed and notions of demoniacal possessions had not disappeared, it was clearly the business of the clergyman; the soul was afflicted and no disease was present. Another smaller group saw in lunacy, disorder of the mind and advocated the claims of the pedagogue to have charge of the afflicted ones. These views were successfully combated by physicians but were revived from time to time until Dr. John P. Gray established at the Utica Asylum the first laboratory in connection with a mental hospital (1870) and appointed the first full time pathologist, Dr. Edward R. Hun. The latter

published so much in support of brain pathology of one kind or another that the controversy soon ended.

When paresis was first described it was a high triumph for the medical protagonists. Here was a mental disorder as plain as a pikestaff, with characteristic pathology, *ergo* insanity is nothing more than the manifestation of diseases of the brain. No half-truth was ever more generally received by men of science or given greater publicity. What followed was that general paresis became the paradigm of the group of diseases called in those days insanity. It was not even universally recognized to be a group of diseases but was thought by many to be one morbid process with protean manifestations. In a determined attempt to align all of the then-known types into groups with specific brain lesions, the field of research was the laboratory to the exclusion of the clinic and bedside. When pathology could not be demonstrated, the theory remained unshaken. It was the inefficient microscope, the lack of discriminating staining methods and reagents that were blamed. In the meanwhile psychiatry was on a treadmill, looking to physics and chemistry to solve its dilemma.

Another development in science of this period gave benediction to this quest for lesions. It was the rise of the new science of pathology. More and more diseases were yielding their secrets to the laboratory workers; more and more symptoms were being related to organic changes in the affected organs. It was the era made notable by Virchow and his cellular pathology, further developed and brought into general acceptance through the contributions of Traube, Cohnheim and contemporaries. Pressing close behind these startling developments came the epoch-making work of Louis Pasteur and the rise of the science of bacteriology, the discoveries of Koch and his contemporaries. The medical world was agog. Each decade produced some startling new truth to shed light upon the nature of disease and all the new knowledge stressed the importance of cellular pathology. No one could now conceive of dysfunction except it resulted from cellular changes.

Psychiatry could not resist. Indeed it made no attempt to resist the sweep of opinion which attributed all of its manifestations to cellular changes and which assigned the locus to the cerebral cortex. When hysteria obtruded itself to sound a sour note in this harmony, psychiatry disowned it. Its baffling array of symptoms

present today, gone tomorrow, only to return, exasperated psychiatry as well as general medicine. It was not included in the classification of mental diseases. It was a bastard that no medical discipline was willing to father. For this sin of omission and exclusion, psychiatry suffered long and painfully.

During this fruitful period covering the last four decades of the 19th century, medicine and surgery pushed on to new and thrilling conquests. New sciences were set apart—bacteriology, epidemiology, immunology, and others, but psychiatry marked time. The great depression through which we have passed or are still passing, is as nothing to the gloom and pessimism which enshrouded psychiatry like a pall until the end of the century. Interest in the subject on the part of the scientific world almost completely died out. Hardly any attempt had yet been made to teach it. At the meeting of the Association for its semi-centennial anniversary, the membership was severely castigated for its failure to keep abreast of its sister sciences and show better results. Dr. Weir Mitchell meant his criticism to be constructive and he was reluctant to accept the invitation to speak before the Association, and when he was finally persuaded, his remarks were deeply resented by some members. They pointed out that he himself had no worthwhile program to proffer, but only scolded. His omission to come forward with a sound and constructive program is worthy of remark. He was as blind as the psychriatrists for he had given more study to the subject of hysteria than any other American physician. He had devised a method of treatment that gave results better than any other up to that time. For all that, he did not recognize that many of the cases reported by him as neurasthenia can now, with our better understanding of the subject be recognized from his own descriptions to be examples of minor hysteria. His cures were the result of persuasion and suggestion, Babinski's method, as can be seen in perspective, whereas he attributed them to rest and forced feeding; and he did not see that hysteria was the open sesame to the comprehension of the mechanisms of the psychoses.

Dr. Weir Mitchell's Philadelphia address had a remote result which proved in the end to be in the highest degree beneficial for, smarting under the sting of his rebuke, several research institutions were established or reorganized and improved, but it was still along the same old lines that had yielded so little in the past. More

winnowing of old straw, but the stimulus to research drew better equipped men to the psychiatric field in America.

In the midst of all this bewildered activity and just after the turn of the century, it came somewhat as a shock to hear a quiet voice saying: "You are not doing all that can be done with the opportunities within reach of your hands; think less about disease processes and study the man himself as a biological unit, his equipment and environment." And lo, psychobiology was conceived to be born later and christened later still.

Psychoanalysis came at a fortunate time to supplement and objectify the conceptions of psychobiology. It shed new and much needed light upon the exact nature of environmental disharmonies and inner conflicts, which heretofore had been perceived but vaguely. It supplied a minute anatomy and a physiology of the personality, which had now become so important. It was not at first primarily psychiatry for it was in the beginning concerned with hysteria and with more than hysteria for it brought into being a new and dynamic conception of the mental systems, which in turn illumined psychiatry. The new psychology was to be the philosophers' stone, which transmuted the superstitions, the doubts and misconceptions of the past into the gold of understanding and knowledge. As the years pass by, more and more will the names of Sigmund Freud and Adolf Meyer be honored in psychiatry. It is fortunate, too, that it was a man with state hospital training who brought psychoanalysis from the Old World and introduced it as an adjunct to psychiatry. It was fortunate that he possessed the courage to valiantly and untiringly champion its right to recognition as belonging here when it might so easily have miscarried into some cult of mysticism or into speculative and abstract channels. I am glad of the opportunity to make an expression of the obligation we owe to our distinguished colleague, Dr. A. A. Brill.

History informs us that every epoch-making truth when first enunciated was damned as heresy and had to make its way against the determined opposition of leaders, who should have been the first to acclaim it. The procedure has always been much the same; first, the new truth was coldly ignored; when that attitude could no longer be maintained came the flat denial that it was true and warnings against being deceived by false teachings. When these measures failed to halt its advocates and new adherents aligned

themselves under its banner, the last recourse was a concerted and furious attack upon the man himself who was berated, vilified and misrepresented. That had been the fate of William Harvey, Edward Jenner, D. Hack Tuke and Joseph Lister, to mention but a few in our own profession and in English speaking lands. Each of these pioneers was made to feel the sting of ridicule and condemnation by contemporaries too bigoted to give serious study to the new teachings, but condemned without understanding. Instructors in the Glasgow Infirmary, Lister's own colleagues, warned students not to go into Lister's wards for he was hopelessly unorthodox. The London surgeons for years ignored him and his history making conquest of sepsis and, when forced by public sentiment to test the value of phenol for its control, reported that even though they had made improvements on his technique it was still useless.

It was inevitable that Freud should meet opposition; it was inevitable that his work should be first ignored by his contemporaries ensconced in their comfortable academic chairs; it was inevitable that when his work and the conclusions derived from it could no longer be overlooked that he should be assailed with a storm of abuse and vilification. The world of sciences was running true to form and tradition.

There are striking similarities in two great movements of the 19th century, which gave impetus and direction to scientific thought and cleared the way for notable progress in several fields. I refer to the work of Charles Darwin and Sigmund Freud. The similarity begins with the characters of the men themselves. A description of one applies strikingly well to the other. Each was quiet, modest and unassuming; each was devoted to study and strongly imbued with the love for truth and each presented his views only after a long period of reflection and checking and rechecking of his conclusions; each disturbed intellectual complacency and stagnation of thought by stimulating fruitful activity along collateral lines; each was subjected to opposition which went beyond cool logic and sound criticism and degenerated into personal abuse; each made no reply to his critics except to bring forward additional studies tending to confirm or to modify his formerly expressed views. It is noteworthy that each was maligned and ridiculed by

men who did not take the trouble to study and understand the theories they so savagely attacked.

The authorities of Trinity College, Cambridge, refused to allow a copy of "The Origin of Species" to be placed in the college library and at Oxford it was seriously proposed to endow a chair for the purpose of combating the errors of Darwin. In 1872 when his name was proposed for membership in the French Academy, less than one-third of the votes cast were favorable and this—13 years after the appearance of "The Origin of Species."

Looked at from the perspective of 30 years, it can be seen that the mortal offense of Freud was not that he had the presumption to investigate a subject which theretofore had been shrouded in ignorance and superstition—the sexual instinct. The offense he gave to contemporaries was that he disturbed the complacency of the leaders of thought of that day, convicted them of having taught half-truths or error and required them to execute an "about face" and assume a new stance. This they were incapable of doing. They had been identified too long and too prominently with the old teachings. Their minds were closed to the new ideas. Their attitude was very human. They were incapable of seeing truth in any theory that was novel and disturbing. In this, they were no different from the generations which had preceded them.

The major conflict has now happily come to its inevitable conclusion. Only an occasional reverberation indicates that there are here and there a few who have not been silenced. The younger men and others in their middle years who constitute the great body of psychiatrists of today, make daily use of its concepts and phraseology with little realization of the conflict that was waged two decades and more ago when the elders of that time, with a few brilliant exceptions, were either skeptics, scoffers or bitter opponents of all that pertained to psychoanalysis. Who were these elders? They were men who were in much the same position as were the London surgeons when called upon to abandon the theories and practices of a lifetime and learn at the feet of a young man from the "provinces." Even this very rostrum reverberated to the thunderous denunciation of all that pertained to the new and outlandish teachings of Freud.

After 25 years those doughty warriors have nearly all gone to their rewards but not before it could be perceived that the psycho-

logical system of Freud was being absorbed into psychiatry to a degree that would soon make distinction only academic. One needs only to examine the newer textbooks to see that what is becoming known as American psychiatry is tinctured with it.

Psychobiology is a development of the utmost significance in mental medicine. With it psychoanalysis holds much in common. Each is living, dynamic; each is progressive yet incomplete. The essence of progress is modification, as is also adaptation. Nothing is wholly true. To say that something is true is to imply that it is static and, in psychiatry at least, sterile. The psychiatry of the future will be dynamic for it will be human; there will be of it much psychobiology, much that is derived from psychoanalysis; there will be of it much that is now unpredictable but it will not be static. It it were it would not reflect human emotions, strivings and frustrations and so would not be psychiatry.

RICHARD HENRY HUTCHINGS, M. D.

PRESIDENT, 1938-1939.

A BIOGRAPHICAL SKETCH,

BY CLARENCE O. CHENEY.

It is much more pleasant to undertake the writing of a biographical sketch of a friend who is still living than to attempt to give an account of one who has passed away. It is particularly gratifying to be given this opportunity of writing a brief account of the life and works of a friend who has just completed a most successful year as President of The American Psychiatric Association.

Richard Henry Hutchings was born in Clinton, Ga., August 28, 1869. He was named after his father whose family had been pioneers in Georgia, having immigrated from Virginia some time after the Revolution. His mother, Cornelia Greaves, had been born in the same county, the daughter of Joseph Greaves, a Tennesseean, and of Mary Shorter whose family were prominent in the affairs of Georgia and Alabama. The estates of the father in Clinton had been devastated by the ravages of the Northerners during the Civil War, and a few weeks after the birth of Richard Henry the family moved to Macon, Ga. The father died when Richard was five years of age. Richard Henry was the youngest of six children; two brothers died in infancy and he had the care and devotion not only of his parents but of three older sisters. From his eldest sister he acquired his love for flowers which he still retains. From an early age he attended a small prviate school in Macon and there was introduced to the classics by his second sister who was one of the teachers. At the age of 14 he entered Middle Georgia Military School at Milledgeville, a junior college, and was graduated in 1887 at the age of 18. After a year in Georgia University preparing for the study of medicine, he entered the Bellevue Medical College and received his degree of Doctor of Medicine in 1891.

Dr. Hutchings has told how by chance he was offered a substitute interneship at the Almshouse Hospital on Blackwell's Island during

the summer between the junior and senior years in the medical college. This led to his accepting an interneship in the same hospital after he was graduated, and this later in turn led to his appointment as a physician to the City Asylum on Ward's Island in April, 1892, by Dr. Macy. It was thus that Dr. Hutchings entered psychiatry. After a few weeks at Ward's Island Dr. P. M. Wise, then superintendent of the new State Hospital at Ogdensburg, N. Y., came looking for a promising assistant. Dr. Macy recommended his recently appointed assistant and Dr. Hutchings went to Ogdensburg to become a member of the staff of the St. Lawrence State Hospital, May 24, 1892. He rapidly passed by promotion through the various grades on the medical staff and in September, 1903, was made superintendent at the age of 34. To our knowledge at no time before or since in the New York State Hospital Service has a superintendent been appointed at as early an age.

Dr. Hutchings' scientific interest and curiosity were evidenced in his investigation in 1903 of the cause of a typhoid epidemic at the hospital when he demonstrated for the first time on record that typhoid bacilli could be carried in ice. The hospital discontinued thereafter the use of ice from the St. Lawrence River and established an uncontaminated source with undoubted saving of many lives of patients and personnel. Under Dr. Hutchings' superintendency the hospital grew in number of patients and in its efficiency and facilities. He early recognized the advantages of voluntary admissions and the rate of such admissions at St. Lawrence State Hospital exceeded that of most, if not all, of the other state hospitals. The first clinic held regularly at a New York state hospital for community advice and treatment was established in 1909 by Dr. Hutchings at Ogdensburg. In view of the present recognition of the important service the state hospital can give to persons in the community needing advice and treatment, it is interesting to learn that some doubt was expressed at that time whether a state hospital should undertake to give advice and treatment to persons not committed to the hospital.

Dr. Hutchings was also a pioneer in organizing in 1908 habit training and recreation for the otherwise idle and deteriorated patients. He established a kindergarten class with a special teacher for the habit training of such patients, and promoted the dissemination of music through the hospital by the assignment of a trained

musician to carry out this function. Three years later he was able to report that a number of these patients had been able to return home apparently because of the benefit received by this individual treatment. He felt that in all probability none of them would have ever gone home except for such help.

In 1907 Dr. Hutchings had presented a discussion on the preparation of annual reports before the conference of the then State Commission in Lunacy. As a result he was appointed chairman of a committee to revise the statistical tables and to make recommendations for the preparation of annual reports. This committee became the Committee on Statistics and Dr. Hutchings has continued as its chairman up to the present time.

Early in the World War Dr. Hutchings volunteered his services, was commissioned as a captain of the Reserve Corps, in August, 1917, was ordered into active service, was promoted to the rank of major and was assigned to Columbia, S. C., to examine the 81st Division. In January, 1918, he was transferred to the office of the Surgeon General in the Division of Special Hospitals for Reconstruction, representing neurology and psychiatry. In July, 1918, he was assigned as Chief of Neuropsychiatry of General Hospital No. 31 at Plattsburg Barracks, N. Y. He continued in the service until February, 1919.

Meanwhile he had been offered the appointment as superintendent at the Utica State Hospital. This offer interested him as an extensive building program of a branch of the Utica Hospital at Marcy was about to start and it appealed to him to have a part in building and equipping such a new hospital, the estimated cost of which was $10,000,000. He left Ogdensburg in April, 1919, and took up his new duties as superintendent at Utica where he has continued. Under his direction the Marcy State Hospital was constructed and developed until it was made a separate hospital by legislative action in 1931.

Dr. Hutchings during his entire career has been keenly interested in the training of nurses. Under his administration at the St. Lawrence State Hospital the training school was developed to a high degree of efficiency. Soon after coming to Utica he helped to organize the Central Training School for Nurses in which the state hospital nurses received their preliminary training with pupil nurses of the general hospitals of Utica. He was made president of this

training school. During more recent years Dr. Hutchings has given strong support to the training of affiliating nurses at the Utica State Hospital and the training school continues to be an outstanding one in the state. He has continued throughout the years to give lectures to student nurses and has maintained a personal interest in their studies and training. His hospital has also been recognized as a training center for occupational therapists.

In 1908 Dr. Hutchings became a lecturer in psychiatry at the Medical School of Syracuse University. He continued his teaching after he came to Utica, giving a course of lectures at the medical school and conducting clinical demonstrations at the hospital. Recently he was made Professor of Clinical Psychiatry Emeritus of Syracuse University.

Dr. Hutchings has never allowed his administrative duties to interfere with his interest and activity in clinical psychiatry. Rarely if ever has he missed a staff meeting and at these he contributed to the knowledge and understanding of the younger members of the staff by his keen clinical observations and interpretations. At an early date he became interested in Freud's theories and their application to clinical psychiatry and has continued through the latter years to study and weigh these theories and to exercise his sound and mature judgment in determining their validity and their usefulness in psychiatry. Dr. Hutchings has long been a member of the American Psychoanalytical Society and was chairman of the section on psychoanalysis during 1936-1937.

Dr. Hutchings has always had an interest in community activities and welfare. At Ogdensburg he was recognized by the community as one of its outstanding citizens and such recognition has continued in Utica not only from the medical profession but from others who are imbued with humanitarian principles. He has been president of the Associated Charities of Utica, president of the Utica Torch Club, and for many years was a member of the Utica Rotary Club.

The Psychiatric Word Book which has passed through six editions is an outstanding contribution by Dr. Hutchings to psychiatry. In 1933 he was persuaded to accept the editorship of the Psychiatric Quarterly, the publication of the State Department of Mental Hygiene, and under his editorship this publication has increased materially in value and interest. It is not feasible here to list all of

Dr. Hutchings' other and numerous articles on the care and treatment of the mentally ill.

The life of any man is influenced for good or ill by his family. Dr. Hutchings is one of those fortunate persons who has had around him a happy, harmonious family. He married Lillie Beall Compton September 6, 1903. He had met and become attracted to his future wife when she was a girl with her hair down her back. Before he left for the study of medicine his troth was plighted and his acceptance of the position at Ogdensburg was influenced by the fact that the superintendent of that hospital was agreeable to having married men on his staff. Mrs. Hutchings had come from a prominent Georgia family; her grandfather had been comptroller of the state during the Confederacy, and she had finished her education in the Southern Female College at La Grange. Mrs. Hutchings has always been an interesting and interested member of the hospital community where her husband has been assigned and has endeared herself to all those who have had the privilege of knowing her.

Dr. and Mrs. Hutchings have had three children born to them. Richard Henry, the oldest, was graduated from Syracuse Medical School and followed his father's footsteps in psychiatry, becoming a member of the staffs successively of the Utica State Hospital, Marcy State Hospital, and of Harlem Valley State Hospital where he was latterly clinical director. His sudden and tragic death from coronary thrombosis December 14, 1938, was a severe blow to his parents as well as his many friends. Dr. Richard, Jr., had married Margaret Mackesy Hutchings in December, 1914. The family name of Richard Henry is carried on to their son who was born in 1921. The second son of Dr. Hutchings, Charles Wyatt, likewise was graduated from Syracuse Medical School and has followed his father in psychiatry, at present being senior assistant physician on the staff of the Marcy State Hospital. Charles was married December 9, 1921, to Madeleine E. Gorman; they have three children, Elizabeth born in 1930, David William born in 1933 and Cynthia now an infant. The only daughter of Dr. and Mrs. Hutchings, Dorothy Compton, has also shown her interest in psychiatry by entering psychiatric social service after her graduation from Mary Baldwin College. She is at present on the social service staff of the Utica State Hospital.

This brief sketch of the events in the life of our President and of some of his interests cannot give a picture of him as a man. Only those who have had the privilege of knowing Dr. Hutchings and of being associated with him can appreciate not only the keenness of his mind but his indefatigable enthusiasm in many fields and his genial, kindly interest in people, particularly his friends. One sees in him a man who, coming from pioneer stock, has made the most of his heritage, has added to the long record of his ancestors' achievements, and who now stands as an eminent physician and psychiatrist of whom the present generation and future generations may be proud. We wish for him many happy years of continued activity along with the satisfaction that may come to him from a serene contemplation of his many accomplishments.

THE BIOCHEMISTRY OF MANIC-DEPRESSIVE PSYCHOSIS.

A Review.[1]

By R. A. McFARLAND, Ph. D.,*
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"Manic-depressive psychoses rival dementia præcox in their interest to the research worker, be he clinical psychiatrist, pathologist or social statistician. In this so-called benign group are found the most hopeful as well as the most mysterious and most baffling forms of mental disease. The prevalence of these psychoses, their frequent recurrence and their favorable prognosis make them a most important subject of investigation" (64).

The manic-depressive psychosis offers a distinct challenge to the medical world. For centuries the picture of this mental disorder alternating in its various phases has occupied a conspicuous place in the psychiatric textbooks. The manic phase of the disorder, characterized by "psychomotor excitement with pressure of activity, flight of ideas, distractibility and happy though unstable emotional attitude" (31) is probably the average layman's visualization of all mental illness. In the depressed phase there are usually evident "psychomotor retardation, absence of spontaneous activity, dearth of ideas and depressed emotional attitude" (31). The mixed phase is characterized by symptoms drawn from both the manic and depressed phases in varying degrees.

In manic-depressive cases there is no clear-cut alteration in the histology of the nervous system. Many hypotheses have been put

[1] This article is the third of a series of four dealing with the blood biochemical studies of the psychoneuroses (52), dementia præcox (53), manic-depressive psychosis, and epilepsy.

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forth in an attempt to arrive at an etiology of this psychosis. These attempts have proceeded along many different channels. Besides the pathologist, the endocrinologist, the physiologist, the psychiatrist, the psychoanalyst and the psychologist have played their respective and several rôles. The manic-depressive's brain, endocrine glands, central and sympathetic nervous systems, blood pressure, libido and id, eidetic images and personality type, to mention a few, have come under the microscope of scientific investigation. But, in general, the studies have been either barren or inconclusive.

As in the case of the investigations of other baffling maladies, the chemist has been called in to determine the possibility of a chemical approach to a solution, since the various biochemical, endocrine and psychological manifestations are all parts of a disturbed neuro-physiology.[2]

The results emanating from the chemical laboratory have been confusing and of no considerable help either in solving this perplexing problem or in differentiating the various types of the psychoses. The causes of this confusion may lie in a variety of uncontrolled factors, such as faulty diagnosis, poor research technique in sampling of cases, unsatisfactory experimental set-ups and inadequate statistical treatment of the obtained results. Previous articles have discussed these factors as they affect studies of the psychoneuroses (52) and dementia præcox (53).

If the cause of manic-depressive psychosis is inexplicable, it may well be said that its nature is inexplicable also. There is still doubt in the minds of some as to whether this form of illness is a disease entity or merely a symptom-complex evinced as a result of other more or less organic disturbances. Perhaps a more varied symptomatology is difficult to find in any group of related disorders. Extreme affective manifestations characterize these disorders. Ease

[2] Freeman (23) stresses the importance of the chemical approach in the following: "Human constitution has been likened by Pende to a pyramid, the base of which is formed by heredity and the three faces respectively, the structural, chemical and psychological facets of the whole personality. No study is complete, therefore, which leaves out of consideration the chemical aspects. The immensity of this side is indicated not only by the organic and inorganic constituents of the body, but also by the endocrine secretions, the serology, and the whole field of immunities and susceptibilities to disease."

of excitation and depression, mood fluctuations running the range from maniacal bursts of frenzy to profound stuporous depression and, in general, a defective emotional homeostasis are part of the symptom picture.

Those mental disorders characterized by emotional excesses have been generally, categorically and uncritically classified as cyclothymic. Furthermore, the difficulty of affect labelling is ever present; for instance, the clinical distinction between "apathy" and "depression," or between "manic excitement" and "catatonic excitement" is subtle. Not only is there danger of a tendency to faulty psychiatric diagnosis and classification, but there is very real and serious possibility of misinterpretation of the particular emotion.

The manic-depressive disorders, probably more than any others, have been neglected from a classification viewpoint.[3] This neglect, dating generally from the time of Kraepelin's classification, is not without cause, since this group seems to cover a vast symptomatology and since "there is no proof to indicate that the various phases of the psychosis are aspects of one basic or fundamental psychological or physiological process" (50). It is certain that many cases of dementia præcox or paranoia or other mental conditions have been grouped with the affective psychoses because

[3] Partridge (61) discusses this point adequately. "The class of 'manic-depressive psychoses,' it seems, has been too loosely conceived, with too great a tendency to easy generalization and synthesis, with the result that the ordinary clinical consideration of the manic depressives is clouded in obscurity, and the research approach has also been backward. We doubt seriously whether all the reactions now commonly included in this group are very significantly related, and we should say that the two-phase reaction which has stood as the prototype of the class, and the bipolar unit cooperating all the great congeries of manifestations, is open for much more critical treatment from the standpoint of classification and methodology; especially since both in its clinical manifestations and in the personality background there is so much that approximates the formations we see in the types which we are accustomed to call psychopathic personalities, and so much that is allied to schizophrenia. We have in fact some justification for saying that the concept of 'manic-depressive psychosis' has still to defend itself as a valid basis for any class name that does more than coordinate, at the plane of superficial resemblances and affiliation the great collection of phenomena to which the term 'manic-depressive' has been applied."

of similar symptoms.[4] There is still much to do in the matter of proper clinical classification.[5]

The errors of diagnosis and classification bear their fruit in the confusion of results of physiological group studies. The biochemist cannot, in all fairness to science and himself, state that there are no biochemical correlates of manic-depressive psychosis when he is not even sure that the group he is testing is 100 per cent manic depressive.

The difficulty of diagnosis based upon mental symptoms is, however, well appreciated. Mental symptoms are noted for their elusiveness, uncertainty, vagueness and difficulty of description. Furthermore, there is no specific test to establish or corroborate diagnosis as in the case of many organic diseases.

The literature on the subject is in a chaotic state. This paper will concern itself with some biochemical studies of blood that have been made in manic-depressive psychosis. The biochemical constituents that have been most frequently investigated are blood sugar, acidity and alkaline reserve, calcium and phosphorus, nitrogenous substances (proteins, total nitrogen, nonprotein nitrogen, ammonia, uric acid, etc.), cholesterol and chlorides. The list of studies cited below makes no claim to completeness, but is rather a cross-section of the research in this field based upon the completeness of data from experimental studies.

Blood Sugar.—Kilduffe (37, page 513) states, "the sugar metabolism is controlled by the endocrine secretions acting as inhibitors and accelerators. Normally these are accurately balanced and by their compensatory interaction maintain the blood sugar

[4] Rachlin (67) reported that of 132 cases of benign stupor admitted at Manhattan State Hospital over a period of 17 years, 56 were followed and 71.4 per cent of these had a revision of diagnosis to schizophrenia. These facts led him to conclude that the diagnosis of benign stupors (which comprise a sub-group of the manic-depressive group) lacks objective verification, thus appearing to throw doubt on the value of this concept as a psychiatric clinical entity.

[5] Lewis and Hubbard (42) "feel that clinical psychiatry has not yet finished with its descriptive phases from the standpoint of classification, and that more attention could be paid with profit to making more accurate clinical groupings." They "make this statement in the light of the knowledge that biochemists and pathologists are trying to use these labelled groups for correlative studies."

within normal limits." Normal blood contains from 80 to 120 mg. per 100 cc. blood.

Some investigators have reported normal blood sugar for the manic depressive. Table 1 presents a summary of the essentially negative findings.

Not all investigators, however, have reported normal values in the manic depressive. Kooy (39) using the Bang micro-method in 1919 studied 19 melancholics, 11 manics and 20 normals and found that emotions, especially those of a depressive character, caused an increase in blood sugar. Hyperglycemia was most frequent in melancholia, although it was not a constant symptom. With marked anxiety there was the maximum amount of blood sugar, although hyperglycemia might occur with any emotional state in mental disorders, such as general paresis, epilepsy, dementia præcox, traumatic neuroses, etc.

Heidema (27) in 1919 using the same method studied 17 melancholics and three manics and found that in most cases there was a hyperglycemia. More than half of the cases of melancholia showed high blood sugar values. Of these 10 only four showed glycosuria. In all of the manics (3) there was a hyperglycemia without a glycosuria. The investigator felt that the hyperglycemia present in manic-depressive psychosis was a result of increased muscle action and consequently an elevated metabolism.

Wuth (94) in 1920 using the Neubauer reduction method made a study of 30 melancholics and found hyperglycemic values in 18 of these cases. The average value was 115 mg. A regular dependency of hyperglycemia upon the affect could not be demonstrated. It was felt by the investigator that the causes of hyperglycemia lay probably in the internal secretory processes. Di Renzo (19) in 1925 studied 18 melancholics and five manics and could find no relation between hyperglycemia and emotional tone or severity of disease. The investigator felt that the hyperglycemia might be related to a neuro-endocrine disturbance.

Craig (13) in 1927 tested a group of late agitated melancholics and reported a definite degree of hypoglycemia in the starvation level.

Labin (41) in 1927 studied 23 manics and 23 melancholics. In the former group the investigator found 17 cases of hyperglycemia and 16 cases of hyperglycemia in the latter group. The variations

TABLE 1.

Blood Sugar: Summary of Essentially Negative Findings.

(Results in mg. per 100 cc. blood.)

Investigator. Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Weston (89) 1916	Myers-Bailey	10	6 hypomanics, 3 depressives, 1 interval	121.5	...	...	102.5–140.0	Normal. Individual differences were considerable.
Wigert (93) 1916	Bang micro-method	15	depressives	....	...	...	.....	In 12 cases of the 15, found normal results.
Weston (90) 1920	Myers-Fine	10	3 hypomanics, 4 depressives, 3 interval	97.5	...	...	85 –120	Normal.
Newcomer (55) 1922	Folin-Wu	5	cyclothymics	....	...	...	.....	Normal. No relation between blood sugar content or reactions to changes in it and mental state.
Lorenz (46) 1922	Lewis-Benedict	17	10 manics	112	...	...	100 –126	Normal.
			7 depressives	106	...	...	82 –122	
Bowman (8) 1923	Folin-Wu	101*	69 depressives	109.1	17.8	16.3	65 –166	Normal results except where organic physiological conditions were present.
			28 manics	104.0	15.7	15.1	74 –133	
			4 mixed	101.0	11.6	11.5	93 –121	
				107.4	17.0	15.8		
Schryver (72) 1923	Lewis-Benedict	4	melancholics	....	...	...	.....	Normal.
Genzel (25) 1923	Benedict modification	13	depressives	....	...	...	.....	Normal in 9 of the 13 cases.
Mumford and Parkin (54) 1923	McLean	33	manics	....	...	...	.....	No evidence that their mechanism of sugar metabolism differs from the normal, although excitement may cause increased blood sugar until excitement abates.
Barrett and Serre (6) 1924	Folin-Wu	20	5 manics	119.0	...	...	105 –131	Normal.
			15 depressives	105.3	...	...	82 –132	
				109.0				

TABLE 1.—Continued.

Investigator. Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Barrett and Serre (6) 1924	Folin-Wu 20	5	manics	119.0	...	...	105 –131	Normal.
		15	depressives ...	105.3	...	...	82 –132	
				109.0				
Looney (44) 1924	Folin-Wu 41	11	manics	87.5	...	...	81 – 99	
		30	depressives ...	94.5	...	...	72 –126	
				92.6				
		19	controls	89.8				
Dide, Fages and Bauduin (17) 1924	 8		cyclothymics ..	108	...	...	84 –140	Normal in periods of calmness, but "pre-seizure" period marked by decreased blood sugar.
Henry and Mangam (29) 1925	Folin-Wu 8	7	depressives ...	138	...	...	130 –154	Normal.
		1	manic	129	...	...	.....	
Reid (70) 1927	Folin-Wu 18		melancholics ..	99	...	...	75 –145	Normal.
Craig (13) 1927	Calvert 7		recurrent melancholics	120	...	...	80 –200	Normal.
Bowman and Kasanin (9) 1929	Folin-Wu 73	30	manics	104.5	14.7	14.1	75 –140	Normal. No correlation between mood and blood sugar values.
		34	depressives ...	102.1	17.1	16.7	65 –140	
		3	hypomanics ...	98.0	...	...	95 –115	
		6	mixed	98.3	...	...	65 –125	
				107.2	15.9	14.8		
		14	controls	91.0	7.5	8.2		
Whitehorn (92) ...1934	Folin-Wu 520	345	depressives ...	....	...	...	.....	Emotional excitement rarely, if at all, produces any rise in the fasting blood sugar. There is found more hyperglycemia in the depressive reactions of the later decades of life.
		151	manics	....	...	...	.....	
		24	mixed	....	...	...	.....	
Sacristán (71) 1934	Hagedorn-Jensen. 17	11	depressives ...	106.0	...	...	90 –138	Normal.
		6	manics	106.3	...	...	90 –133	
				106.1				
Marshall (49a) 1939	Calvert 4		alternating cyclothymics:					Normal.
			depressive phase ..	93.3	...	...	70 –111	
			manic phase	93.8	...	...	70 –110	
				93.5				

TABLE I.—CONTINUED.

* Omitting diabetics.

from the normal limits were often slight. Bogen (7) in 1928 using the Hagedorn-Jensen method made a study of 23 cyclothymics (11 very depressed, seven mildly depressed, three manics, one mixed, one mild hypomanic) and found blood sugar in 13 cases at a high level and above the normal limits.

Some investigators have attempted to determine what relation, if any, exists between manic-depressive psychosis and sugar tolerance. Table 2 gives the negative findings in this regard.

Some workers in the field, however, have found evidence of abnormal sugar metabolism in the manic depressive. Kooy (39), using the Bang micro-method in the study cited above, found the highest tolerance curves in the cases of marked anxiety and melancholia. Emotions, especially those of a depressive character, caused increases in blood sugar.

Raphael and Parsons (69) using the Lewis-Benedict method in 1921 made a study of 11 manic depressives and seven normal controls. Tolerance curves differing from normal and among themselves were obtained in the hypomanic and depressed phases of the psychosis. In each group common features suggested a type curve. In the hypomanic and agitated depression cases there was a tendency toward initial hypoglycemia and a flat tolerance curve. In the depressed cases there were indications of initial hyperglycemia with delayed tolerance more marked than in acute dementia præcox. Olmsted and Gay (56a) in 1922 using the Myers-Bailey method studied seven manic depressives and reported that, in the majority of cases, high curves were revealed.

In 1924 Tsuchiya (87) using the Bang micro-method tested five manic depressives and five normals and found that the curves of the patients were different from those of the normal controls. The normal individuals showed the highest sugar content one hour after ingestion of sugar; after three hours blood sugar content was the same as before ingestion and after five hours it had fallen to 90 mg. per 100 cc. blood. In the manic depressives the blood sugar rose after one hour, but after two hours it fell only to rise again to a higher level than ever before from the third hour. The investigator believed that these findings were in some way dependent upon the internal secretions. Cowie, Parsons and Raphael (12) in 1924 studied the glucose tolerance curves of seven manic depressives

TABLE 2.

SUGAR TOLERANCE: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. per 100 cc. blood.)

Investigator.	Date.	Method.	No. of subjects.	Kind of subjects.	Average change. (+ increase.) (− decrease.)	Comments or conclusions.
Lorenz (46)	1922	Lewis-Benedict	17	10 manics	+ 37.0	Normal sugar tolerance although depressives showed a curve higher than normal.
				7 depressives	+ 60.0	
					+ 46.5	
Barrett and Serre (6)	1924	Folin-Wu	30	15 anxious depressives	+ 88.0	Normal sugar tolerance in mild depressives and manics, but higher curves in anxious depressives.
				9 mild depressives	+ 23.0	
				6 hypomanics	− 32.0	
					+ 34.0	
Craig (13)	1927	Calvert	12	5 late agitated melancholics	+ 80.0	Normal sugar tolerance but a tendency to reach high levels during the test meal noted in recurrent melancholics.
				7 recurrent melancholics	+ 60.0	
					+ 68.3	
Katzenelbogen and Muncie (36)	1935	Folin-Wu	3	depressives		No correlation between blood sugar curves and various kinds of emotional reactions separately considered or improvement in the mental state.
Marshall (492)	1939	Calvert	4	alternating cyclothymics:		Depressive phases showed an alimentary hyperglycemia with delay, but spontaneous hyperglycemia is absent. In quiescent phases and during mild mania, the blood sugar curve approximates the normal.
				depressive phase	+138.5	
				manic phase	+ 92.3	
					+115.4	

and one normal individual. They found higher initial values and higher final values for the patients.

Henry and Mangam (29) in 1925 tested a group of depressives and a group of manics and reported that the glucose tolerance tests showed more or less characteristic changes in the glucose content of the blood of patients in either phase of the affective psychoses. These changes indicated a definite retardation of the functions of the vegetative nervous system in manic-depressive depression and an acceleration of the same functions in manic-depressive excitement.

Drury and Farran-Ridge (20) in 1925 studied 100 psychotics of various classifications, including manic depressive. They found that the blood sugar curves of the psychotic showed more variation than those of normal people, leading the investigators to believe that the general metabolism is far more disordered in mental illness than one would be led to believe by casual observation. They also reported that among the mental symptoms, confusion and melancholia are associated with the greatest disturbance of the sugar metabolism.

In 1925 Mann (48) using the Calvert modification of the Folin-Wu method tested 152 cases of mental disorder, including a group of cases of melancholia. He found that a condition of sustained hyperglycemia with high blood sugar levels was most marked in the cases of acute melancholia, although several cases of melancholia had normal blood sugar curves. Craig (13) in 1927 found defective sugar tolerance in a group of early agitated melancholics.

Raphael, Ferguson and Searle (68) using the Folin-Wu method in 1928 made a long-section blood sugar tolerance study in one case of manic-depressive depression uncomplicated and paradigmatic somatically, genetically and psychiatrically. Fifteen determinations over a period of eight months were made in order to attempt to correlate the carbohydrate metabolism with clinical phase of the depression. There was revealed a curve of decreased tolerance resembling that of diabetic mellitus. There was greater decreased tolerance the more profound the depression; that is, the rise in the sugar curve ranged from 160 mg. during profound depression to 38 mg. when practically recovered. The abnormality of carbohydrate metabolism seemed to be phasic and to disappear with clinical recovery. The investigators felt that the physiologic factor

might be either primary or secondary to mental symptomatology, since it was difficult to separate one from the other.

Mann and Scott (41) in 1929 tested four melancholics, two depressives and eight controls and found abnormal results in the majority of patients. The abnormality of the curve in the psychotics mainly took the form of delayed glycogenesis. The investigators suggested that these abnormal sugar curves might be related to a severe defect in carbohydrate metabolism and that its main importance might be in giving some information about the acid-base equilibrium. McCowan and Quastel (51) in 1931 tested 43 melancholics and four manics. In the manic-depressive group they found a close parallelism between the magnitude of the hyperglycemic index (which is a quantitative measure of the departure of a sugar tolerance curve from that found in a normal case) and the emotional tension of the patient as measured by the psychogalvanic reflex. Of 43 melancholic patients only 10 gave a normal or slightly abnormal index. Those of the melancholic group who failed to show a raised blood-sugar level proved to have an hysteriform element in their psychoses. (It was believed to be significant that patients manifesting hysteria usually have a normal blood-sugar level.) It cases of mania a low index was found, except when the excitement was accompanied by an aggressive paranoid mood. Northcote (55a) in 1932 studied seven melancholics and two manics and reported that in six melancholics and in one manic there was a sustained hyperglycemia which was interpreted in the light of McCowan and Quastel's (51) hypothesis, *i. e.*, that the sustained hyperglycemia was fundamentally an index of the emotional tension of the patient at the time of the test.

Hackfield (26) in 1932 studied 20 manic depressives and found that these patients showed, in general, a delay in return of blood sugar to normal in tolerance tests. Ström-Olsen (79) in 1932 using the Folin-Wu method tested a group of 36 patients which included cases of manic depression and melancholia. A severe disturbance of carbohydrate metabolism was found in 21 of these 36 patients. However, nine of the 21 had definite organic disease. Katzenelbogen and Friedman-Buchman (34) in 1933 studied 50 manic depressives and found abnormal curves in 72.9 per cent of these patients.

Sacristán (71) in 1934 using the Hagedorn-Jensen micro-analytic method tested 17 female manic depressives (seven melancholia, four mania, two hypomania, four hypomelancholia) and found that the sugar curve is of a different kind in manic-depressive psychosis. He did not believe that the depressive state could be considered the cause of the hyperglycemia, for it was present in the manic as well. However, he intimated that the cause of the alterations in carbohydrate metabolism was a condition of hyper-excitability of the vegetative nervous system. He felt that the manic-depressive psychosis was the psychosis of endogenous nature that most frequently showed changes in the carbohydrate metabolism. The investigator suggested that the diversity of results obtained might depend on constitutional factors, since the patients with the pyknic habitus presented the greatest number of carbohydrate anomalies in metabolism. Tod and Jones (83) in 1937 made a study of 20 manic depressives and reported that the hyperglycemic index varies in depressions as the emotional tension; that is, the greater the depression the greater the hyperglycemic index or retardation of the carbohydrate metabolism. Ansani (1a) using the Folin-Wu method in 1938 reported after a study of 10 cyclothymics that, although the rise in glycemia following administration of sugar was not high, the rise was retarded as was the drop after the peak had been reached.

Acidity and Alkaline Reserve.—Kilduffe (37, page 59) in speaking of the pH of the blood states, "The reaction of the blood is maintained, in spite of the ingestion of acids and basic salts and the metabolic production, neutralization, and excretion of acids and bases, by a very sensitive mechanism of adjustment. The three processes mainly concerned are the excretion of acids and nonvolatile bases by the kidney, the base forming properties of hemoglobin, and the excretion of carbon dioxide through the lungs, the two latter being more rapid and important. The buffer system of the blood also functions, but is not of as great importance nor capable of as rapid adjustments as those just mentioned." Kilduffe (37) states that the normal range for pH is from 7.3 to 7.5, and for plasma carbon dioxide combining capacity, from 50 to 75 volumes per cent, and from 23.0 to 32.0 millimoles per liter. The normal range for total base is from 149.0 to 163.0 milliequivalents per liter serum.

TABLE 3.

ACIDITY AND ALKALINE RESERVE: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

[Results in plasma volumes per cent (alkali reserve).]

Investigator. Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Pugh (65) 1903	Wright 25		15 manics .... 10 melancholics	...	...	...	.....	Alkalinity of the blood normal, although it was lowered in manics during excitement.
Suitsu (80) 1920	Van Slyke-Cullen	54	39 depressives 15 manics ..	.. 64.42	9.1	14.1	52.75– 75.38	Average alkaline reserve was normal. Variability of the alkaline reserve in patients was slightly higher than in normal controls. No demonstrable difference between alkaline reserve of excited and depressed patients.
		14	controls	62.60	8.3	13.3	50.20– 71.04	
Jarloev (33) 1921	Hasselbalch	8	cyclothymics	...	...	...	7.32– 7.36	Normal, in pH.
Thomas (82) 1922	Van Slyke		cyclothymics	...	...	...	.....	In the majority of cases there was no acidosis whatever.
Henry and Mangam (29) 1925	Van Slyke-Cullen	89	55 depressives .. 58.5 34 manics 61.2	...	...	...	44.3 – 69.1 51.9 – 73.0	Alkaline reserve unaffected by psychosis unless there is some definite accompanying physical disease.
			59.5					
Ballif and Ghersco-vici (5) 1931	23		12 manics .... 11 melancholics	...	...	...	.....	Normal pH and alkaline reserve.
Appel, Farr, and Hodes (2) 1931	Van Slyke and Stadie-Ross	65	26 manics: alk. reserve .. 22.9 total base ....155.6 19 depressives: alk. reserve .. 22.6 total base ....154.5 20 mixed: alk. reserve .. 22.2 total base 157.8		3.5 6.7 3.6 4.0 3.0 8.6	15.3 4.3 15.9 2.5 13.5 5.4	15.2 – 30.7 143.6 –168.9 14.2 – 31.4 149.8 –161.2 17.9 – 27.9 146.8 –166.2	Normal total base. The alkaline reserve was normal except in cases complicated by other factors, *e. g.*, toxic conditions, physical conditions, etc. (Alkaline reserve expressed as millimoles per liter and total base expressed as milliequivalents per liter serum.)
			alk. reserve .. 22.6 total base 156.0		3.4 5.9	15.0 3.8	....	

Many investigators have attempted to determine whether there is any relation between the acid-base equilibrium of the blood and manic-depressive psychosis. Some investigators have reported normal results. Table 3 gives the essentially negative findings.

Not all investigators, however, have reported normal acid-base values for the manic depressive. Poli (63) using a pH colorimetric method and the Van Slyke alkaline reserve method in 1931 studied a group of 12 depressives and 10 manics and reported a constant and marked lowering of pH (7.01) in excitable states and a normal or slightly lowered pH (7.21) in depressive states. The alkaline reserve was markedly diminished (average, 37.0± 2.8; range, 31.1-41.9) in excitable states and almost normal in depressive states (average, 47.8±6.4; range, 39.2-64.3). Notkin *et al.* (56) using the Van Slyke method in 1933 studied four manic depressives and found a decreased oxygen content, an increased oxygen unsaturation, and an increased carbon dioxide content.

Calcium and Phosphorus.—Kilduffe (37, page 551) remarks that "calcium is concerned with maintenance of physiological equilibrium and the normal irritability of the nervous system." Calcium has an influence on the excitability of the motor systems of animals. In tetany there is a deficiency in the calcium concentration of the blood. The calcium content of normal blood serum is 9-11 mg. per 100 cc. Kilduffe (37, page 570), discussing the function of phosphorus in the blood, states that "phosphates act as buffer, but the relation to normal and pathological states is not yet determined." The normal inorganic phosphorus content of the blood plasma is from 3 to 5 mg. per 100 cc.

Some investigators here reported normal calcium values in the cyclothymic individual. These findings are shown in Table 4.

Some investigators have failed to confirm normal calcium results in the manic depressive. Klemperer (38) in 1926 using a modification of the Kramer-Tisdall method tested 18 melancholics and 12 cases of mania or depression and reported that calcium was diminished in melancholia agitata. Cases of melancholia with stupor had low calcium, but cases of mania had high calcium. Tómasson (85) in 1927 using the de Waard method studied a group of 170 mental cases, including a subgroup of manic depressives. He found deviations in the serum calcium in both mania and depression. Of the manic-depressive patients, all of those examined during the manic phase and half of those examined

TABLE 4.

CALCIUM AND INORGANIC PHOSPHORUS: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

TABLE 4.

CALCIUM AND INORGANIC PHOSPHORUS: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

[Results in mg. per 100 cc. serum (calcium) and mg. per 100 cc. plasma (inorganic phosphorus).]

Investigator. Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Weston (90) 1920	Lyman 10		cyclothymics ...	8.14	...	...	7.0 – 9.1	Normal.
Weston and Howard (91) 1922	Kramer-Tisdall ...27	17	manics	10.4	1.2	11.1	9.5 –12.0	Normal. Individual variations from the averages were small.
		10	depressives	10.6	1.0	9.5	9.0 –12.7	
				10.5	0.8	7.6		
Tómasson (84) 1924	de Waard		cyclothymics		...	...	.....	No value below 10.0 mg. per cent. No absolute agreement between calcium deviation and the particular psychiatric diagnosis or motor restlessness.
Siegler (76) 1925	de Waard 13		manics		...	...	.....	Ten cases normal but toward lower limits; 3 cases were hypocalcemic.
Henry and Ebeling (28) 1926	Clark-Collip 28	16	manics:					Normal except that there was a slight increase in calcium and phosphorus in manic states and a slight decrease in agitated depressed cases.
			calcium	11.92	...	...	9.86–16.55	
			inorganic phosphorus	3.82	...	...	3.02– 5.28	
		12	depressives:					
			calcium	11.49	...	...	9.87–12.50	
			inorganic phosphorus	3.63	...	...	2.40– 5.77	
			calcium	11.74				
			inorganic phosphorus	3.74				
		7	controls:					
			calcium	10.65	...	...	7.89–12.50	
			inorganic phosphorus	4.15	...	...	3.54– 4.68	
Armstrong and Hood (3) 1927	Clark-Collip 24	15	melancholics ...	9.9	...	...	9.0 –10.9	Normal calcium. A high or low content was not diagnostic of any spec. condition.
		9	manics	9.9	...	...	9.3 –10.5	
Katzenelbogen and Goldsmith (35) ..1931	Clark-Collip 10	3	agitated depressives		...	...	9.2 –10.1	Normal.
		2	manics		...	...	9.2	
		5	depressives		...	...	9.0 –10.0	
Parhon and Werner (60) 1932	13	7	manics	11.1	...	...	10.4 –12.0	Normal.
		6	melancholics ...	10.2	...	...	8.7 –11.2	
				10.7				
H. T. Edwards (unpublished) 1936	42	11	manics	11.1	...	...	.....	Normal.
		31	depressives	10.8	...	...	.....	
				10.9				
		16	controls	10.4				

during the depressive phase showed deviations in serum calcium. In the manic phase, all of those who deviated showed fluctuating on constantly high values, but none low. Deviations in serum calcium occurred in patients of all types, but pyknic patients showed a somewhat more frequent deviation than other types of patient or patients without a definite type. The deviation of the attack did not seem to influence the frequency of the serum calcium deviations. These deviations could not be due to motor activity, as they were not found after light physical work, short strenuous or prolonged strenuous work or complete rest. The investigator felt that the deviations found in manic depressives were due to the altered affectivity of these patients, as he found a relationship between mood and the absolute amounts of serum calcium. The investigator was also of the belief that in manic-depressive psychosis there is a humoral instability characterized by abnormal fluctuations in the electrolytic conditions of the blood. This instability occasioned an irritability, especially of the vegetative nervous system. In 1927 Kora (40) using the Kleinman total phosphorus method tested nine manics and three melancholics. In both the manic (average, 37.64 mg.) and melancholic (average, 39.76 mg.), he found increased total phosphorus in the blood serum. Bogen (7) in 1928 tested 23 manic depressives and reported serum calcium values above normal. Tómasson (86) in 1932 tested nine cyclothymics and found a tendency to hypercalcemia as well as a decrease in the irritability of the vegetative nervous system.

Atkin (4) in 1937 using the Kramer-Tisdall method studied a group of manic depressives and reported that they exhibited lower values for the depressed phase and higher values for the manic phase, suggesting a parallel between serum calcium levels and emotional tone. The average of 47 melancholics was 10.07 mg., while that of 13 manics was 10.55 mg.

Nitrogenous Substances.—The picture of nitrogen metabolism is an important reflection of the general state of anabolic and katabolic activities of the body. The urea nitrogen of normal whole blood is 10-20 mg. per cent, non-protein nitrogen 28-39 mg. per cent, of creatinine 1-2 mg. per cent. In serum the total protein content is 6.7-8.7 per cent, total albumin 4.95-7.7 per cent and total globulin 1.0-2.5 per cent.

Many investigators have reported normal values for the cyclothymic. Table 5 presents these essentially negative findings.

TABLE 5.

NITROGENOUS SUBSTANCES: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. per 100 cc. blood.)

Investigator. Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Adler and Ragle (1).1914	Folin-Denis	5	cyclothymics: uric acid	1.84	...	...	0.74– 2.80	Normal.
Weston (90) 1920	Folin-Denis	10	cyclothymics: nonprotein N .	33.4	...	...	31 –36	Normal.
			total N	3.57	...	...	3.20– 3.85	
			uric acid	2.24	...	...	1.80– 2.75	
			urea	21.9	...	...	16 –27	
			creatinine	1.92	...	...	1.60– 2.15	
			creatinine plus creatine ...	7.80	...	...	7.00– 8.90	
Frigerio (24) 1921	.....	10	4 manic: nonprotein N .	16.5	...	...	5 –22	Normal.
			6 melancholic: nonprotein N .	27.2	...	...	17 –42	
				22.9				
Newcomer (55) 1922	Folin-Wu	5	cyclothymics ...	...	...	...	.....	Normal nonprotein N, uric acid, urea N. Found no relation between blood content and mental state.
Bowman (8) 1923	Folin-Wu	104	70 depressives: nonprotein N .	30.8	5.9	19.2	21 –50	Normal.
			uric acid (N = 42)	1.61	0.68	42.2	0.4 – 3.8	
			30 manics: nonprotein N .	28.3	5.4	19.1	17.6 –39.9	
			uric acid (N = 22)	2.14	1.33	62.2	0.8 – 6.6	
			4 mixed: nonprotein N .	28.7	3.5	12.2	25.5 –33.6	
			uric acid (N = 3)	1.90	0.37	19.5	1.5 – 2.4	
			nonprotein N .	30.0	7.9	26.3		
			uric acid	1.80	0.95	52.8		

TABLE 5.—CONTINUED.

Investigator.	Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Looney (44)	1924	Folin-Wu	41	11 manics:					Normal.
				total N	30.8	...	...	23.4 –37.0	
				urea N	14.4	...	...	7.5 –19.4	
				amino-acid N	6.85	...	...	5.12– 7.90	
				uric acid	1.01	...	...	0.84– 1.18	
				creatinine N	0.54	...	...	0.38– 0.75	
				creatine N	2.38	...	...	1.43– 3.66	
				30 depressives:					
				total N	32.6	...	...	25.5 –42.3	
				urea N	13.8	...	...	8.1 –26.1	
				amino-acid N	6.89	...	...	4.83– 9.40	
				uric acid	1.02	...	...	0.73– 1.47	
				creatinine N	0.50	...	...	0.36– 0.67	
				creatine N	2.59	...	...	1.59– 4.05	
Barrett and Serre (6)	1924	Folin-Wu	20	15 depressives:					Normal.
				total NPN	37.5	...	...	26 –50	
				urea	17.9	...	...	12.0 –28.7	
				uric acid	2.1	...	...	1.2 – 3.3	
				creatinine	1.3	...	...	0.9 – 2.0	
				5 manics:					
				total NPN	39.6	...	...	31.9 –55.0	
				urea	18.5	...	...	14.3 –30.0	
				uric acid	2.4	...	...	2.0 – 3.3	
				creatinine	1.3	...	...	1.0 – 1.6	
Henry and Mangam (29)	1925		62	22 manics:					Normal.
				urea N	15.0	...	...	10.0 –31.3	
				40 depressives:					
				urea N	15.2	...	...	8.3 –25.0	
					15.1				
Parfitt (58)	1933	Urease	55	15 manics:					Eleven of the 15 manics had normal blood urea. Practically all of the melancholics normal.
				urea N	...	...	...	.....	
				40 melancholics:					
				urea N	...	...	...	.....	
Vuanello (88)	1938		13	7 depressives:					Normal.
				amino-acid N	6.66	...	...	5.00– 8.00	
				6 manics:					
				amino-acid N	6.93	...	...	4.15–11.00	

Normal findings have not been reported by all investigators. Looney (44) in 1924 also tested 30 cases of depression and reported a somewhat high plasma undetermined nitrogen (average, 7.75 mg. per cent; range, 0-14.00 mg. per cent). He was of the opinion that toxic amines were present in the blood of markedly depressed cases. Reid (70) in 1927 using the McLean method tested 18 melancholics and found in 14 of these depressed patients an increased nonprotein nitrogen content (average, 43 mg. per cent) which the investigator thought might support the theory of Looney (44) that a relationship existed between certain toxic amines and melancholia. However, using the McLean method, he found normal blood urea (average, 13 mg. per cent) in these patients.

Ludlum, Taft and Nugent (47) in 1931 using physico-chemical methods to determine specific gravity and relative viscosity found indications in 20 manic depressives of lowered values for total serum protein in the manic and depressed cases.

Lipoidal Substances.—In discussing the need for studies of the lipoid constituents of the blood, Looney and Childs (45) state, "The fact that approximately 50 per cent of the brain is made up of lipoid constituents has led naturally to the question whether patients showing mental disturbances would not also show changes in cholesterol metabolism. . . . . Variations in the level of the blood cholesterol have been ascribed to changes in emotion and to disturbances of the endocrine glands, especially the thyroid gland, the suprarenals, and the gonads." Duncan (22) believes cholesterol to be one of the controlling factors of cellular metabolism. Normal blood contains from 140 to 200 mg. of cholesterol, from 250 to 450 mg. of lecithin, and from 290 to 410 mg. of fatty acids per 100 cc.

Several investigators have reported normal values in the blood of the manic depressive. Table 6 presents the negative findings.

Many other investigators have reported abnormal lipoidal values in the blood of the cyclothymic. Pighini (62) in 1911 studied a group of manic depressives and found comparatively high values of cholesterol and oxycholesterol in their blood.

Parhon, Urechia and Popea (59) in 1913 using the reaction of Neumann and Hermann tested a group of cyclothymics and reported lipoidal increases in most cases. Targowla, Badonnel and Berman (81) in 1923 using the colorimetric Grigant method tested four cases of anxiety melancholy and reported higher amounts of

TABLE 6.

Lipoidal Substances: Summary of Essentially Negative Findings.

(Results in mg. per 100 cc. blood.)

Investigator. Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Cruickshank and Tisdall (14) 1916	Cruickshank-Tisdall.	7	cyclothymics: cholesterol (serum)	116.0	...	...	80 –200	Normal total cholesterol.
Newcomer (55) 1922	Weston	5	cyclothymics: cholesterol	...	...	...	.....	Normal cholesterol.
Lockwood (43) 1932	Sackett modification of Bloor 17		cyclothymics: cholesterol	171.2	22.0	12.8	144 –230	Normal.
Laursen (41a) 1936	10		cyclothymics: cephalin	2.00	...	...	0.91– 2.71	Normal.

cholesterol than normal. Ornstein (57) in 1925 found hypercholesteremia predominant in cases of the affective psychoses.

In 1927 Jacobi (32) using the Myer method tested 20 manic depressives. A leaning toward hypercholesteremia was found which the investigator thought probably had a close relation to inner secretory mechanisms. Stenberg (78) in 1929 using the Blix modification of the Bang method studied a group of cyclothymics and found both in the manic and melancholic phases of the manic-depressive psychosis a statistically significant increase of total cholesterol and total fatty acids, while during remission the return to normal values was also significant.

Heuyer (30) in 1930 also using the Blix modification tested 14 manic depressives and found an increase in blood cholesterol in the excitable period and a decrease in periods of remission. He also found that depressive states tended to have low blood cholesterol, while excitable states showed increases. Duncan (21) using the Liebermann reaction in 1930 tested a group of manic depressives and reported that the serum cholesterol was raised in most cases, especially during remission (average, 205 mg. per cent in acute phase). The investigator found that patients with excessive emotional reaction had a lower serum cholesterol than others in the same psychiatric group. This was observed in the mood changes of the psychosis.

In 1931, Shaw and Sharpe (75) reported abnormal increase of blood cholesterol content in states of mania. Duncan (22) in 1933 tested several cases of manic-depressive insanity and found that blood cholesterol was lowered in heightened emotional states that affect the individual as a whole, while it was increased in apathetic states. Slight and Long (77) in 1933 using the Myers-Wardell and the Stoddard-Drury methods tested a group of cyclothymics and a group of normals and found that the total fatty acids, cholesterol and lipoid phosphorus of the plasma were higher in the patients than in the normal controls. The average total fatty acids was 30.8 per cent higher in the patients than in the normals, while average cholesterol was 29.8 per cent higher. The patients' average lipoid phosphorus was 2.4 per cent higher than that of the normals. After a fat meal given to both patients and the normal controls these percentages increased still further. After the meal the postprandial rise in total fatty acids for normals was 8.1 per cent, for

manic depressives 34.0 per cent; in cholesterol for normals 3.3 per cent, for manic depressives 10.6 per cent; in lipoid phosphorus for normals 2.4 per cent, for manic depressives 11.5 per cent. The investigators concluded that in certain manic depressives there was a disturbed lipoid metabolism.

Sharpe (74), in a review of the literature, reported that cases of recurrent mania showed a very high blood cholesterol (average, 320 mg. per cent) especially during an acute attack, while in cases of melancholia the blood cholesterol was slightly on the high side (average, 240 mg. per cent), but the variations were small. Claude (11) in 1935 using the methods of Grimbert, Laudat and Weill, tested seven melancholics and three normal controls and found in the patients an increased blood cholesterol content (average, 202 mg. per cent; range, 150-228 mg. per cent) and a decreased blood fatty acid content (average, 3.94 mg. per cent). The increases seemed to be noted when there was an increase in emotional tone, regardless of the malady. Schube (73) using the modified Myers-Wardell method in 1936 studied the cholesterol metabolism of 71 manic depressives (median, 104.6 mg.; range, 50-428 mg.) and 53 normal controls (median, 150 mg.; range, 110-195 mg.) and reported that as a group the patients presented evidence of abnormal cholesterol metabolism, 75 per cent of them showing abnormal values for blood cholesterol. In those patients characterized by increased physical or mental activity there was a definite tendency to a low cholesterol; where there was decreased mental or physical activity, or both, there was a tendency for values to be above or normal. No relationship was observed between blood cholesterol and clinical types, *e. g.*, pyknics, asthenics, etc.

Chlorides.—Chlorides are found in all the tissues of the organism. The chloride ion is the major anion of the blood. The chloride value of the blood plasma estimated as sodium chloride is from 570 to 620 mg. per 100 cc. and from 99 to 108 milliequivalents per liter.

Some investigators have reported normal blood chloride values for the manic depressives, as shown in Table 7.

Not all investigators, however, have reported normal values. Delaville and Tscherniakofsky (15) in 1929 tested 11 anxious and depressive psychotics and found in most cases chloride values in whole blood and in plasma that were increased above normal. The

TABLE 7.

CHLORIDES: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. of NaCl per 100 cc. blood.)

Investigator.	Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Weston (90)	1920	Potassium thiocyanate titration	10	cyclothymics	494.0	...	...	480 –510	Normal.
Newcomer (55)	1922	.....	5	cyclothymics	....	...	...	.....	Normal.
Bowman (8)	1923	Whitehorn	19	13 depressives	494	51.3	10.4	445 –616	Normal.
				6 manics	492	27.4	5.6	461 –523	Normal.
					493.4	44.8	9.1		
Henry and Mangam (29)	1925	Whitehorn	89	55 depressives	.....	...	...	....	Normal.
				34 manics	....	...	...	.....	
Appel, Farr, and Hodes (2)	1931	Wilson-Ball	64	25 manics	103.3	5.5	5.3	91.7–112.7	Normal. (Chlorides expressed as milliequivalents of NaCl per liter plasma.)
				19 depressives	102.7	4.6	4.5	99.1–111.7	
				20 mixed	103.0	2.5	2.4	98.9–107.5	
					103.0	5.1	5.0		

manic depressives 34.0 per cent; in cholesterol for normals 3.3 per cent, for manic depressives 10.6 per cent; in lipoid phosphorus for normals 2.4 per cent, for manic depressives 11.5 per cent. The investigators concluded that in certain manic depressives there was a disturbed lipoid metabolism.

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(Results in mg. of NaCl per 100 cc. blood.)

Investigator.	Date.	Method.	No. of subjects.	Kind of subjects.	Mean.	S.D.	Coeff. of variation.	Range.	Comments or conclusions.
Weston (90)	1920	Potassium thiocyanate titration	10	cyclothymics	494.0	...	...	480 –510	Normal.
Newcomer (55)	1922	.....	5	cyclothymics	....	...	...	.....	Normal.
Bowman (8)	1923	Whitehorn	19	13 depressives	494	51.3	10.4	445 –616	Normal.
				6 manics	492	27.4	5.6	461 –523	Normal.
					493.4	44.8	9.1		
Henry and Mangam (29)	1925	Whitehorn	89	55 depressives	....	...	...	....	Normal.
				34 manics	....	...	...	.....	
Appel, Farr, and Hodes (2)	1931	Wilson-Ball	64	25 manics	103.3	5.5	5.3	91.7–112.7	Normal. (Chlorides expressed as milliequivalents of NaCl per liter plasma.)
				19 depressives	102.7	4.6	4.5	99.1–111.7	
				20 mixed	103.0	2.5	2.4	98.9–107.5	
					103.0	5.1	5.0		

amount of chloride generally decreased with improvement in the patient's mental health.

Variability.—When the variability of the blood constituents is studied over an extended period of time, and when the variability is compared with that of normal controls, some valuable information about the cyclothymic is obtained. "Many factors enter into the total variation and, if the data have been collected adequately, it has been possible by the technique of the analysis of variance to break up the total variation into those parts which are contributed by fluctuations *within* the individuals, by differences *between* individuals, by seasonal trends, by technical error, and by other trends" (53). Studies of the manic depressive have tended to reveal both an intra-individual and an inter-individual variability that is greater than that shown by normal controls. Furthermore, there appear to be indications of a slightly greater variability in the personal constants of the depressive when compared with that of the manic patient.

Relatively few studies that relate to the intra-individual variability of the manic depressive have been made because of the difficulty of obtaining sufficient repetitions of a test on each patient. Raphael and Parsons (69) in 1921 studied the sugar tolerance of 11 cyclothymics and found tolerance curves differing among themselves in the hypomanic and depressed phases. Tómasson (84) in 1924 tested a group of manic depressives and found that some patients gave different blood calcium values depending on the stage of the cycle. Lockwood (43) in 1932 studied a group of 17 manic depressives and found variations in hyperglycemic index and in cholesterol by repeated tests made on the same individual at different phases. Marshall (49a) in 1939 after studying the sugar tolerance of four alternating cyclothymics at various phases reported that the curve varied with the phase, that is, depressive phases showed alimentary hyperglycemia with delay, while quiescent or mild manic phases showed curves approximately normal.

The majority of studies of the variability of the cyclothymic relate to inter-individual variability. Table 8 lists the results of several studies and shows how manics, depressives and normal controls differ with relation to standard deviation, coefficient of variation and range in the various blood constituents. A glance at the table reveals the fact that in the majority of the studies

TABLE 8.

VARIABILITY: SUMMARY OF FINDINGS WITH MANICS, DEPRESSIVES AND NORMAL CONTROLS.

Blood constituent.	Investigator.	Date.	Method.	No. of subjects.	Kind of subjects.	S.D.	C.V.	Range.	Comments or conclusions.
Blood sugar	Weston (89)	1916	Myers-Bailey	10	6 hypomanics	...	...	115.0 –122.5	Individual differences quite considerable.
					3 depressives	...	...	102.5 –137.5	
					1 interval	...	...	140.0	
Blood sugar	Lorenz (46)	1922	Lewis-Benedict	17	10 manics	...	...	100 –126	
					7 depressives	...	...	82 –122	
Blood sugar	Bowman (8)	1923	Folin-Wu	101	69 depressives	17.8	16.3	65 –166	
					28 manics	15.7	15.1	74 –133	
					4 mixed	11.6	11.5	93 –121	
						17.0	15.8		
Blood sugar	Barrett and Serre (6)	1924	Folin-Wu	20	5 manics	...	...	105 –131	
					15 depressives	...	...	82 –132	
Blood sugar	Looney (44)	1924	Folin-Wu	41	30 depressives	...	...	72 –126	
					11 manics	...	...	81 – 99	
Blood sugar	Bowman and Kasanin (9)	1929	Folin-Wu	73	30 manics	14.7	14.1	75 –140	
					34 depressives	17.1	16.7	65 –140	
					3 hypomanics	...	...	95 –115	
					6 mixed	...	...	65 –125	
						15.9	14.8		
				14	controls	7.5	8.2		
Blood sugar	Sacristán (71)	1934	Hagedorn-Jensen	17	6 manics	...	...	90 –133	
					11 depressives	...	...	90 –138	
Alkaline reserve and acidity	Suitsu (80)	1920	Van Slyke-Cullen	54	39 depressives, 15 manics	9.1	14.1	52.75– 75.38	Variability of patients slightly greater than that of normals. (Volumes per cent.)
				14	controls	8.3	13.3	50.20– 71.04	

TABLE 8.—CONTINUED.

Blood constituent.	Investigator. Date.	Method.	No. of subjects.	Kind of subjects.	S.D.	C.V.	Range.	Comments or conclusions.
Alkaline reserve and acidity	Henry and Mangam (29) 1925	Van Slyke-Cullen.	89	55 depressives	...	...	44.3 – 69.1	
				34 manics	...	...	51.9 – 73.0	
				26 manics:				
				alk. reserve.	3.5	15.3	15.2 – 30.7	(Millimoles per liter.)
				total base..	6.7	4.3	143.6 –168.9	(Milliequivalents per liter.)
				19 depressives:				
Alkaline reserve and acidity	Appel, Farr, and Hodes (2) 1931	Van Slyke-Stadie-Ross	65	alk. reserve.	3.6	15.9	14.2 – 31.4	
				total base..	4.0	2.5	149.8 –161.2	
				20 mixed:				
				alk. reserve.	3.0	13.5	17.9 – 27.9	
				total base..	8.6	5.4	146.8 –166.2	
Calcium	Weston and Howard (91) 1922	Kramer-Tisdall	27	10 depressives	1.0	9.5	9.0 – 12.7	Individual variations from the averages were small.
				17 manics	1.2	11.1	9.5 – 12.0	
					0.8	7.6		
				16 manics:				
				calcium	...	...	9.86– 16.55	
				inorganic phosphorus	...	...	3.02– 5.28	
Calcium and phosphorus.	Henry and Ebeling (28) 1926	Clark-Collip	28	12 depressives:				
				calcium	...	...	9.87– 12.50	
				inorganic phosphorus	...	...	2.40– 5.77	
			7	controls:				
				calcium	...	...	7.89– 12.50	
				inorganic phosphorus	...	...	3.54– 4.68	
Calcium	Armstrong and Hood (3) 1927	Clark-Collip	24	15 melancholics	...	...	9.0 – 10.9	
				9 manics	...	...	9.3 – 10.5	
Calcium	Parhon and Werner (60) 1932		13	7 manics	...	...	10.4 – 12.0	
				6 melancholics	...	...	8.7 – 11.2	

TABLE 8.—CONTINUED.

Blood constituent.	Investigator.	Date.	Method.	No. of subjects.	Kind of subjects.	S.D.	C.V.	Range.	Comments or conclusions.
Nitrogenous substances	Frigerio (24)	1921		10	4 manics: nonprotein N	...	...	5 – 22	
					6 melancholics: nonprotein N	...	...	17 – 42	
Nitrogenous substances	Bowman (8)	1923	Folin-Wu	104	70 depressives: nonprotein N	5.9	19.2	21 – 50	
					uric acid (N = 42)	0.68	42.2	0.4 – 3.8	
					30 manics: nonprotein N	5.4	19.1	17.6 – 39.9	
					uric acid (N = 22)	1.33	62.2	0.8 – 6.6	
					4 mixed: nonprotein N	3.5	12.2	25.5 – 33.6	
					uric acid (N = 3)	0.37	19.5	1.5 – 2.4	
					nonprotein N	7.9	26.3		
					uric acid	0.95	52.8		
Nitrogenous substances	Looney (44)	1924	Folin-Wu	41	11 manics: total N	...	...	23.4 – 37.0	
					urea N	...	...	7.5 – 19.4	
					amino-acid N	...	...	5.12– 7.90	
					uric acid	...	...	0.84– 1.18	
					creatinine N	...	...	0.38– 0.75	
					creatine N.	...	...	1.43– 3.66	
					30 depressives: total N	...	...	25.5 – 42.3	
					urea N	...	...	8.1 – 26.1	
					amino-acid N	...	...	4.83– 9.40	
					uric acid	...	...	0.73– 1.47	
					creatinine N	...	...	0.36– 0.67	
					creatine N.	...	...	1.59– 4.05	

TABLE 8.—CONTINUED.

Blood constituent.	Investigator.	Date.	Method.	No. of subjects.	Kind of subjects.	S.D.	C.V.	Range.	Comments or conclusions.
Nitrogenous substances	Barrett and Serre (6)	1924	Folin-Wu	20	15 depressives:				
					total NPN.	...	...	26 – 50	
					urea	...	...	12.0 – 28.7	
					uric acid	...	...	1.2 – 3.3	
					creatinine	...	...	0.9 – 2.0	
					5 manics:				
					total NPN.	...	...	31.9 – 55.0	
					urea	...	...	14.3 – 30.0	
					uric acid	...	...	2.0 – 3.3	
					creatinine	...	...	1.0 – 1.6	
Nitrogenous substances	Henry and Mangam (29)	1925		62	22 manics: urea N	...	...	10.0 – 31.3	
					40 depressives: urea N	...	...	8.3 – 25.0	
Nitrogenous substances	Vuanello (88)	1938		13	7 depressives: amino-acid N	...	...	5.00– 8.00	
					6 manics: amino-acid N	...	...	4.15– 11.00	
Lipoidal substances	Schube (73)	1934	Myers-Wardell	71	33 manics: cholesterol	...	...	50 –160	Seventy-five per cent of the patients showed abnormal values (56 per cent below normal limits—19.6 per cent above normal limits).
					38 depressives: cholesterol	...	...	50 –428	
				53	controls	...	...	110 –195	
Chlorides	Bowman (8)	1923	Whitehorn	19	13 depressives	51.3	10.4	445 –616	
					6 manics	27.4	5.6	461 –523	
						44.8	9.1		
Chlorides	Appel, Farr and Hodes (2)	1931	Wilson-Ball	64	25 manics	5.5	5.3	91.7 –112.7	(Milliequivalents per liter.)
					19 depressives	4.6	4.5	99.1 –111.7	
					20 mixed	2.5	2.4	98.9 –107.5	
						5.1	5.0		

presented the manic depressive is a more variable biochemical entity than the normal control and that, generally, the depressive individuals are apt to show a greater biochemical heterogeneity than the manic ones. For instance, in the study by Bowman and Kasanin (9), both the manic and depressive groups have a statistically reliable greater variability in blood sugar than the control group. As a rule, where the number of subjects is sufficiently large, it is possible to demonstrate statistically the greater variability of the manic depressive.

Summary and Discussion.—In reviewing the foregoing studies, one becomes aware of several significant trends. There appears, in general, to be evidence of a tendency for the manic depressive to have slightly increased blood sugar, for the cases of acute depression and melancholia to be defective in their sugar tolerance, for the manics to have a slightly increased blood calcium, and for the lipoid values to be rather high in the manic depressive. The disturbed carbohydrate and lipoid metabolisms are perhaps the most striking findings.[6]

Whereas some investigators have not been able to find any relation between sugar content and mental state, affect, mood, emotional tone or severity of the disease, others have been able to detect abnormally high values in the cyclothymic, slightly higher values being found in the depressed states than in the excited. The increases noted might be due to any of a great variety of causes. It might be that resistance or emotional reaction to the needle upon being bled might produce in some cases certain physiological changes that would be reflected in increased sugar values. It may well be that, since the increases have been found by some investigators and not found by others, the chronic or late manic depressive becomes physiologically adapted to affective swings. Emotional excesses do not disturb, physiologically speaking, any more than would normal emotional behavior, that is, he has become habituated to a high emotional level and that is his normal reaction. The acute

[6] Lockwood (43) states that a "sustained hyperglycemia and quantitative changes in the blood cholesterol are common findings in the manic-depressive psychosis . . . ." In an effort to determine whether any relationship existed between blood cholesterol and hyperglycemia, he made parallel observations of the hyperglycemic index and blood cholesterol in 17 cyclothymics and found an inverse relationship ($-.81 \pm .05$).

or early manic depressive, on the other hand, might respond physiologically to emotional excesses with the same sympathetico-endocrine reactions that are characteristic of normal individuals under stress of emotions. It is obvious, of course, that high blood sugars induced in normal individuals would no more produce manic-depressive psychosis than would induced emotions. Another cause of normal blood sugars in one study and high blood sugars in another might be, as mentioned above, the error of faulty psychiatric diagnosis and classification. Another might be the fact that individuals exhibiting the same mental disorder may differ greatly in their physiological abnormalities. Still another might be the variability of the manic depressive that would be related perhaps to a defective emotional control, and that in turn to a defective homeostasis (10).[7] In view of the fact that the manic depressive is characterized by phasic alternations in some cases, it can easily be seen that not only is the kind and degree of emotion different in the same patient from time to time (or in different patients at the same time), but the extent of muscular activity also varies. We do know that the manic individual is apt to be more excited and use more muscular reactions than does the depressed individual (in similar fashion, the individual in a manic phase uses more muscular reactions than he does in a depressed phase). It is possible, however, that the muscular and emotional tension may be as great, if not greater, in the depressed as in the manic state. Consequently, with muscular activity metabolism is increased, more energy is used up and more sugar is burned. This may possibly serve to explain why some investigators have found slightly lower blood sugar in the manic cases than in the depressed ones, even though both were abnormally high.

There is striking evidence of disturbed carbohydrate metabolism in the manic depressive, particularly in the depressed cases. In these cases the retardation of the functions of the vegetative

[7] Diethelm (18) has indicated that the occurrence of different phases may be of fundamental importance. He feels that, in certain cases, the somatic findings may vary according to which emotion happens to dominate the clinical picture. As some of these emotions subside and others rise to dominance, the clinical picture may change. He feels that in studying the emotion it is essential to consider the type, depth, acuteness and duration of the emotion and the constitutional setting.

nervous system is particularly marked. Since it has been shown that the sugar tolerance curve bears a close relation to the presence of emotional tension (66) the picture of defective carbohydrate metabolism in manic-depressive psychosis is probably just another symptom, chemical as it were, of a defective emotional control. The greater incidence of disturbed carbohydrate metabolism among the cases of marked depression and anxiety may be related to the fact that the pre-prandial blood sugar in these cases is usually higher than that of the manic cases. Hence the rise in blood sugar and its duration after a test meal in the depressive states is apt to be greater than in the excitable ones. It may well be, however, that it is not the emotion per se that is responsible for the abnormalities of sugar metabolism in these cases, but rather the factors of constitution (71), that is, perhaps a pyknic individual, whether he be cyclothymic or normal, might be predisposed to abnormal sugar tolerance. Some believe that the discrepancies between the results of various writers on the presence or degree of sugar intolerance may be ascribed to some extent to the numerous variables which are present in the available material (36).

The sugar tolerance curve cannot, of course, be used as an instrument in differentiating manic-depressive psychosis from other psychoses or in the differential diagnosis of the various phases of the disorders. The sugar tolerance test, since it is sensitive to physical conditions, such as toxemia, endocrine disorders, etc., and since it appears to be so closely correlated with emotional tension regardless of malady, is unreliable for use in establishing diagnosis or prognosis. The curves of manic-depressive cases are not very different from those of acute dementia præcox. The factors of "variability, irregularity and inconsistency of the reactions to the test and complexity of the factors involved (16, page 208) militate against its use as a differentiating tool."

The tendency for the manics to have slightly increased calcium values may be explained in terms of muscular activity, physical exertion and degree of resistance on the part of the patient to being bled, since it has been found at the Harvard Fatigue Laboratory by H. T. Edwards [8] that physical exercise may produce increases of 10 per cent to 15 per cent, or even 20 per cent, in blood calcium.

[8] Personal communication to authors.

These calcium increases in the manic cannot be held conclusive, since several studies have reported normal as well as low calcium values in manic patients.

Evidence of a disturbed lipoid metabolism appears in the majority of studies. Not only are high pre-prandial cholesterol values usually found in the manic depressive, but abnormal fat tolerance curves have been manifested by these patients after fat test meals. However, it is probable that a disturbed lipoid metabolism, like a disturbed carbohydrate metabolism, is another chemical symptom of defective emotional control, possibly "to a certain extent, due to some imbalance in the triad (endocrine glands, the autonomic nervous system, and the emotions)" (73), since increases in lipoid values have been noted when there was increase in emotional tone regardless of the malady. Thus, the value of lipoid determinations for purposes of diagnosis or prognosis is distinctly limited. Normal, high and low lipoid values have been reported in different studies of the cyclothymic. Not only has the manic depressive shown this great variability, but even in the case of the same state, excited or depressed, high and low values have been variously reported.

Very few studies have been made on the same individual over a long period of time so as to determine the extent of biochemical variability in the manic depressive. In the few studies of this nature which have been carried out, there appeared to be greater variability in pre-prandial sugar, sugar tolerance, alkaline reserve, calcium and cholesterol values than that observed in normal subjects. It appears possible that the great variability [9] of the cyclothymic patient in emotion may be duplicated in the biochemistry of his body fluids. This may all be related directly to an unstable vegetative nervous system and indirectly to an inherently defective homeostasis. It is certain that there is urgent need of more long-section serial studies [10] of the manic depressive over protracted periods of time

[9] Lewis and Hubbard (42) state that ". . . . it is quite obvious, as in all other problems in psychiatry, that we are dealing with individuals who vary much among themselves, in fact so great are these differences and so dependent upon concatenated factors that it is difficult to draw lucid generalizations."

[10] Partridge (61) leaves little unsaid when he states: "The main conclusion for methodology is that not only in the manic-depressive reactions but, in general, in the field of mental disorders, there is a tendency to attempt too much over too broad an area."

before a definite statement can be made as to whether or not there are any biochemical correlates of the psychosis.[11] It is essential that research along this line be undertaken in order that we may know not only how much a manic depressive differs biochemically from the normal, but also how much he himself differs from day to day. Evidence of the slightly greater variability of the depressive over the manic patient points to possible differences in the degree of excitability of the vegetative nervous system that need investigation.

BIBLIOGRAPHY.

1. Adler, H. M., and Ragle, B. H.: A note on the increase of total nitrogen and urea nitrogen in the cerebrospinal fluid in certain cases of insanity, with remarks on the uric acid content of the blood. Boston Med. Surg. Jour., 171: 769-771, 1914.

1a. Ansani, A.: La glicemia differenziale capillare-venosa nei maniaco-depressivi in arresto psicomotorico. Ann. osp. psichiat. di Perugia, 30: 33-46, 1938.

2. Appel, K. E., Farr, C. B., and Hodes, P. J.: The acid-base equilibrium of the blood in psychotic patients. Manic-Depressive Psychosis, Ass'n Research in Nerv. and Mental Dis., pp. 203-216, 1931.

3. Armstrong, E., and Hood, W.: The calcium content of serum in mental invalids. Jour. Ment. Sci., 73: 111-114, 1927.

4. Atkin, J.: Serum calcium in the psychoses. Lancet, 1: 439-440, 1937.

5. Ballif, L., and Gherscovici, I.: L'équilibre acido-basique dans les maladies nerveuses et mentales. Compt. rend. Soc. Biol., 108: 995-996, 1931.

6. Barrett, T. M., and Serre, P.: Blood analysis and sugar tolerance tests in mental disease. Jour. Nerv. and Ment. Dis., 59: 561-570, 1924.

7. Bogen, D.: Blutuntersuchungen der Cyclophreniker mit der biologischen Methode. Ztschr. f. d. ges. Neurol. u. Psychiat., 117: 569-584, 1928.

8. Bowman, K. M.: Blood chemistry in mental diseases. Amer. Jour. Psychiat., 79: 379-408, 1922-3.

9. Bowman, K. M., and Kasanin, J.: The sugar content of the blood in emotional states. Arch. Neurol. Psychiat., 21: 342-362, 1929.

10. Cannon, W. B.: Stresses and strains of homeostasis. Amer. Jour. Med. Sci., 189: 1-14, 1935.

11. Claude, H., Dublineau, J., and Bonnard, M.: Les lipides du sang dans les maladies mentales. Biol. Med., 25: 97-114, 1935.

[11] Diethelm (18) points out that "the mere accumulation of data especially without repetition of tests on the same patient and under varying conditions cannot be interpreted. Investigations of physiologic functions in personality disorders have frequently, therefore, given varying and even contradictory results."

12. Cowie, D. M., Parsons, J. P., and Raphael, T.: Insulin and mental depression. Arch. Neurol. Psychiat., 12: 522-533, 1924.
13. Craig, R. N.: Blood-sugar curves in certain mental disorders. Lancet, 1: 925-927, 1927.
14. Cruickshank, J., and Tisdall, C. J.: The cholesterol content of the serum in mental diseases. Jour. Ment. Sci., 62: 168-173, 1916.
15. Delaville, M., and Tcherniakofsky, P.: Le chlore sanguin au cours de divers syndromes neuro-psychiatriques. Compt. rend. Soc. Biol., 100: 342-343, 1929.
16. Devine, H.: Recent Advances in Psychiatry. Philadelphia, Blakiston Son, 1933, pp. 364.
17. Dide, M., Fages, G., and Bauduin, A.: Glycémie et glycorachie chez les aliénés. Presse med., 32: 425-427, 1924.
18. Diethelm, O.: Influence of emotions on dextrose tolerance. Arch. Neurol. Psychiat., 36: 342-361, 1936.
19. Di Renzo, F.: Ricerche sulla glicemia. Nota III. La glicemia nelle psicosi affettive. Cultura med. mod., 4: 159-166, 1925.
20. Drury, K. K., and Farran-Ridge, C.: Some observations on the types of blood-sugar curve found in different forms of insanity. Jour. Ment. Sci., 71: 8-29, 1925.
21. Duncan, A. G.: The serum cholesterol in mental disorders. Jour. Ment. Sci., 76: 284-291, 1930.
22. Duncan, A. G.: Cholesterol: Its relation to mental disorder. Jour. Ment. Sci., 79: 626-637, 1933.
23. Freeman, W.: Human Constitution. The Problem of Mental Disorder. Pp. 227-233, New York, McGraw-Hill, 1934, 388 pp.
24. Frigerio, A.: Ricerche sull'azoto non coagulabile del sangue in alcune malattie mentali. Riv. di patol. nerv. e ment., 26: 301-331, 1921.
25. Genzel, A.: Über den Blutzuckergehalt bei Psychosen. Monatschr. f. Psychiat. u. Neurol., 55: 327-336, 1923-4.
26. Hackfield, A. W.: Are physiologic disturbances related to the acute psychotic process in the mentally ill? Arch. Neurol. Psychiat., 28: 883-890, 1932.
27. Heidema, S. T.: Blutzuckerbestimmungen bei psychiatrischen und neurologischen Patienten. Ztschr. f. d. ges. Neurol. u. Psychiat., 48: 111-129, 1919.
28. Henry, G. W., and Ebeling, W. W.: Blood calcium and phosphorus in personality disorders. The effects of ultraviolet radiation. Arch. Neurol. Psychiat., 16: 48-54, 1926.
29. Henry, G. W., and Mangam, E.: Blood in personality disorders. Biochemical studies. Arch. Neurol. Psychiat., 13: 743-749, 1925.
30. Heuyer, G.: Cholestérine et acides gras du sang dans les psychoses. Rev. crit. de path. et de therap., 1: 195-197, 1930.
31. Hinsie, L. E., and Katz, S. E.: Treatment of manic-depressive psychoses. Manic-Depressive Psychosis, Ass'n Research in Nerv. and Mental Dis., 679-733, 1931.

32. Jacobi, W.: Cholesterinstoffwechsel und Konstitutionspathologie der Psychosen. Monatschr. Psychiat. u. Neurol., 62: 296-302, 1927.
33. Jarloev, E.: Sur l'équilibre acido-basique du sang humain, étudié dans ses rapports avec diverses affections. Compt. rend. Soc. Biol., 84: 156-158, 1921.
34. Katzenelbogen, S., and Friedman-Buchman, E.: Studies of blood sugar curves in mental disorders. Amer. Jour. Psychiat., 90: 321-343, 1933-34.
35. Katzenelbogen, S., and Goldsmith, H.: The distribution of calcium between blood and cerebrospinal fluid in mental diseases. Amer. Jour. Psychiat., 88: 9-19, 1931-32.
36. Katzenelbogen, S., and Muncie, W. S.: Studies of blood sugar curves in mental disorders. Jour. Nerv. and Mental Dis., 82: 162-168, 1935.
37. Kilduffe, R. A.: The Clinical Interpretation of Blood Examinations. 629 pp., Philadelphia, Lea and Febiger, 1931.
38. Klemperer, E.: Untersuchungen über den Stoffwechsel bei manischen und depressiven Zustandsbildern. II. Veränderungen des Kalzium- und Kalium-spiegels des Gesamtblutes. Jahrb. f. Psychiat. u. Neurol., 45: 32-62, 1926-27.
39. Kooy, F. H.: Hyperglycaemia in mental disorders. Brain, 42: 214-289, 1919-20.
40. Kora, T.: On the phosphorus content of the blood serum of mental patients and of healthy individuals under special mental influences. Jap. Jour. Med. Sci., VIII, 1: 53-70, 1927.
41. Labin, B.: Recherches sur la glycémie chez les aliénés et dans quelques maladies nerveuses. Compt. rend. Soc. Biol., 96: 1172-1174, 1927.
41a. Laursen, L.: Aminolipoid (cephalin) content of plasma in mental disease. Hospital-Tidende, 79: 1227-1231, 1936.
42. Lewis, N. D. C., and Hubbard, L. D.: The mechanisms and prognostic aspects of the manic-depressive-schizophrenic combinations. Manic-Depressive Psychosis, Ass'n Research in Nerv. and Ment. Dis., 539-608, 1931.
43. Lockwood, M. R.: A note on the relationship between the blood cholesterol and hyperglycaemic index in manic-depressive psychosis. Jour. Ment. Sci., 78: 901-907, 1932.
44. Looney, J. M.: A biochemical study of the blood in mental disorders. Amer. Jour. Psychiat., 81: 29-39, 1924-5.
45. Looney, J. M., and Childs, H. M.: The blood cholesterol in schizophrenia. Arch. Neurol. Psychiat., 30: 567-579, 1933.
46. Lorenz, W. F.: Sugar tolerance in dementia præcox and other mental diseases. Arch. Neurol. Psychiat., 8: 184-196, 1922.
47. Ludlum, S. DeW., Taft, A. E., and Nugent, R. L.: The results of certain physicochemical studies in manic-depressive psychosis. Manic-Depressive Psychosis, Ass'n Research in Nerv. and Mental Dis., 374-386, 1931.
48. Mann, S. A.: Blood-sugar studies in mental disorders. Jour. Ment. Sci., 71: 443-473, 1925.

49. Mann, S. A., and Scott, F. L.: Further blood-sugar studies in mental disorders. Contributions to Psychiatry, Neurology, and Sociology, 279-294, Mott Memorial, 1929.

49a. Marshall, J. K.: Disturbances of glucose tolerance and of the acid base equilibrium in manic-depressive insanity. Jour. Ment. Sci., 85: 222-244, 1939.

50. Matz, P. B., and Willhite, O. C.: A study of manic-depressive psychosis among ex-service men. Manic-Depressive Psychosis, Ass'n for Research in Nerv. and Mental Dis., 629-654, 1931.

51. McCowan, P. K., and Quastel, J. H.: Blood-sugar studies in abnormal mental states. Jour. Ment. Sci., 77: 525-548, 1931.

52. McFarland, R. A., and Goldstein, H.: Biochemistry of the psychoneuroses: A review. Amer. Jour. Psychiat., 93: 1073-1095, 1937.

53. McFarland, R. A., and Goldstein, H.: The biochemistry of dementia præcox: A review. Amer. Jour. Psychiat., 95: 509-552, 1938.

54. Mumford, P. B., and Parkin, G. G.: Some observations on the fasting blood-sugar in certain mental states. Jour. Ment. Sci., 69: 330-339, 1923.

55. Newcomer, H. S.: Blood constituents and mental state. Amer. Jour. Psychiat., 78: 609-611, 1921-2.

55a. Northcote, M. L. M.: Somatic changes in the psychoses. Jour. Ment. Sci., 78: 263-287, 1932.

56. Notkin, J., Greeff, J. G. W., Pike, F. H., and Killian, J. A.: Changes in the clinical signs and laboratory findings in various types of psychoses under the influence of subcutaneous administration of oxygen. Amer. Jour. Psychiat., 89: 1271-1303, 1933.

56a. Olmsted, W. H., and Gay, L. P.: Study of blood sugar curves following a standardized glucose meal. Arch. Int. Med., 29: 384-399, 1922.

57. Ornstein, I.: Sur la cholestérinemie chez les aliénés. Compt. rend. Soc. Biol., 93: 1622-1623, 1925.

58. Parfitt, D. N.: The blood-urea in psychotics. Jour. Ment. Sci., 79: 501-510, 1933.

59. Parhon, C., Urechia, C., and Popea, A.: Note sur la lipoidhémie chez les aliénés. Ztschr. f. d. ges. Neurol. u. Psychiat., 7: 1021, 1913.

60. Parhon, C., and Werner, G.: Recherches sur la calcémie, la potassémie et le rapport K: Ca dans les maladies mentales. Ann. méd. psychol., 2: 87-88, 1932.

61. Partridge, G. E.: Personality and the concept "manic-depressive psychosis." Manic-Depressive Psychosis, Ass'n Research in Nerv. and Mental Dis., 405-412, 1931.

62. Pighini, G.: Uber die Menge Cholesterins und Oxycholesterins des Serums bei verschiedenen Formen von Geisteskrankheiten. Ztschr. f. d. ges Neurol. u. Psychiat., 4: 629-44, 1911.

63. Poli, C.: pH e riserva alcalina in malati di mente. Riv. sper. di freniat, 55: 51-69, 1931.

64. Pollock, H. M.: Prevalence of manic-depressive psychoses in relation to sex, age, environment, nativity, and race. Manic-Depressive Psychosis, Ass'n for Research in Nerv. and Mental Dis., 655-667, 1931.

65. Pugh, R.: The alkalinity of the blood in mental diseases. Jour. Ment. Sci., 49: 71-81, 1903.
66. Quastel, J. H.: Biochemistry and mental disorder. Lancet, 2: 1417-1419, 1932.
67. Rachlin, H. L.: A statistical study of benign stupor in 5 New York State hospitals. Psychiat. Quart., 11: 436-444, 1937.
68. Raphael, T., Ferguson, W. G., and Searle, O. M.: Long section blood sugar tolerance study in a case of depression. Arch. Neurol. Psychiat., 19: 120-124, 1928.
69. Raphael, T., and Parsons, J. P.: Blood sugar studies in dementia præcox and manic-depressive insanity. Arch. Neurol. Psychiat., 5: 687-709, 1921.
70. Reid, B.: A biochemical study of the blood and urine in mental disorders. Jour. Ment. Sci., 73: 254-257, 1927.
71. Sacristán, J. M.: El metabolismo hidrocarbonado en la psicosis maniaco-depresiva endógena. Arch. de neurobiol., 14: 691-748, 1934.
72. Schryver, D.: Blutzucker und Schizophrenie. Ztschr. f. d. ges Neurol. u. Psychiat., 87: 179-196, 1923.
73. Schube, P. G.: Blood cholesterol and the manic-depressive psychosis. Jour. Lab. Clin. Med., 22: 240-245, 1936.
74. Sharpe, J. S.: Some observations on lipoid metabolism in mental disorders. Jour. Ment. Sci., 80: 75-81, 1934.
75. Shaw, B. H., and Sharpe, J. S.: The significance of cholesterol in cellular oxidation and its bearing on mental disorder. Jour. Ment. Sci., 77: 53-66, 1931.
76. Siegler, A.: La calcémie dans quelques états pathologique. Compt. rend. Soc. Biol., 93: 1615-1616, 1925.
77. Slight, D., Long, C. N. H., and Salter, R. W.: Plasma lipoids in mental depression. Amer. Jour. Psychiat., 90: 141-152, 1933-34.
78. Stenberg, S.: Psychosis and blood lipoids. Quantitative variation of total cholesterin and total fatty acids in the blood. I. In manic-depressive psychosis. Acta Med. Scand., 71: 558-597, 1929.
79. Ström-Olsen, R.: The blood sugar curve in mental cases. Lancet, 1: 128-130, 1932.
80. Suitsu, N.: Studies on the alkaline reserve of the blood of the insane. Amer. Jour. Physiol., 54: 147-152, 1920-21.
81. Targowla, R., Badonnel, M., and Berman, A.: Sur la cholestérinémie dans les maladies mentales. Encephale, 18: 138-140, 1923.
82. Thomas, C. J.: The acid-base equilibrium in cases of mental disorder. Jour. Neurol. Psychopath., 3: 120-123, 1922-23.
83. Tod, H., and Jones, M. S.: Studies in carbohydrate metabolism in nervous and mental disorders. II. A comparison of the hyperglycemic index and choline esterase activity in anxiety and depressive states. Edinburgh Med. Jour., 44: 46-49, 1937.
84. Tómasson, H.: Psychische Beeinflüssung des Serumcalciumspiegels. Klin. Wchnschr., 3: 2055-2056, 1924.

85. Tómasson, H.: Blodets Elektrolyter og det Vegetative Nervesystem. Levin & Munksgaards, Copenhagen, 1927.
86. Tómasson, H.: Fortgesetzte Untersuchungen über die Elektrolyten des Blutes und das vegetative Nervensystem bei Patienten mit manisch depressiver Psychose. II. Beitrag zu einer physiologisch-chemisch *begründeten Behandlung*. Acta Psychiat. et Neurol., 7: 679-697, 1932.
87. Tsuchiya, S.: Über den Verlauf der Blutzuckertoleranz bei Psychosen. Ztschr. f. d. ges Neurol. u. Psychiat., 91: 23-25, 1924.
88. Vuanello, G.: Il comportamento dei gruppi aminici (NH_2 dei polipeptidi degli aminoacidi e delle amine) nel sangue e nel liquor di alcune gruppi di malati mentali. Rassegna di studi psichiat., 27: 268-280, 1938.
89. Weston, P. G.: Sugar content of the blood and spinal fluid of insane subjects. Jour. Med. Research, 35: 199-208, 1916-7.
90. Weston, P. G.: Analyses of blood of insane patients. Arch. Neurol. Psychiat., 3: 147-150, 1920.
91. Weston, P. G., and Howard, M. Q.: The determination of sodium, potassium, calcium, and magnesium in the blood and spinal fluid of patients suffering from manic-depressive insanity. Arch. Neurol. Psychiat., 8: 179-183, 1922.
92. Whitehorn, J. C.: The blood sugar in relation to emotional reactions. Amer. Jour. Psychiat., 90: 987-1004, 1934.
93. Wigert, V.: Studien über den Zuckergehalt des Blutes bei Psychosen mit depressiven Affekten. Ztschr. f. d. ges Neurol. u. Psychiat., 44: 179-211, 1918-19.
94. Wuth, O.: Der Blutzucker bei Psychosen. Allg. Ztschr. f. Psychiat., 76: 817-818, 1920-21.

AGE OF ONSET OF EPILEPSY.

Differences Between Deteriorated and Non-Deteriorated Patients.*

By HARRY A. PASKIND, M. D., and MEYER BROWN, M. D.

Although there are many reports dealing with the age of onset of epilepsy, we are convinced that such studies have incomplete value because they have been based almost exclusively on institutional patients with epileptic deterioration or other epileptic psychosis. Writers on epilepsy, since they were mainly workers in institutions and colonies, had the opportunity of studying only epileptic patients incarcerated on account of deterioration or other psychosis, and there are good reasons for believing that these constitute only a part, perhaps a small part of persons with this disease. Since the studies of most writers were limited by institution walls the great number of non-deteriorated persons with epilepsy who adjust in the outside world eluded the observation of those who described the age of onset of the disease.

Further, the conclusions of some writers have been beclouded by their inclusion as epileptics many feebleminded persons in whom seizures were a manifestation of developmental anomaly of the brain, while others have included those with old inflammatory or other diseases of the brain giving rise in some cases to localizing signs in addition to convulsive seizures. Thus, it might not be amiss to state that in the literature, cases which are often considered epilepsy are not epileptics at all, but those who have seizures due to some cause other than idiopathic epilepsy.

Our purpose in the present communication is to describe the age of onset of a group of well-adjusted, non-psychotic, non-feebleminded, extramural persons with epilepsy, free from demonstrable organic brain disease and to determine whether these patients differ in age of onset from the institutional, deteriorated patients with epilepsy as given in the literature.

* From the department of nervous and mental diseases, Northwestern University School of Medicine and the Minnie Frances Kleman Memorial Fund.

Statistical formulations of the age of onset of epilepsy may be found in communications by Bouchet and Cazauvieilh,[1] Beau,[2] Leuret,[3] Herpin,[4] Moreau de Tours,[5] Sieveking,[6] Musset,[7] de Cailleux,[8] Reynolds,[9] Echeverria,[10] Hammond,[11] Hamilton,[12] Mendel,[13] Berger,[14] Binswanger,[15] von Tereszkiewicz,[16] Aronsohn,[17] Doran,[18] Gowers,[19] Spratling,[20] Siebold,[21] Turner,[22] Munson,[23] Starr,[24] Ricci,[25] Sarbo,[26] Finkh,[27] Ehrhardt,[28] Lange,[29] Kraepelin,[30] Lüth,[31] Biro,[32] Stoddart,[33] Dana,[34] Ostmann,[35] and Lennox and Cobb.[36]

The material used for the present study consists of the records of 368 adult non-deteriorated patients with epilepsy. Of these, 284 were from the private practice of Dr. Hugh T. Patrick and 84 from the outpatient clinic of Northwestern University Medical School. In order to allow sufficient time for deterioration to occur no patient was accepted for this study unless seizures were present for at least six years. Of these patients 36.0 per cent had had the disease for from 6 to 10 years; 24.8 per cent, 11 to 15 years; 17.1 per cent, 16 to 20 years; 9.1 per cent, 21 to 25 years; 6.3 per cent, 26 to 30 years; 6.7 per cent, over 30 years. Patients with defective mental development and with focal signs of neurologic disorder were not accepted for this study.

A comparison of the age of onset of the non-deteriorated patients in our series with that of similar data given in the literature is shown in Table I. Study of this table discloses some interesting differences. In our series 11.7 per cent of the patients had the onset before the age of five years. With two exceptions, this ratio is lower than that found by any of the other writers. Thus Ehrhardt found that in his series 43.9 per cent had the onset before the age of five years; Doran found this in 46 per cent, Siebold in 32.8 per cent and Ostmann in 32.7 per cent. Kraepelin found that 12.4 per cent of his patients had the onset before the age of five; this figure approximates ours the most closely. Lennox and Cobb in a series of non-institutional patients found that 14 per cent had the onset under five years of age, a figure 2.3 per cent higher than ours. Similar ratios as given by the other writers vary from a minimum of 14 per cent to a maximum of 29 per cent. The two exceptions are in the findings of Reynolds and Hammond, who found the onset under 10 years of age in 11.0 per cent and 10.5 per cent, respectively; but these findings are so at variance with

TABLE I.

PERCENTAGE OCCURRENCE OF THE AGE OF ONSET OF EPILEPSY AS GIVEN IN THE LITERATURE AND AS NOTED IN THE PRESENT SERIES.

Author	Number of Cases	0–5yr.	6–10	11–15	16–20	21–25	26–30	31–40	41–50	51–60	61–70	71–80	Under 10 yr.	Over 20 yr.	Over 40yr.
Bouchet and Cazauvieilh	66	27.2	16.7	16.7	15.2	7.6	6.1	4.5	4.5	1.5	0.0		43.9	24.2	6.0
Beaue	223	59.6				12.5		5.2	6.8	1.7	0.4			26.5	8.6
Leuretee	100	14.0	5.0	24.0	18.0	16.0	8.0	7.0	7.0	1.0	0.0		19.0	39.0	8.0
Herpin	68	25.0	2.9	20.6	19.2	16.2		2.9	4.4	2.9	1.5	4.4	27.9	32.3	13.2
Moreau de Tours**	995	39.5		36.6		11.2		5.9	5.1	1.3	0.4		39.5	23.9	6.8
	301	18.0	14.9	26.2	16.3	6.6	5.3	6.3	4.7	1.0	0.7		32.9	24.6	6.4
Sieveking	56	20.7	8.6	22.4	17.2	5.2	3.5	8.6	12.1	1.7	0.0		29.3	31.1	13.8
Musset	307	38.8		34.8		10.8		6.8	6.2	1.6	1.0		38.8	26.4	8.8
de Cailleux	136	27.2				27.9		24.3	13.2	3.7	3.7			72.8	20.6
Reynolds	172	11.0		61.6		27.4							11.0	27.4	
Echeverria	262	24.0		45.0		31.0									
Hammond	572	10.5		87.5		38.0							10.5	32.0	
Hamilton	183	14.2		38.8		22.4		21.9		2.7			14.2	47.0	
Mendel	904	94.2								5.8					5.8
Berger	91	24.2	15.4	14.3	12.0	15.4		15.4	1.1	1.1	0.0	1.1	39.6	34.1	3.3
Binswanger	132	18.9	16.7	22.7	18.9	8.8	4.6	9.9	1.5	0.0	0.0		35.6	22.8	1.5
Von Terzeskiewicz	221	24.4	17.2	19.0	34.4	5.0							41.6	5.0	
Aronsohn	508	82.0				18.0								18.0	
Doran	1217	48.0		38.0		16.0							48.0	16.0	
Gowers	3002	27.5		46.5		15.5		6.3	2.4	1.3	0.5		27.5	26.0	4.2
Spratling		29.2	19.2	24.5	13.7	5.4	3.3	4.0	1.0	0.6	1.8		48.4	16.1	3.4
Siebold		32.8	13.4	22.7	14.1	6.0	3.2	2.9	1.6	9.5	0.3	0.1	46.2	23.6	11.5
Turner		17.0	14.3	28.1	18.6	6.9	5.3	5.8	2.6	1.0	0.4		31.3	22.0	4.0
Munson	2608	28.0	18.8	24.1	13.1	5.1	3.7	4.0	2.0	0.7	0.5		46.8	16.0	3.2
Starr		18.1	13.3	20.1	17.8	12.3	6.6	8.4	3.1	0.9	0.3		31.4	31.6	4.3
Ricci		14.0	17.8	15.5	12.7	7.1	5.1	1.8	1.0	0.5	0.8		31.8	16.3	2.3
Sarbo	180	76.0				24.0								24.0	
Finckoh		40.5		36.7		20.8							40.5	20.8	
Ehrhardt	321	43.9	18.1	19.6	10.9	3.4	1.9	1.8	0.6	0.3	0.0		62.0	7.5	0.9
Lange		53.4		35.6		11.0							53.4	11.0	
Kraepelin		12.4	10.9	18.9	20.4	12.6	12.1	8.3	2.8	0.8	0.8	0.1	23.3	37.5	4.5
Lüth		72.6				27.4								27.4	
Biro		69.0				31.0								31.0	
Stoddart		76.0				16.0		9.0						25.0	
Dana		15.7	76.0			26.0								26.0	
Ostmann	392	32.7	12.2	11.8	14.8	6.9	7.7	7.1	4.3	2.0	0.5		44.9	23.5	6.8
Lennox and Cobb*	7350 deteriorated	22.0	17.0	18.0	13.0	6.0	4.0	4.5	1.3	3.0	0		46.0	16.8	1.8
	944 non-deteriorated	14.0	13.0	20.0	17.0	10.0	10.0	12.0	4.5	2.0	0.5		27.0	39.0	7.0
Present Series	368	11.7	13.9	18.7	16.0	10.3	11.2	10.3	7.1	0.8	0		25.6	39.7	7.9

* Twenty-five percent of these patients had a hemiplegia.
** Divisions here are 0–4 years, 5–9 years, 10–14 years, etc.

those of the other writers that their validity is open to serious question.

Thus, it seems reasonable to believe that patients with epilepsy who do not deteriorate have the onset under five years of age less frequently than those who do deteriorate.

In our series 25.6 per cent had the onset before the age of 10 years. Table I shows how this ratio compares with that of the other writers. Leuret, Reynolds, Hammond, Hamilton and Kraepelin found the onset under 10 years of age in fewer instances than we did (19.0, 11.0, 10.5, 14.2, 23.3 per cent respectively). All others found a greater proportion than we did of patients with onset before the age of 10 years. In some instances these differences are very large; thus, in Ehrhardt's series 62.0 per cent had the onset before the age of 10 years, and in Lange's series 53.4 per cent. Lennox and Cobb in their non-deteriorated patients found that 27.0 per cent had the onset before the age of 10 years, a ratio only 1.4 per cent higher than ours.

It appears then that another difference between deteriorated and non-deteriorated patients with epilepsy is that a smaller proportion of the latter have the first seizure before the age of 10 years.

In our series 39.7 per cent of the patients had the first seizure after the age of 20. How this compares with similar data as given by other writers is shown in Table I. In only two instances were there reports of a greater percentage of patients having the onset after the age of 20 (de Cailleux, 72.8 per cent and Hamilton, 47.0 per cent). All the other writers found a smaller proportion of patients with the onset after 20 years of age than we did, in some instances these differences were very great (von Tereskiewicz, 5.0 per cent, Ehrhardt, 7.5 per cent). Lennox and Cobb in non-deteriorated patients found that 39.0 per cent had the onset after 20 years of age, a ratio very close to ours.

We believe that the above data afford evidence to support a view that an onset after 20 years of age is more common in epileptic persons without deterioration than in those who deteriorate.

In our series 7.9 per cent of the patients had the first seizure after 40 years of age. Table I shows how this ratio compares with that of other writers. Some (Beau, Leuret, Herpin, Sieveking, Musset, de Cailleaux) found a higher proportion of cases than we did with the onset after 40 years of age; these were mainly

the older writers. The majority, however, found that a smaller proportion of their cases had the first seizure after the age of 40.

SUMMARY AND CONCLUSIONS.

Descriptions of epilepsy in the literature have been written mainly by men working in institutions who had access only to institutionalized patients. Consequently, reports on epilepsy are based almost exclusively on deteriorated, incarcerated patients. Since most writers had no opportunity of observing non-deteriorated, extramural patients with epilepsy these were not included in their studies. Their inclusion, however, is important because there are good reasons for believing that deteriorated patients constitute only a small part of the number of persons with epilepsy.

In this study the age of onset of 368 adult non-deteriorated patients with epilepsy was determined and compared with similar data for institutional deteriorated patients.

The trends observed were as follows:

1. Fewer non-deteriorated patients have the onset before five years of age.

2. Fewer non-deteriorated patients have the onset before 10 years of age.

3. More non-deteriorated patients have the onset after 20 years of age.

4. More non-deteriorated patients have the onset after 40 years of age.

It is concluded that deteriorated patients with epilepsy differ from the non-deteriorated ones in that there is a tendency to later onset in the non-deteriorated patients.

BIBLIOGRAPHY.

1. Cited by Binswanger, Reference 15.
2. Beau: Archiv. gen. de med., 2d Series, 11: 328, 1836.
3. Leuret: Recherches sur l'Epilepsie. Archiv. gen. de med., 4th Series, 2: 32, 1843.
4. Herpin, T.: Du Pronostic et du Traitement Curatif de l'Epilepsie. Paris, J. B. Balliere, 1852.
5. Cited by Echeverria, Reference 10.
6. Sieveking, E. H.: On epilepsy and epileptiform seizures. London, John Churchill, 1858.
7. Cited by Echeverria, Reference 10.
8. Cited by Echeverria, Reference 10.

9. Reynolds, J. R.: System of medicine. London, The MacMillan Company, 1868.
10. Echeverria, M. G.: Epilepsy. New York, William Wood and Company, 1870.
11. Hammond, W. A.: Diseases of the nervous system. New York, D. Appleton and Company, 8th Edition, 1890.
12. Hamilton, A. M.: Nervous diseases. Philadelphia, H. C. Lea, 1878.
13. Mendel: Epilepsy tardive. Rev. Neurolog., 1: 668, 1893.
14. Cited by Binswanger, Reference 15.
15. Binswanger, O.: Die Epilepsie. Vienna, Alfred Hölder, 1899.
16. Cited by Binswanger, Reference 15.
17. Aronsohn, O.: Cited by Berkeley, H. J.: A treatise on mental diseases. New York, D. Appleton and Company, 1900.
18. Doran, R. E.: A consideration of the hereditary factors in epilepsy. Am. J. Insanity, 60: 61, 1903.
19. Gowers, W. R.: Epilepsy and other chronic convulsive disorders, 2d Edition. London, J. and A. Churchill, 1901.
20. Spratling, W. P.: Epilepsy and its treatment. Philadelphia, W. B. Saunders, 1904.
21. Siebold: Statistische Beiträge zur Aetiologie der Epilepsie. Psychiat. neurol. Wchnschr., 8: 1147, 1906.
22. Turner, W. A.: Epilepsy, a study of the idiopathic disease. New York, The MacMillan Company, 1907.
23. Munson, J. F.: Death in epilepsy. Med. Rec., 77: 58, 1910.
24. Starr, M. A.: Organic and functional nervous disorders. Philadelphia, Lea and Febiger, 1913.
25. Cited by Kraepelin, Reference 30.
26. Sarbo, A.: Der heutige Stand der Pathologie und Therapie der Epilepsie. Berlin, Urban und Schwarzenberg, 1905.
27. Finckh, J.: Beiträge zur Lehre von Epilepsie. Arch. f. Psychiat., 39: 820, 1904-1905.
28. Ehrhardt, A.: Ein statistischer Beitrag zur Entstehung der Epilepsie. Allg. Ztschr. Psychiat., 70: 937, 1913.
29. Cited by Kraepelin, Reference 30.
30. Kraepelin, E.: Psychiatrie. Leipzig, J. A. Barth, 1913, Volume 3.
31. Cited by Hartman, F., and di Gaspero, H.: Epilepsie in Lewandowsky, M.: Handbuch der Neurologie, Vol. V, Berlin, Julius Springer, 1914.
32. Cited by Hartman, F., and Gaspero, H. See Reference 31.
33. Stoddart, W. H. B.: Mind and its disorders, 3d Edition. London, H. K. Lewis and Company, 1919.
34. Dana, C. L.: Diseases of the nervous system, 10th Edition. New York, Wm. Wood and Company, 1925.
35. Ostmann: Gesammelte Notizen über unsere Epileptiker. Allg. Ztschr. Psychiat., 89: 397, 1928.
36. Lennox, W. G. and Cobb, S.: The non-institutional epileptic. Assoc. for Research in Nervous and Mental Disease, 7: 358, 1931.

FREQUENCY OF SEIZURES IN EPILEPSY.

Differences Between Deteriorated and Non-Deteriorated Patients.*

By HARRY A. PASKIND, M.D., and MEYER BROWN, M.D.

Discussions of the frequency of seizure in epilepsy are based almost entirely on studies of institutional patients. Writers of textbooks and monographs on epilepsy were mainly institution men who found institution material convenient for study. Here there are concentrated large numbers of patients with convulsive disorder; their observation is rendered easy because many patients and records are available for study over a long period of time. It must be remembered, however, that most persons with epilepsy are institutionalized not because they are epileptic, but because they have developed mental disturbances that make incarceration imperative. There are, however, many patients with epilepsy who never reach an institution, they remain without mental change, and adjust in the outside world on the same level as their non-epileptic fellows. There are good reasons for believing that these outnumber the institutional patients. The purpose of the present communication is to compare the frequency of seizures in the two groups; that is, to determine if the seizures have a different frequency in the deteriorated, institutional patients than they have in the non-deteriorated, well-adjusted epileptic of the outside world.

Data regarding the frequency of seizures in epilepsy have been made available by Beau,[1] Leuret,[2] Herpin,[3] Delasiauve,[4] Reynolds,[5] Starr,[6] Gowers,[7] Spratling [8] and Turner.[9]

The material used for the present study consists of the records of 317 adult, non-deteriorated, extramural patients with epilepsy. Of these, 238 were from the private practice of Dr. Hugh T. Patrick and 79 from the outpatient clinic of Northwestern University School of Medicine. In order to allow time for deterioration to occur no patient was accepted for this study unless he had

* From the department of nervous and mental diseases, Northwestern University School of Medicine and the Minnie Frances Kleman Memorial Fund.

had seizures for at least six years. Of these patients, 37.2 per cent had had the disease for from 6 to 10 years; 24.4 per cent, 11 to 15 years; 16.8 per cent, 16 to 20 years; 9.0 per cent, 21 to 25 years; 6.1 per cent, 26 to 30 years; and 6.5 per cent over 30 years. Patients with defective mental development and with focal signs of neurologic disorder were not accepted for this study. The seizures studied were in patients who had had no treatment or only inadequate, irregular treatment.

TABLE I.

PERCENTAGE-FREQUENCY OF MAJOR ATTACKS AS GIVEN BY SOME AUTHORS AND AS NOTED IN THE PRESENT SERIES.

Interval Between Attacks	Beau	Leuret	Herpin	Delasiauve	Reynolds	Starr	Gowers	Spratling	Turner	This Series
Less than 1 day	10.9	18.3	20.0	46.3	17.2	20.4	11	56.0	12.0	5.7
1 day to 1 week	12.7		33.3		1.6	20.4	27		30.3	8.8
1 week to 2 weeks	14.1	15.9	15.0	34.6	7.8	5.1	15	12.3	30.3	12.0
2 weeks to 1 month	17.0	36.6			38.9	16.1	24	22.0		25.6
1 to 2 months	26.9	20.7	25.0	19.1	17.2	18.3	10	6.5	20.5	12.9
2 to 4 months	5.2	4.9			14.0	11.7	8			20.5
4 to 6 months	3.3	0.0			1.6	0.0	2	2.2	6.0	6.0
6 to 12 months	9.0	1.2			1.6	1.5	2			2.8
Over 1 year	0.9	2.4	6.7			6.5	1			5.7
Irregular					3.1					

Comparison of the frequency of major seizures in our series of non-deteriorated patients with that of other writers is shown in Table I.

In our series 5.7 per cent had grand mal seizures at intervals of less than one day. This ratio is significantly smaller than similar figures given by other writers. Thus Starr found such intervals in 20.4 per cent of his patients, Herpin in 20.0 per cent and Reynolds in 17.2 per cent. The figure which most closely approximates ours is that of Beau; 10.9 per cent of his patients had seizures at intervals of less than one day.

In the present series 14.5 per cent of the patients had major seizures at intervals of one week or less. Table I shows how markedly this figure contrasts with similar ratios given by other writers. Herpin found that 53.3 per cent of his patients had major seizures at such frequency; Delasiauve found this in 46.3 per cent; Starr in 40.8 per cent, Spratling in 56.0 per cent, Turner in 42.3 per cent. The figure that most closely approximates ours in this respect is that of Leuret who found that 18.3 per cent of his patients had seizures at intervals of one week or less.

In the present series of non-deteriorated patients grand mal seizures occurred at intervals of one month or less in 52.1 per cent of cases. This is in marked contrast to similar figures of the other writers (Table I). Thus in Spratling's cases 90.3 per cent had seizures at such frequency; Delasiauve found this in 80.9 per cent; Gowers in 77 per cent, Leuret in 70.8 per cent, Turner in 72.6 per cent, Herpin in 68.3 per cent, Reynolds in 62.5 per cent, Starr in 62.0 per cent and Beau in 54.7 per cent. The latter figure most closely approximates ours.

In our series 47.9 per cent had major seizures at intervals greater than one month. This ratio is greater than that given by any of the other writers. Beau found that 45.3 per cent of his patients had major seizures at such intervals; amongst the other writers this value ranges from 8.7 per cent to 38.0 per cent.

Thus, it appears that extramural, non-deteriorated patients have major seizures less frequently than do the deteriorated patients in institutions.

In a thorough search of the literature we could find only one statistical statement regarding the frequency of petit mal attacks; Gowers [7] stated that in half the cases the attacks occurred daily. In our series the frequency of petit mal attacks could be determined in 137 cases. They occurred at intervals not exceeding one day in 21.2 per cent of cases. In the remainder of the present series they occurred as follows:

Intervals of more than one day, not exceeding one week, in 22.6 per cent.

Intervals of more than one week, not exceeding two weeks, in 17.5 per cent.

Intervals of more than two weeks, not exceeding one month, in 18.2 per cent.

Intervals of more than one month, not exceeding two months, in 5.8 per cent.

Intervals of more than two months, not exceeding four months, in 6.6 per cent.

Intervals of more than four months, not exceeding six months, in 2.2 per cent.

Intervals of more than six months, not exceeding 12 months, in 1.5 per cent.

Intervals of more than 12 months, in 4.4 per cent.

CONCLUSIONS.

The frequency of seizures has heretofore been reported mainly in institutional deteriorated patients. A study of the frequency of seizures in a group of 317 non-institutional, non-deteriorated patient with epilepsy discloses that seizures are significantly less frequent in such a group than they are in deteriorated institutional patients.

BIBLIOGRAPHY.

1. Beau: Archives gen. de. med., 2d Series, 11: 328, 1836.
2. Leuret: Rechérches sur l'épilepsie. Archiv. gen. de. med., 4th Series, 1843, ii: 32.
3. Herpin, T.: Du pronostic et du traitément curatif de l'épilepsie. J. B. Balliere, 1852.
4. Delasiauve, L. J. F.: Traité de l'épilepsie. Paris, Victor Masson, 1854.
5. Reynolds, R.: Epilepsy. London, John Churchill, 1861.
6. Starr, M. A.: Familial forms of nervous disease. New York, William Wood and Company, 1890.
7. Gowers, W.: Epilepsy and other chronic convulsive disorders, Ed. 2. London, J. and A. Churchill, 1901.
8. Spratling, W. P.: Epilepsy and its treatment. Philadelphia, W. B. Saunders Co., 1904.
9. Turner, W. A.: Epilepsy: A study of the idiopathic disease. New York, The Macmillan Co., 1907.

EXPERIENCES WITH PSYCHIATRIC DEPARTMENTS IN THE GENERAL HOSPITALS OF OMAHA.*

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Pharmacologic shock therapy of the psychoses, as developed in this country during the past two years, has brought into sharper relief the question as to whether the psychiatric department should be recognized as a proper and desirable component of the general hospital organization. For various reasons, there has been much opposition in the past to the inclusion of the acute psychoses among the admissions to the general hospitals. At the present time, many of these reasons no longer hold good, and a revaluation of the situation is in order.

In Omaha, with nine general hospitals, there are five that are provided with psychiatric departments. The largest of these, in the Douglas County Hospital, has at present 160 patients. The other four, associated with private hospitals, have a total normal capacity of 106 beds. The respective hospital managements are emphatic in their support of the departments.

The development of the psychiatric departments in Omaha has derived from several sources. As the location of two Class A medical schools, associated respectively with the University of Nebraska and Creighton University, Omaha has long been a medical center, and the medical profession of the city have drawn their clientele from the surrounding states. The specialties of neurology and psychiatry have been represented since 1901, and there are now eight physicians who limit their practice to this type of work, all of whom are on the faculties of one of the two schools. Inasmuch as psychiatric consultation and treatment constitute a large part of the neuropsychiatrist's activities, the need for special hospital facilities for the treatment of the acute psychoses has been obvious. Acute deliria, depressions and early schizophrenias were being treated in private rooms in the general hospital. Needless to

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say, accidents occurred with a frequency sufficient to make psychotic patients a decided risk for the hospital, and the psychiatrist felt his patients were unwelcome.

Before entering private practice in Omaha in 1910, I had had an experience in connection with psychiatric nursing that had convinced me that psychiatric cases could be cared for advantageously in the general hospital, and that there existed no incompatibility between psychiatric work and the general hospital medical and nursing disciplines. In 1906, after several years of state hospital medical work, I was appointed superintendent of the Norfolk State Hospital at Norfolk, Nebraska, to reconstruct a hospital service badly disorganized by inexperienced political appointees.

As a part result of the free hand given me, I was able to establish a nursing department reform in making, for the first time in the state hospitals of Nebraska, the superintendent of nurses an official of the hospital in charge of all nurses and attendants. A training school was established, women nurses placed on male wards and a medical and nursing service was developed for the acute cases, which, I am sure, equaled in many respects our modern psychiatric clinic prior to the days of insulin and metrazol.

Miss Helen C. Sinclair, the superintendent of nurses, a woman of great intelligence and force of character, who had her first experience in state hospital service at Norfolk, later became widely known in connection with her work with the Department of Hospital Service of the National Committee for Mental Hygiene. This was after Nebraska politics had again stepped in with the election of a new governor, and a two year old, smoothly working organization had been dissolved. Dr. H. Douglas Singer, who shortly before had come to Omaha from London, accepted the position of first physician and began his state psychiatric experience at Norfolk.

It was the memory of the hospital department of the Norfolk State Hospital in the years 1907-08 that had much to do with my conviction that there was a place in the general hospital for psychiatric work. In 1924, St. Joseph's Hospital, a large Catholic hospital of some 400 beds, established a psychiatric department of 29 beds in a separate wing erected at a cost of $70,000.00. Dr. G. W. Dishong and Dr. Ernest Kelley, of Creighton Medical College, and Dr. H. A. Wigton and myself, of the University of Nebraska Medical College, were at that time taking psychiatric

patients to St. Joseph's Hospital, and were in part responsible for this new development. Dr. Dishong and Dr. Kelley had been resident physicians at the Norfolk State Hospital during the years 1907-08, and were familiar with Miss Sinclair's work. Miss Sinclair was well known to the Sisters of St. Joseph's Hospital, and contributed her influence in a most helpful way.

This institution remained the only psychiatric department in Omaha until 1928, when the Methodist Hospital, capacity 160 beds, remodeled a floor of one of the wings at a cost of $2400.00, and provided 13 beds for the psychiatric care of women. No male beds were included, owing to lack of space.

About the same time, the German Lutheran Church bought the Wise Hospital, capacity 75 beds, and remodeled an adjacent nurses' home, making of it a psychiatric unit with a bed capacity of 34.

The fourth psychiatric department was opened January 1, 1937, when the Clarkson Hospital, an established Episcopalian hospital, having bought and remodeled the five-story Lister Hospital, and acting on the initiative of Dr. A. E. Bennett, also reconstructed two wings of the second floor for psychiatric purposes at a cost of $20,000.00. A bed capacity of 30 was provided, equally divided as to sex. This institution has the advantage of more recent ideas of construction, such as air-conditioned, sound-proof seclusion rooms, which in the other hospitals are not fully sound-proof. On the other hand, it has, with the Lutheran Hospital, the disadvantage of its location in a downtown district, without the adjacent parks of the Methodist and St. Joseph's Hospitals.

The general principles of arrangement are the same in all, the combination of a hallway with single rooms, two-bed rooms, dormitories on either side, one or two continuous tubs, showers, recreation and occupational rooms and sun-rooms which serve also as dining rooms.

The details of construction of a psychiatric department vary according to the amount of money available and according to whether a new wing or building is to be erected or present space remodeled. The bed capacity, or size, naturally depends on the size of the hospital and the demand for beds, which in turn depend upon the population factor and the number of local psychiatrists. The education of the public along psychiatric lines is also a factor in determining demand. If a large enough group of psychiatrists

should choose to work together it would be possible to erect a substantial building either separately or in coordination with a general hospital, in which every detail of convenience and safety could be amply provided. However, it is more in accord with the purpose of this paper to stress the feasibility of remodeling available space in a general hospital at a moderate cost to provide adequate and serviceable psychiatric facilities for the attending staff.

For instance, the Omaha Methodist Hospital, with 160 bed capacity, equipped 13 beds for women at a cost of $2400.00. This included an automatically controlled continuous tub, one sound-proof seclusion room, one observation room, a five-bed dormitory, one two-bed room, and six private rooms, nurses' service and chart-room, bathroom and toilet room, and a sun-porch which serves as a combined recreation and work room. Meals are served either in the rooms or at a common table in the sun-porch.

The windows of the department are very tall, and surmounted by ventilating transoms. The windows are not barred, but the lower sash is fitted with a steel cross-bar grill, the glass-filled meshes of which measure 7 x 6 inches. The sashes are so arranged that each sash can be raised or lowered eight inches only, thus, with the transom, providing ample ventilation as well as safety. The whole ward has operated efficiently under the control of a trained psychiatric nurse, with the assistance of pupil nurses of the second and third year classes. It is now accepted as a working unit of the hospital, on a par with pediatrics or the obstetrical service.

The Clarkson Hospital, with a total capacity of 150 beds, has practically the same arrangement, except that two wings of a double "L" floor plan were remodeled, providing 15 beds each for men and women. Two air-conditioned, fully sound-proofed rooms were constructed on each wing with separate toilet arrangements.

The psychiatric department of the Douglas County Hospital, with its 150 to 160 patients, is housed in a modern, well-equipped building which was erected in 1928. Here there had been previously very primitive quarters, with a total capacity of about 30 beds, divided into two wards. Owing to ancient construction and board floors, they were fertile breeding places for vermin. They were occupied by psychotic patients, alcoholics and drug addicts, who

were under observation pending commitment to the state institutions.

About 1920, owing to economy programs of state administrations, the state hospital building program fell badly behind requirements. Omaha and Douglas County were unable to obtain admission for their patients, and the number of patients cared for at the County Hospital has since steadily increased. With an average yearly number of 300 patients coming before the Board of Commissioners of Insanity, approximately 50 would be committed to the state hospitals. The rest had to be cared for at the County Hospital. To meet this need, the present psychiatric department was constructed.

As physician member of the Board for the past 23 years, I have been largely responsible during that time for the medical care of the patients treated in the Douglas County Psychiatric Department. There has been steady progress in nursing and medical care, with graduate nurses and attendants and a resident psychiatrist. Since October, 1936, treatment by insulin and metrazol has been actively employed.

Mention perhaps should be made here of the psychiatric liaison ward of the University Hospital of the University of Nebraska Medical School where patients of various departments are studied when psychiatric consultation is requested. This is not a psychiatric department properly speaking, but in connection with the County Hospital, supplies clinical material for psychiatric teaching in the University of Nebraska Medical School. The erection of a separate psychiatric hospital on the university hospital campus is anticipated within the next two years.

It is not to be expected that these departments were developed without some opposition. Resistance had to be overcome. The hospital management had to be convinced of the feasibility and economic soundness of the projects. The fears of the noisy, destructive, violent or suicidal patient had to be reduced to actual facts. In these respects, experience has provided the answer. Financially, the psychiatric department compares favorably with the rest of the services.

The Methodist Hospital reports the collections are 3 per cent to 4 per cent larger per bed unit than the other departments for the year 1937. The Clarkson Hospital states that the collections

from the psychiatric department were 16.4 per cent of the total collections of the hospital, and a financial asset. St. Joseph's Hospital considering their investment finds the psychiatric department a liability financially, but considers it an asset from the standpoint of nurses' and internes' training, and as filling a community need. The Lutheran Hospital has found it financially profitable for several years.

The hospital charges are the same in all four hospitals—$35.00 per week for the private rooms, $25.00 per week for a dormitory bed, and $28.00 and $31.50 per week for semi-private or two-bed rooms. The extra charges, drugs, laboratory and X-ray, are as in other departments. Private nurses are discouraged.

The number of noisy or violent patients is relatively very small. With our present methods of treatment, prolonged narcosis in the rare manics, and insulin therapy with the excited catatonics, very little trouble has been experienced. The types of patients admitted are largely depressions and schizophrenias. Of 400 cases admitted, approximately 70 per cent belonged to these two groups. Psychoneurotic patients are also in many instances treated in the department in preference to the general medical department.

One of the objections raised to the psychiatric department was the duration of illness. During 1937, the average hospital stay per patient for the four hospitals was 41 days. It is interesting to note that since early last fall, Dr. Richard Young and I have treated by metrazol 24 cases of depressive psychosis of affective type, with an average period of treatment of 22 days. Of these, all but one were discharged as recovered or markedly improved. Such an experience, similar to that recently reported by Low and others in the *Archives of Neurology and Psychiatry,* will reduce the average hospital residence materially when more generally adopted.

Another significant fact is the changed attitude of both the pupil nurses and the nursing management of the hospital to the psychiatric department work. Particularly since the introduction of insulin and metrazol therapy, with the consequent striking recoveries in both schizophrenic and depressive cases, has there been a keen interest shown. This fact is not without value from the standpoint of community education along psychiatric lines.

Another feature in general hospital psychiatric work is the opportunity for research. Not only do the individual patients have ready

access to the consultation services of the hospital, but the full-time pathologist, with complete laboratory equipment, gives scope for investigation of the many psysico-chemical problems incident to psychotic states. For instance, the Methodist Hospital, in addition to now having a full-time resident in psychiatry, is employing, under a private fund, a full-time chemist for research in the psychiatric department.

DISCUSSION.

DR. SAMUEL W. HAMILTON (New York, N. Y.).—Picture to yourself the gratification of coming into a community of over 225,000 and finding five hospitals in that community with psychiatric services. In my opinion it is a most creditable accomplishment and due very largely to Dr. Young's quiet and tactful persistence. The rest of the country is going to be affected by this, I am sure. The movement for psychiatric service in general hospitals is of slow, deliberate growth but is coming along. Communities like New York, that pride themselves on their hospital development, must learn something from a city like Omaha in order to get the proper distribution of hospital beds.

I suppose that Dr. Heldt at the Ford Hospital made these psychiatric services respectable. In that brand-new hospital, much talked about, he established a psychiatric service taking the wards as they stood and demonstrating that a man who knows how to handle his patients can do it in a general hospital without undue trouble for the rest of the hospital; demonstrating also that it is of the very greatest value to other services to have a psychiatric service alongside for consultation; and demonstrating in the third place that the beds for these services are very popular and the number of free cases or low-cost cases that go to them is not greater than those in other wards, surgical, medical, etc. The mentally sick are not all in such condition that they need to be put into huge institutions and managed by wholesale feeding or treatment or nursing. Many of them are at a point where something quite different is demanded, and in the city of Omaha they are very likely to receive it.

This is of importance to us who live elsewhere. Every now and then we are asked what shall be done with a person who has had a breakdown out in the middle states. We can say of certain communities like Omaha, "Certainly, that patient can get just the treatment he needs. You can choose among several hospitals."

I think the paper and the discussion may be summed up just in one sentence, really: That there exist a large number of psychiatric cases that are general hospital cases. These acute psychoses, early in their development, are very proper cases for general hospital care.

CELL MINERALS IN AMAUROTIC IDIOCY, TUBEROUS SCLEROSIS AND RELATED CONDITIONS, STUDIED BY MICROINCINERATION AND SPECTROSCOPY.*

Examples of Degenerative and of Neoplastic Cell Disease.

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In a recent series of investigations [1, 2, 3, 4] we found that physiological processes associated with active growth and neoplastic and inflammatory diseases are characterized by an increase of heat resistant mineral ashes in the tissues, as shown by the technique of microincineration. Contrariwise, in disease processes with changes of a degenerative nature the heat resistant mineral substances of the degenerated elements or of parts of them are diminished in quantity, although there may be a shift of these heat resistant mineral ashes to other parts or elements. In view of the importance of neoplastic (anaplastic) and degenerative (cataplastic) conditions among the diseases of the central nervous system in childhood, especially among the wide and heterogeneous groups of idiocies and mental defects, we decided to study the pathological manifestations of some of these diseases by means of microincineration and spectroscopy.

Our material consists of 12 cases,† namely: three cases of Tay-Sachs' disease (infantile amaurotic idiocy), two cases of tuberous sclerosis, three cases of spongioblastomatous periventricular hamartoma of the basal ganglia, a condition somewhat related to tuberous sclerosis, and four cases of "Mongolian idiocy." The method of study is identical with that described previously.[1, 3]

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† Drs. Sidney Farber, Richard C. Wadsworth (Boston), Clemens Benda (Wrentham) and Abner Wolf (New York City) contributed material.

Amaurotic Idiocy (Tay-Sachs' Disease).

The essential pathological substratum of amaurotic idiocy is a regressive, degenerative change of all or most ganglion cells throughout the entire nervous system, associated with swelling and with accumulation of abnormal lipoid material (prelipoid, lecithinoid) in the cytoplasm of the diseased ganglion cells.[5, 6, 7] The Nissl bodies and the intracellular neurofibrillæ are lost. In contrast to the severe intracellular changes the extracellular neuropilema of the grey matter remains remarkably well preserved. The glial reaction is variable and obviously secondary, as shown by the fact that in Case 1, that of a one year old infant, there was no glial reaction at all; in Case 2, that of an infant two and one-half years old, glial proliferation was active in some places only; while in Case 3, that of an infant two years and eight months old, oligodendroglial, microglial and astrocytic proliferative reaction was extremely active.

Figs. 1 A and B show low and high power views of sections of diseased cerebral cortex (temporal region), stained according to Bodian's silver method.[8] The swollen diseased ganglion cells, imbedded in an approximately normal neuropilema, stand out equally well by low as by high power; their nuclei are eccentric and darkly stained; their cytoplasm is either unstained with silver or contains faintly stained and loosely arranged finely granular material. The appearance of the diseased ganglion cells in Masson stained preparations is similar (Figs. 3 D and G). Nissl preparations show the cytoplasm of the diseased ganglion cells either unstained, or for the most part unstained and traversed by a fine trabecular network (Figs. 3 C and F); in other instances it is either completely or partially filled with faintly stained fine granular material (Figs. 3 A, I, J). The cell membrane is always stained rather distinctly; the nuclei are often pyknotic (Figs. 3 A, C, D, G, I, J). Weigert preparations show the cytoplasm of the diseased ganglion cells densely filled with masses of coarse prelipoid granules.

In microincinerated preparations the cytoplasm of the diseased ganglion cells is completely or almost completely void of heat resistant mineral ashes (Figs. 2 B and 3 B); in other instances the heat resistant mineral ash is reduced to a fine granulotrabecular

network, most of which is only faintly luminous in the darkfield (Figs 3 E and H). The cell membrane, however, is usually well outlined by heat resistant mineral ash. The pyknotic and greatly shrunken nuclei of the diseased ganglion cells contain densely aggregated ash residue (Figs. 2 B and 3 H), mainly due to mere condensation of the mineral constituents of nucleolus, nuclear membrane and oxychromatine. The demineralized cell bodies of the diseased ganglion cells stand out even at low power (Fig. 2 A), in sharp contrast to their pyknotic nuclei and to the cortical ground substance in which they are imbedded, which contains an approximately normal amount of heat resistant mineral ash. In those cases in which there is glial proliferation, the proliferating glial cells, in contrast to the demineralized ganglion cells, are hypermineralized (Figs. 3 B, E, H). The nuclei, cell bodies and processes of these growing glia cells: microglia, oligodendroglia and astrocytes alike, are densely filled with ample deposits of white, water insoluble heat resistant mineral ash. By virtue of this rich content in brightly luminous mineral ash, these growing glia cells and their processes, many of which hug the demineralized ganglion cells closely, are well outlined in microincinerated preparations (Figs. 3 B, E, H).

Spectroscopic examination of the brain tissue reveals a significant diminution of calcium and phosphorus (Fig. 4, Table 1). The quantitative changes shown by the other mineral ashes enumerated in Table 1 cannot yet be regarded as significant in the light of our experience as it stands at present. It should be added that most of the heat resistant ash as seen in microincinerated preparations consists of calcium ash.

Tuberous Sclerosis (Bourneville's Disease).

Cerebral pathology of tuberous sclerosis may briefly be summarized as an alteration in the distribution, shape, and size of ganglion cells and glia cells. The deformities are mainly swelling and bizarre elongation of processes, and the appearance and occasional accumulation of immature and dedifferentiated forms. The literature is unanimous in considering these alterations as due to a change of blastomatous or neoplastic nature (Globus).[6]

In specimens prepared with the ordinary stains the diseased ganglion cells may resemble those seen in amaurotic idiocy

(Globus),[6] except that the abnormal lipoid characteristic of amaurotic idiocy is absent. In Nissl and Masson stained preparations the resemblance is striking indeed, as can be seen by comparing Figs. 5 A, D, G, 6 A, E, from a case of tuberous sclerosis (Case 4) to Figs. 1 B, 3 A, C, D, F, G, I, J, from cases of amaurotic idiocy. Differences, however, can be made out even in these specimens: the nuclei of the diseased ganglion cells in tuberous sclerosis always show the essential characteristics of normal nerve cell nuclei, namely, a rather large and dark stained nucleolus in a light or un-

TABLE 1.

Mineral constituents of brain tissue (grey and white matter), in per cent of weight of charred tissue, in amaurotic idiocy and in tuberous sclerosis, as compared to normal standards. Quantitative determinations from emission spectra (see Fig. 4). Note diminution of calcium and phosphorus in amaurotic idiocy, increase of calcium in tuberous sclerosis. The quantitative changes shown by the other minerals cannot yet be regarded as significant in the light of our experience as it stands at present. Compare microincinerated preparations shown in Figs. 2, 3, 5 and 6. Most of the heat resistant mineral ash seen in microincinerated preparations is calcium ash (calcium oxide).

	Normal standard.	Amaurotic idiocy.	Tuberous sclerosis.
Silicon	.0024	.0150	.025
Copper	.0078	.017	.023
Iron	.14	.13	.065
Sodium	1.6	.27	.46
Magnesium	.45	.10	.15
Potassium	.030	.009	.015
Calcium	.12	.057	.22
Phosphorus	1.3	.65	1.6

stained nucleus (Figs. 5 A, D), while in amaurotic idiocy the nulcei of most of the diseased ganglion cells are pyknotic (Figs. 3 C, D, G, I, J). In addition, the cytoplasm of the diseased ganglion cells in tuberous sclerosis is more homogeneous and less granular than in amaurotic idiocy.

Striking differences, however, are shown by the microincinerated preparations. While the cytoplasm of the diseased ganglion cells in amaurotic idiocy is demineralized, that of the diseased ganglion cells in tuberous sclerosis contains ample deposits of white and bluish heat resistant mineral ashes (Figs. 5 B, C, F, I,

J, 6 B, C), most of which are insoluble in water. The amount of these ashes by far exceeds that seen in normal ganglion cells.[4] As in all normal ganglion cells, however, their nucleus is essentially free from heat resistant mineral ashes, except for those contained in its fine oxychromatine granules and in its nuclear membrane (Figs. 5 B, C, F, 6 B, C), while their nucleolus contains a normal amount of ash. This is in contrast to the ample mineral deposits in the pyknotic nuclei in amaurotic idiocy.

The diseased glia cells in tuberous sclerosis, many of them "monster glia cells" are distinguishable from the diseased ganglion cells by the characteristic chromatin structure of their nuclei (Figs. 5 H, 6 F, G), by the shape of their processes, and by the fact that they are lacking intracellular neurofibrillæ, which are usually present in the diseased ganglion cells in tuberous sclerosis (Pollak).[9] In microincinerated preparations the diseased glia cells are equally hypermineralized (Figs. 5 E, J); and so are the immature cells which compose the spongioblastic subependymal tumors, frequently found in cases of tuberous sclerosis.

Spectroscopic examination reveals a significant increase in calcium (Fig. 4, Table 1), similar to that found in certain intracranial tumors.[3]

Spongioblastomatous Periventricular Hamartoma.

In a study of the anatomy and pathology of the lateral recesses of the fourth ventricle, one of us[10] described a periventricular spongioblastomatous hamartoma resembling the periventricular tumors of tuberous sclerosis in the brain of a 42 year old man, which showed no other signs of tuberous sclerosis. We have since seen three more instances of this condition, which to us appears to be an attenuated form of tuberous sclerosis. All three cases were those of premature infants, two stillborn, and one who had died shortly after birth. In one of the stillborn infants the direct cause of death was rupture of the tentorium with subdural and subarachnoid hemorrhage; in the two others prematurity and the cerebral tumor formation were the only positive pathological findings. In the first of these cases, a newborn premature infant who had died two days after birth, the gross appearance was that of a small hemorrhage, measuring 5-6 mm. in diameter, located at the

stria terminalis on the right and involving the adjacent parts of the caudate nucleus and the thalamus opticus. Histological section, however, revealed that the apparent hemorrhage actually was an aggregation of small, richly vascularized spongioblastic tumor nodules (Fig. 7) into one of which fresh extravasation of blood had occurred. In the other two cases, the diagnosis was made on the gross specimen and confirmed microscopically. In one of them (Case 8) the vascular pattern of the tumor was studied with the benzidine stain; its wide sinusoid channels with the characteristic arrangement in palisades and "windswept tree" formations corresponded to that typical of polar spongioblastomata (Sahs and Alexander).[11]

Microincineration reveals rich deposits of white, water insoluble heat resistant mineral ashes in the nuclei, somewhat less in the cell bodies and processes of the primitive bipolar and unipolar spongioblasts, which compose these tumor nodules (Figs. 8 B, C, D). At low power these tumor nodules stand out as grossly hypermineralized areas (Fig. 8 A), like all tumors in the central nervous system and in other parts of the body.

"Mongolian Idiocy" (Langdon-Down's Disease).

In this disease the mineral constituents of the brain tissue are esentially unaltered; even the slightly enlarged and somewhat plump pyramidal cells which may be seen occasionally in some parts of the cortex show an essentially normal ash picture.

Conclusions and Summary.

(1) The ganglion cell disease of amaurotic idiocy is characterized by demineralization, that of tuberous sclerosis by hypermineralization of the cytoplasm. In this respect the ganglion cell disease of amaurotic idiocy aligns itself with other degenerative conditions; that of tuberous sclerosis with other conditions of a blastomatous or neoplastic nature. The actively proliferating reactive glia cells in amaurotic idiocy are hypermineralized, like all actively growing tissues.

(2) The demineralization of the diseased ganglion cells in amaurotic idiocy is of especial interest in view of the associated increase in lipoid material. In our studies thus far, it appears that

wherever lipoid is accumulated, normally or pathologically, heat resistant mineral ashes are absent. This is well exemplified by the lipoid body of the normal myelin sheath, which is completely lacking in heat resistant mineral ash.[4]

(3) The "periventricular spongioblastomatous hamartoma," presumably an attenuated form of tuberous sclerosis, which had been described by one of us [10] and three new cases of which are reported here, shows gross hypermineralization which corresponds to that generally found in tumors and other actively growing tissues.

(4) The mineral ash picture of the brain in "Mongolian idiocy" (Langdon-Down's disease) does not show significant alterations from the normal.

BIBLIOGRAPHY.

1. Alexander, Leo, and Myerson, A.: The mineral content of various cerebral lesions as demonstrated by the microincineration method. Am. J. Path., 13: 405-439, 1937.
2. Alexander, Leo, and Campbell, A. C. P.: Local anaphylactic lesions of the brain in guinea-pigs. Am. J. Path., 13: 229-248, 1937.
3. Alexander, Leo, and Myerson, A.: Minerals in normal and pathologic brain tissue, studied by microincineration and spectroscopy. Arch. Neurol. & Psychiat., 39: 131-149, 1938.
4. Alexander, Leo: The neurone as studied by microincineration. Brain, 61: 52-62, 1938.
5. Schaffer, Karl: Über die drei Formen der "amaurotischen Idiotie," richtiger der "gangliocellulären heredodegenerativen Demenz." Ztschr. f. d. ges. Neurol. u. Psychiat., 139: 790-801, 1932.
6. Globus, Joseph H.: Malformation in the central nervous system. In: Cytology and Cellular Pathology of the Nervous System, W. Penfield, Ed., New York (P. B. Hoeber), 3: 1147-1199, 1932.
7. Hausman, L.: Amaurotic family idiocy (Tay-Sachs' disease). Cyclopedia of Medicine, Piersol, Ed., Philadelphia (F. A. Davis), 340-351.
8. Bodian, David: A new method for staining nerve fibers and nerve endings in mounted paraffin sections. Anat. Rec., 65: 89-97, 1936.
9. Pollak, Eugen: Über tuberöse Hirnsklerose. Arb. a. d. Neurol. Inst. d. Univ. Wien, 24: 93, 1922.
10. Alexander, Leo: Tumors and cysts of the cerbellopontine angle and their relation to the lateral recesses of the fourth ventricle. In: Tumors of the Nervous System. An investigation of the most recent advances. Proc. Assoc. for Research in Nervous and Mental Disease, 16: 266-314, Baltimore (Williams and Wilkins), 1937.
11. Sahs, A. L., and Alexander, Leo: Vascular pattern of certain intracranial neoplasms. Arch. Neurol. & Psychiat. 42: 44-66, 1939.

EXPLANATION OF FIGURES.

FIG. 1.—Case I. Amaurotic idiocy. Infant, aged 1 year. A: low power view (×71) of cerebral cortex, temporal region. B: high power view (×1100) of diseased ganglion cell from same section. Bodian's silver stain. Microphotographs.

FIG. 2.—Case 2. Amaurotic idiocy. Infant, aged 1 year. Neighboring section to that shown in Fig. 1, subjected to microincineration. Darkfield illumination. Microphotographs. A: low power view (×71). B: high power view (×1100) of diseased ganglion cell. Note demineralization of cell body. Only nucleus and cell membrane contain heat resistant mineral ash.

FIG. 3.—Case 3. Amaurotic idiocy. Infant, aged 2 years and 8 months. Diseased ganglion cells and some of the surrounding reactive glia cells from various parts of the cerebral cortex. Microphotographs, ×875. A: Nissl stain. B: Microincineration. Darkfield illumination. Note the demineralized cell bodies of the three diseased ganglion cells shown in this picture, while the surrounding glia cells and their processes are hypermineralized. The diseased ganglion cell at the upper right is hugged closely by two greatly hypermineralized astrocytic processes (compare with the ganglion cell at the upper left in Fig. 3 G, which shows a similar relationship in a Masson stained preparation). The two large hypermineralized glia cells at the upper margin of the figure are microglia cells, those at the lower margin oligodendroglia cells. C: Nissl stain. D: Masson's trichrome stain. E: Microincineration. Darkfield illumination. Note demineralization of diseased ganglion cell (to the right and below center of figure), except for ash residue of cell membrane and of fine, wide-meshed granulo-trabecular network within cell body. Hypermineralization of surrounding glia cells which clasp the cell body: microglia cell (in lower left of figure), astrocyte (in upper right) and oligodendroglia cells (in upper left and lower right). F: Nissl stain. G: Masson's trichrome stain. Two diseased ganglion cells (at left and right margin of figure). The dark stained groups of glia cells at the lower left are microglia cells, the one at the upper right is an astrocyte. The ganglion cell at the left is hugged closely by dark stained processes of astrocytes, the cell bodies of which are not seen in this section (compare with ganglion cell at upper right in Fig. 3 B). H: Microincineration. Darkfield illumination. Diseased ganglion cell in upper half of figure, oligodendroglia cells: one in lower half of figure, two at lower margin of diseased ganglion cell. I, J: Nissl stain.

FIG. 4.—Emission spectra of brain tissue (cortical grey and white matter) from cases of amaurotic idiocy and of tuberous sclerosis, and of normal control cases. A: Normal control (11.2 mg. of tissue). B: Amaurotic idiocy (Case 3) (7.2 mg. of tissue). C: Tuberous sclerosis (Case 5) (8.1 mg. of tissue). D: Normal control (10.5 mg. of tissue). E: Normal control (10.5 mg. of tissue). F: Amaurotic idiocy (Case 3) (7.6 mg. of tissue). G: Tuberous sclerosis (Case 5) (7.1 mg. of tissue). H: Normal control (10.1 mg. of tissue). (Compare with Table I.)

FIG. 5.—Case 5. Tuberous sclerosis. Infant, aged 6 months. A: Diseased cortical ganglion cell, Masson's trichrome stain. B and C: Diseased cortical ganglion cells, surrounded by normal glia cells. Microincineration, darkfield illumination. D: Diseased cortical ganglion cell. Masson's trichrome stain. E: Diseased and enlarged glia cell from cortical grey matter. Microincineration, darkfield illumination. F: Diseased cortical ganglion cell. Microincineration, darkfield illumination. G: Diseased cortical ganglion cell surrounded by normal glia cells. Masson's trichrome stain. H: Diseased glia cell from cortical grey matter. Hematoxylin and eosin stain. I: Diseased cortical ganglion cell. Microincineration, darkfield illumination. J: A group of diseased ganglion cells and glia cells from a heterotopic nodule in the subcortical white matter. Microincineration, darkfield illumination. The three cells in the upper left quadrant of the figure are diseased ganglion cells, the two in the right lower quadrant (one of them close to the center) are diseased glia cells. The one near to the lower margin of the figure is a "monster glia cell" with numerous bizarrely elongated processes. All figures are microphotographs, $\times 875$. Note hypermineralization of cell bodies and processes of diseased ganglion cells and glia cells in all microincinerated preparations (B, C, E, F, I, J).

FIG. 6.—Case 5. Tuberous sclerosis. Infant, aged 6 months. A: Three diseased cortical ganglion cells. Nissl stain. B and C: Diseased cortical ganglion cells. Microincineration, darkfield illumination. D and E: Diseased cortical ganglion cells. Nissl stain. F: A group of diseased ganglion cells and glia cells from a nodule in the subcortical white matter. Masson's trichrome stain. G: A diseased glia cell (in upper half of figure) and a diseased ganglion cell (in lower half of figure) from cortical grey matter, among a number of normal glia cells. Nissl stain. H: Diseased cortical ganglion cell. Nissl stain. Microphotographs, $\times 875$.

FIG. 7.—Case 6. Periventricular spongioblastomatous hamartoma. Newborn premature infant, aged 2 days. This section shows one of the group of spongioblastic tumor nodules (T) in the periventricular zone of the right caudate nucleus, laterodorsal to the stria terminalis. E = ependymal covering of caudate nucleus. L = right lateral ventricle. Stain: hematoxylin-eosin. Microphotograph, $\times 90$.

FIG. 8.—Case 6. Periventricular spongioblastomatous hamartoma. Newborn premature infant, aged 2 days. From a section neighboring that shown in Fig. 7, subjected to microincineration. Darkfield illumination. Microphotographs. A: Low power view ($\times 60$) of one of the spongioblastic tumor nodules in the periventricular zone of the right caudate nucleus. Note intense hypermineralization of tumor nodule. B: Part of the section shown in Fig. 8 A at higher enlargement ($\times 240$). Note nodular arrangement of tumor cells and mineral content of the nuclei and processes. C and D: High power views ($\times 875$) of spongioblastic tumor cells, from section shown in Figs. 8 A and B. E = ependymal covering of caudate nucleus. L = right lateral ventricle. S = stria terminalis.





FIG. 1.



FIG. 2.

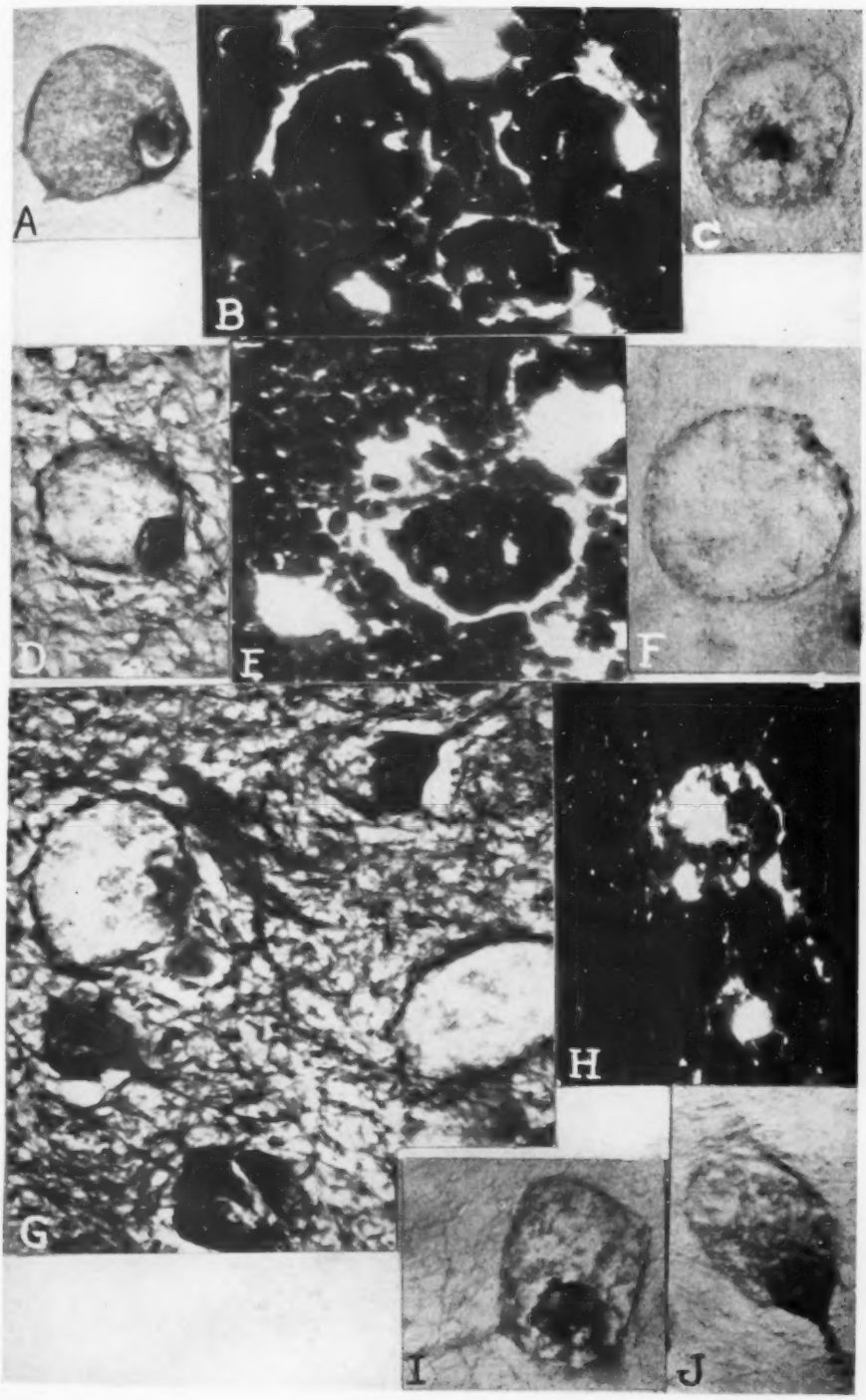


FIG. 3.

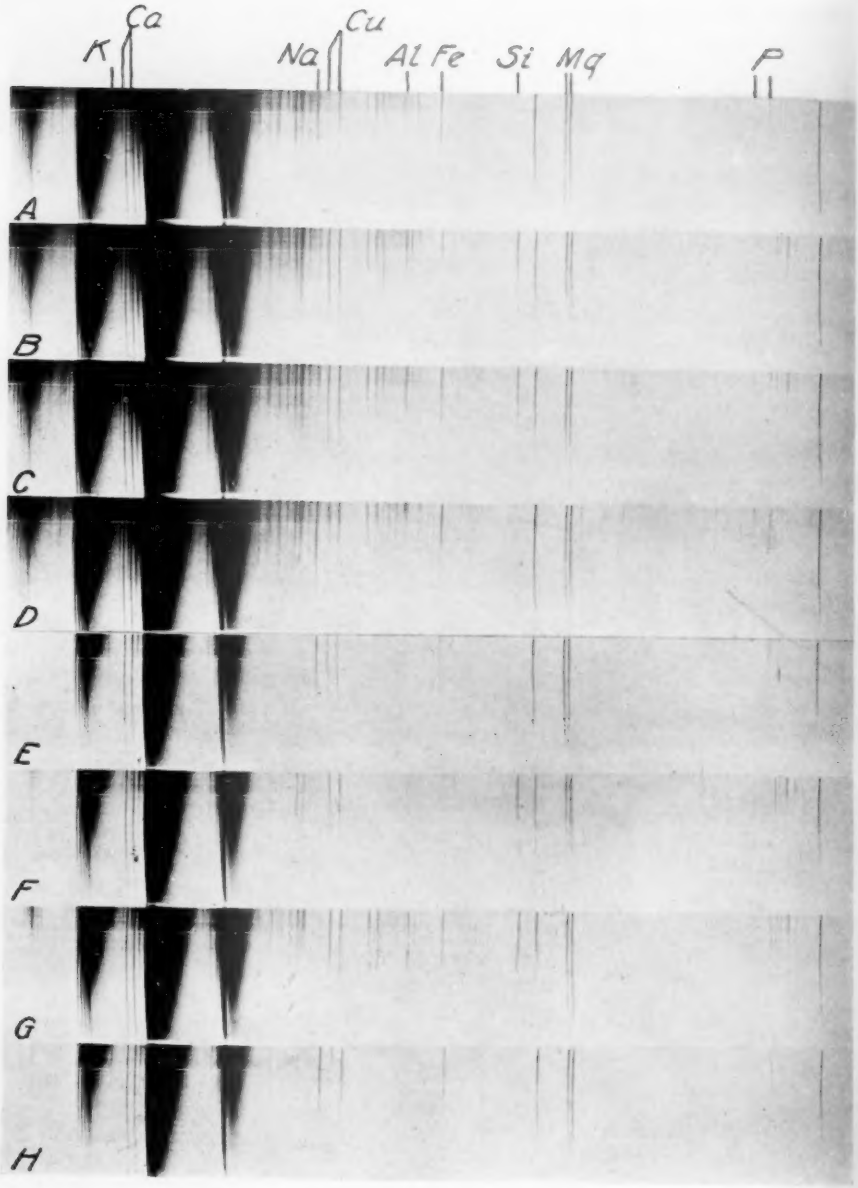


FIG. 4.

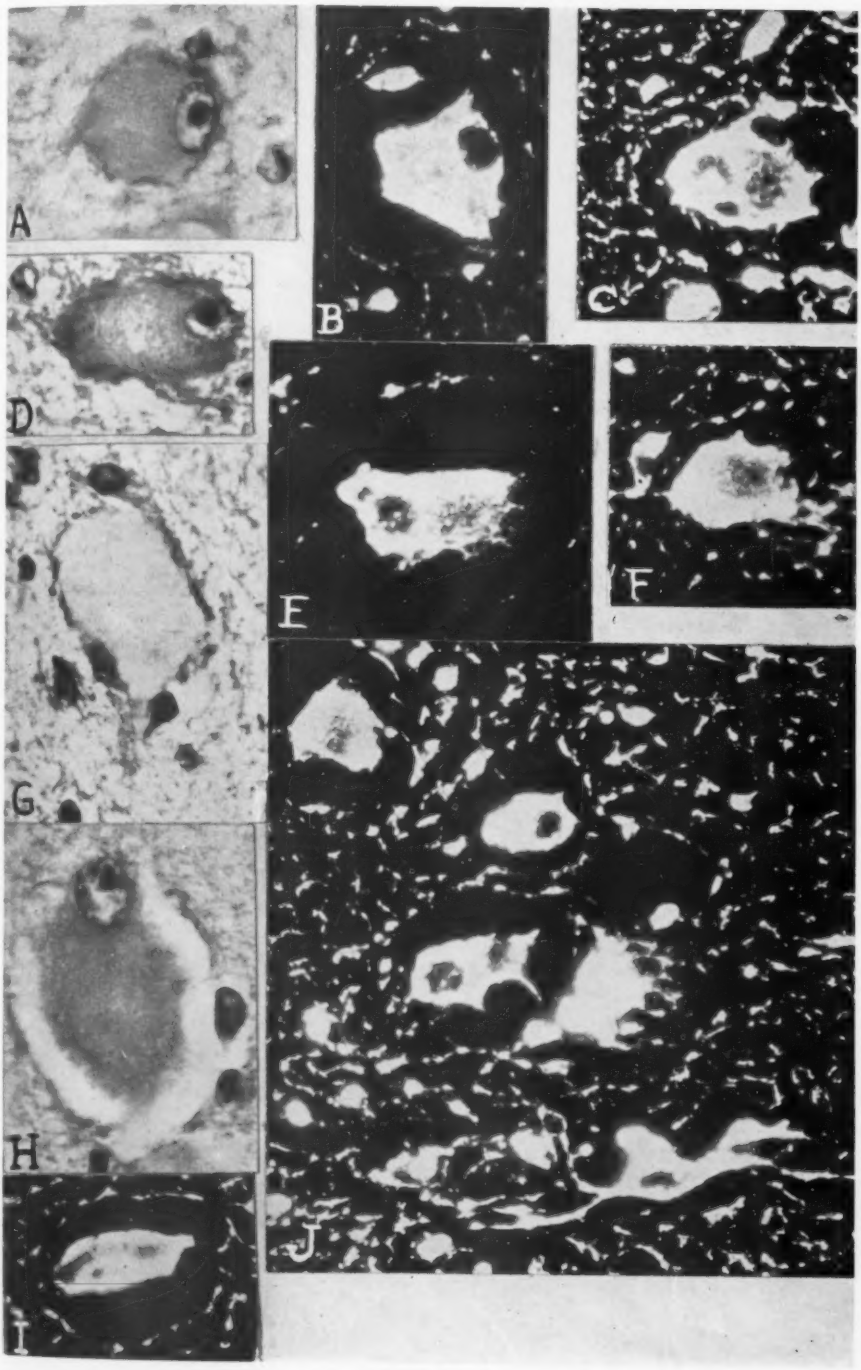


FIG. 5.

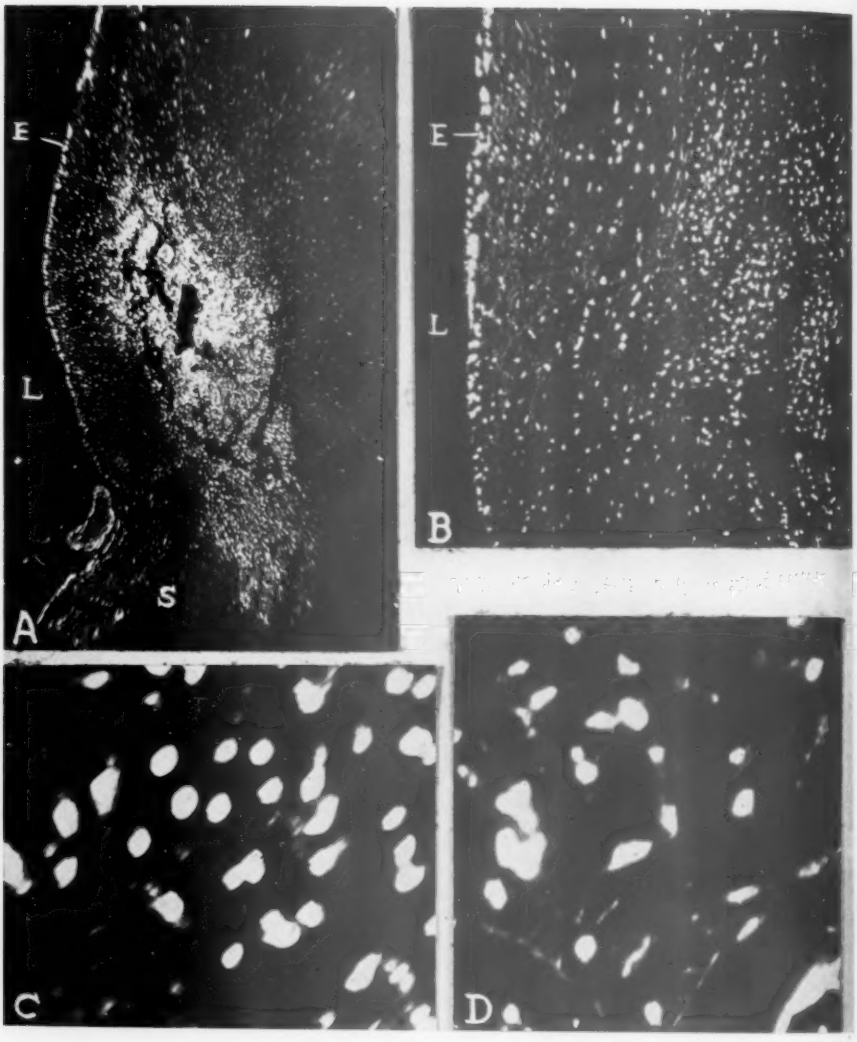


FIG. 8.

PSYCHOGENIC FACTORS IN ASTHMA.*

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Since earliest times it has been suspected that psychological factors play some rôle in connection with bronchial asthma. In the last few decades the discovery of the significance of allergic factors in this disease has tended to divert interest away from earlier observations that asthma is frequently associated with some sort of emotional instability and that asthma attacks are often precipitated by acute emotional conflicts. In the present study we wish to return to the problems presented by these earlier observations. In so doing we have been interested first of all to determine the nature of the emotional disturbances associated with asthma and their relation to the attacks but also secondarily to inquire into the relation between these emotional disturbances and the allergic factors which have been more recently discovered.

The following report is based upon investigations carried out by Doctors Alexander, Bacon, Eisler, French, Gerard, McLean, Mohr, Ross, Saul, Tower and Wilson, supplemented by the writer's detailed and comparative study especially of the dream material of our asthma patients. Twenty-four cases of bronchial asthma were analyzed at the Chicago Institute for Psychoanalysis over periods of one to 26 months. Of these six were men, ten were women and eight were children, four boys and four girls. The cases chosen were patients in whom allergic hypersensitivity had been previously demonstrated by means of allergic history, skin tests, etc., as judged by an experienced allergist. In addition Dr. Siegfried Bernfeld has supplemented our studies by analyzing and summarizing the case records of eight asthmatic children who have been under close observation since birth by the Institute of Child Welfare at the University of California at Berkeley.

Patients who undertake and continue through an analytic treatment must be patients who are suffering from some sort of emo-

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tional disturbance. It is impossible therefore to draw any significant conclusions from the fact that in all the patients that we analyzed the asthma was found associated with emotional difficulties of varying grades of severity. It will be interesting to inquire, however, whether the emotional conflicts and personality structure in these patients show any common features as contrasted with patients suffering from other forms of disorder.

An analysis tends moreover to mobilize emotional conflicts which may previously have been latent, and this might be expected to throw into relief the rôle played by emotional factors as contrasted with allergic factors in precipitating the attacks. It will be of interest nevertheless to inquire as to whether the emotional situations which seem to precipitate the attacks present any specific common characteristics. If they do we may then inquire whether emotional conflicts of this specific character may perhaps also have been present as precipitating factors for other asthma attacks which preceded the analytic procedure.

Upon passing our cases in review, our first impression is that they vary considerably both in their personality traits and in the type of emotional disturbance for which they sought treatment. A number of our patients were particularly good children and were characterized in adult life by an urge to help and give to others. Some of our children's cases on the other hand were brought to treatment because of their particularly aggressive behavior. In one of our adult patients and in one of our children the picture at the beginning of the treatment was of a compulsive character with mild compulsive neurotic symptoms. Still another, a man, sought treatment originally on account of conscious homosexual impulses. Upon superficial examination therefore there seems to be a considerable range of divergence in the personality picture presented by our asthma patients.

It is much easier at the outset to demonstrate common features in emotional situations that seem to precipitate the asthma attacks. Let me cite a few examples.

A male patient of Dr. Saul's reports that he developed asthma for the first time shortly after marrying against his mother's opposition. Another attack is associated with his sleeping with his wife at a camp in a room separated by only a thin partition from the room of the director, an "old woman" who objected to the patient and his wife talking after 10 p. m.

A male patient of Dr. Catherine Bacon reported the following two dreams the day after he had had a basal metabolism:

a. This morning. Was in a Catholic church. Three priests were in front of him and they had gleaming eyes. Their eyes were sparkling with delight. Patient was evidently becoming a Catholic. He thought of wife's remark Catholics are so glad to have a Protestant in their ranks. He thought the gleam was due to their getting him away from Protestants. Their eyes gleamed so brightly that it was like jewels. Remembers no color. He would have had more to this dream if he hadn't waked up wheezing.

b. This morning. He was also awakened from this by wheezing. This dream preceded the first one. A naked woman. She was lying down stretched in front of him with feet toward him and her legs stretched apart. She didn't represent a woman all the time, for part of the time her legs became writhing snakes.

Patient associates to the snakes in the dream last reported (actually the one which occurred first) fear of being poisoned by a rattlesnake; having an affair with a woman—he would be in danger of trouble and maybe disease; snakes that wrap themselves around a victim suggest the idea of a woman twining her legs around him and crushing him; "domination would come into that too" he adds. The dream is in reaction to the analysis as a temptation situation. Patient vainly attempts to ward off the sexual temptation by substituting phallic symbols for the female genitals but the attempt fails and he awakens with asthma. The other side of his conflict is portrayed in the second dream (the one reported first). The three priests correspond to the three men present to observe patient's basal metabolism test of the day before. Their gleaming eyes portray the danger that they will see through patient and detect his incestuous wishes toward the analyst. The manifest dream is an attempt to defend himself from this danger. In the manifest dream the priests are not offended by patient's incestuous desires but their eyes are sparkling with delight at the prospect of converting him to a new faith. In the context it is easy to surmise that the new faith is the psychoanalytic "faith" which permits and encourages one to become conscious even of incestuous desires without fear of offense.

It will be noticed that in all three of these instances we find an identical conflict. In each case the patient is exposed to a temptation which would estrange him from a parental figure, usually the mother. In the dreams reported by Dr. Bacon's patient we see this conflict portrayed particularly vividly. The patient is struggling desperately to ward off the temptation and the associated danger of losing the parents' love. He attempts to deny the temptation by substituting phallic symbols for the female genitals and then in his second dream he attempts to ward off the fear of loss of the parents' love by appealing to the tolerance of psychoanalysis for unconscious impulses and picturing the analysts as

trying to take him into their fold instead of expelling him from the church as he really fears they will do. In both cases he awakens wheezing. It is only a short step to infer that the attack of asthma occurs at the point where his defenses fail at the moment when he is suddenly exposed to the conflict between an acute temptation and his fear of losing the mother's love.

With this sort of fundamental conflict we should expect that the tolerance offered by the analysis to the forbidden unconscious wishes would acquire an ambiguous meaning for the patient. At first the tolerant atmosphere of the analysis might be expected to quiet the patient's fears of losing the mother's love. Here at last is a mother who will not turn away from the patient. But very soon this same tolerance acquires a dangerous seductive character. Forbidden impulses thus encouraged into consciousness now awaken old fears that the analyst, the new mother, will behave like the original mother and will punish the patient's forbidden wishes by withdrawing her love.

I regret that time does not permit me to give examples of the numerous dreams which could be selected to illustrate this double aspect of the transference. Over and over again these patients become blocked just at the point where the unconscious material is leading up to a confession. They fear to make this confession and awaken with asthma.

Most of these patients have a very strong urge to regain the love of the mother by confession. In fact this urge to confess sometimes goes to ridiculous extremes.

For example the patient of Dr. Saul's to whom I have already referred was at one time seduced by a girl almost to the point of having intercourse with her. Then the patient suddenly left her and rushed to his mother to confess the episode. The mother wept and made a great scene. A sequel to this incident is particularly instructive. After the interruption of his analysis the patient, who was a seasonal case, had lived through the spring and fall seasons during which he was usually subject to asthma without an attack. Then in January he responded to the telephone invitation of a girl from out of town, went to her hotel room and they got into bed together. He had an orgasm but not intercourse, then fled in terror to his wife to whom he confessed only the non-sexual part of the story. During the succeeding two months he had a number of attacks of severe asthma. The attacks ceased when he finally gained spontaneous insight into the fact that they were a reaction to this incident.

Incidents of this sort occur frequently in the histories of our asthma patients. So long as this technique of winning back reconciliation with the mother by confession is successful, the patients appear to be protected from asthma attacks. But sometimes one is afraid to confess. Then as in this dream the confession is choked in one's throat and in its place occurs an asthma attack.

Confession is a defense that utilizes speech and speech is a respiratory mechanism. It is therefore perhaps not surprising that it should have so intimate a relationship with the asthma attacks. It is interesting to note moreover that there seems also to be a very intimate relationship between asthma and crying or laughing. There is much to suggest in fact that the asthma attack is really a sort of equivalent of a cry of anxiety or rage which has been inhibited and repressed. One of the first reactions of the child upon separation from the mother at birth is to cry and during early infancy and even later, crying remains the predominant reaction of the child in situations of helplessness when he can only wish for the mother to return to him. In later life this primitive cry is modified into speech and in confession retains the function of maintaining the bond between the child and mother. For some reason, in the situations which provoke asthma attacks the child is unable to cry. Some of our patients state that they have not cried for years and others boast that they have never been afraid. In other cases it is apparently only in certain situations that crying is inhibited. Of particular interest however is the fact that we are sometimes able to observe the cessation of an asthma attack and the appearance of crying in its place. Such replacement of asthma attacks by crying is particularly apt to occur at times when the analysis is beginning to achieve some degree of resolution of the asthma attacks.

So far we have found that the acute asthma attack occurs in place of a repressed cry in reaction to a temptation that threatens the patient with loss of the mother's love. Is it possible to define more accurately the nature of this temptation?

It will be noted that in all the instances cited, the temptation is a sexual one. The relation of the asthma attacks to the Œdipus complex differs somewhat according to the sex of the patient. In male patients the typical conflict is between sexual urges toward the mother and the fear of losing the mother's love. A typical

family constellation in the early childhood of these patients is one in which the mother is over-protective to the little boy and binds him to her in a dependent relationship but immediately rejects the first signs of infantile genital interest in her. This creates a situation in which the first infantile stirrings of genital (phallic) sexuality become a temptation that threatens to deprive the little boy of the mother's love upon which he is so dependent. One is struck with the frequency with which the mothers of these patients play this double rôle, at once seductive and prohibitive. Four of our six adult male patients, for example, had continued to sleep with the mother until the age of puberty or later.

Fear of the father on the other hand seems not to play so great a rôle in connection with the asthma attacks. It is rather the loss of the father's love that is feared. The father seems in fact to play the rôle of a mother substitute.

In girls the situations precipitating attacks seem typically to be ones in which the first stirrings of sexual interest in the father threaten the little girl with estrangement from the mother. A frequent constellation in early childhood is one in which the father plays a somewhat seductive rôle but the mother binds the little girl to her in pregenital depedence upon condition that she betray no sign of awakening genital sexuality.

It should be added that due to the intense repression of sexuality in many of these cases a superficial study of the attacks may be in danger of overlooking the importance of a sexual temptation in precipitating an attack. In such cases the patient will seem to have almost no interest in sex and the attack will be attributed to some other incident of a much more superficial character.

A patient of Dr. George Mohr's for example attributed her first asthma attack in the analysis to her worry about letting her child go with the father on a cold day without underwear. The preceding analytic material, however, pointed to the patient's first unconscious sexual transference wishes toward the analyst. The next attack of asthma some weeks later followed an exhibitionistic dream in which the patient was herself without underwear.

Sexual wishes arising out of the Œdipus complex are not, however, the only wishes that precipitate asthma attacks by threatening to disturb the patient's dependent relationship to the mother. The birth of younger children and especially the discovery that the mother is pregnant plays an exceedingly important rôle in pre-

cipitating asthma attacks. It is not difficult to understand why this is the case. The possibility of another child already threatens the patient with the loss of the mother's love and stirs up aggressive impulses to take the unborn child from her, but these aggressive impulses in turn only increase the danger that the mother will withdraw her love.

In one of Dr. McLean's cases, a man, a period of asthma attacks dated from his discovery that a married woman with whom he was having a sexual relationship was pregnant. Another period of asthma attacks began during his wife's first pregnancy one year after his marriage at which time patient proposed an abortion but his wife refused. During the analysis many attacks of asthma were stirred up by a discussion of what the patient felt to be his wife's neglect of his children, a complaint which upon analysis proved to be a substitute for the unconscious complaint that his wife neglected him for the children.

It is interesting that it is sexual temptation rather than forbidden sexual gratification that usually serves as the precipitating cause of the asthma attack. Much of our material suggests that if the patient has gone so far as to commit himself to an overt sexual act this is apt to be already a sign that he has mastered the panic fear of loss of the mother's love to a degree sufficient to avert the asthmatic attack. The asthmatic attack seems rather to be a reaction of acute helplessness. At the moment of temptation the dependence upon the mother is so great that the threat of losing her completely overpowers the patient. The mechanism is similar to that postulated by Freud for acute anxiety. The asthma attack occurs only when patient is overpowered by a mass of excitation which the ego is powerless to master.

The fantasy material of our asthma patients is full of symbolic representations of intrauterine fantasies in symptom-free periods as well as in the material accompanying asthma attacks and the asthma attacks seem to be associated with material symbolic of interruption or disturbance of the protected intrauterine state. Alexander has attempted to check this impression statistically and finds that the proportion of symbolic intrauterine fantasies in the manifest dream content averages considerably higher in the asthma patients than in any other class of patients who were used as controls. The nearest approximation to the proportion found in asthma cases is in some cases of anxiety neurosis.

The fact that the asthma attack seems to be an acute reaction to a conflict which is too intense for the ego to master enables us to understand better the defenses by means of which the ego is able to protect itself from asthmatic attacks during the symptom-free periods. These defenses vary a great deal and the numerous possible defense mechanisms account for the great variation in the superficial clinical picture which we noted at the beginning of our discussion.

(1) One type of defense is found in practically all cases. I have already alluded to the urge to seek reconcilation with the mother by means of confession. This is but one example of the need that our asthma patients show to seek reassurance that the mother will really not be offended in spite of the patient's unconscious wishes. As already mentioned many of our patients were particularly good children seeking to assure themselves in this way of the mother's love. A disposition to win love by helping and giving to others is very common in our asthma patients and serves the double function (1) of winning parental approval and (2) of vicariously gratifying the patient's own need for love and reassurance by helping someone else as the patient himself would like to be helped and loved. Other patients develop a great facility for making use of rationalizations which were presumably acceptable to their parents.

Still other patients utilize sickness and suffering in the well-known manner as means of regaining the sympathy and affection of the parents. One finds therefore very often that other symptoms such as rheumatic pains or headache for example will tend to protect the patient from attacks of asthma. In cases where the asthma has become more or less chronic the mechanism seems to be one of a secondary adaptation to the symptom by means of which the child has learned to utilize the asthma attacks themselves as a means of regaining for the patient the mother's love. One of our male patients in fact was regularly taken into the mother's bed whenever he had an asthmatic attack.

(2) Another group of defense mechanisms offer a sharply contrasting picture. These are defenses by the familiar method of attempting to master a traumatic event which has been passively experienced by active repetition of it. In this connection we have already noted that it is sexual temptation rather than forbidden

sexual gratification that usually serves as the precipitating cause of the asthma attack. It would seem that overt sexual activity already implies a degree of mastery of the traumatic fear of the loss of the mother's love sufficient to avert the asthmatic attack.

Especially in a number of our children's cases we can observe an attempt to master the fear by aggressive behavior. A little girl will ward off the dangerous temptation to father incest by castrative impulses toward the father, or a little boy will attempt to master his fear of seduction by the mother by aggressive attacks (anal, oral or both) upon her. Similarly our adult women patients defend themselves from the temptation to genital interest in the father by mechanisms of masculine protest and father identification such as are familiar to us in all types of neuroses in women and in one of our male cases competitive attitudes toward the father served in a similar way to ward off the dangers arising from the patient's extreme dependence upon the mother's love.

Sometimes the aggressive impulses are direct reactions to the fear of suffocation and take the form of choking the mother. In a dream reported by Dr. McLean's patient, the patient urinated upon a hot stove in a boxcar as he saw two inspectors approaching and then ran away to watch with glee how they would be choked by the bad smell. Corresponding to this aggressive defense against the dangers arising from the patient's sexual impulses, this dream was not accompanied by asthma.

(3) A third group of defense reactions may be described as attempts on the part of the patient to withdraw from the whole temptation situation and make himself independent of the mother by means of autoerotic and particularly anal erotic substitutes. In many of our cases a marked periodicity is demonstrable in the course of the analysis. There will be long periods during which the patient misses hours and comes only infrequently to the analysis and during which the material is characterized by anal erotic attitudes of independence and often by a peculiarly impersonal attitude toward life. During these periods the patient is free from asthma attacks. Then perhaps after a long interruption of the analysis the patient will begin to respond again to what we have already referred to as the seductive phase of the transference. Forbidden sexual impulses begin to be encouraged back into consciousness. Finally, however, the temptation becomes intense enough to

become again a threat to the patient's bond to the mother and another asthma attack is precipitated.

In connection with these withdrawal mechanisms it is interesting to note the reactions of our asthma patients to the danger of actual physical separation from the mother. It would seem that most asthma patients are particularly prone to attacks of asthma at times when they are undecided or in conflict as to whether or not to leave some mother figure. In the analysis we were early struck with the fact that asthma attacks tended to occur predominantly on weekends or during the period just preceding more prolonged interruptions of the analysis. Interestingly enough, during vacation periods, after the first few days, the patients were often singularly free from attacks. Then just prior to the resumption of the analysis the attacks would again begin to occur. In the history of these patients also there would tend to be a relative freedom from attacks during periods when the patients had actually achieved a physical separation from their mothers. It would seem that the precipitating situation is not the actual fact of separation from the mother but the indecision and conflict between the urge to cling to the mother and the need to separate from her.

A case of Dr. Eisler's presented at the beginning of the analysis a related type of defense in the form of mild compulsive symptoms. It was not difficult to determine the relationship between these compulsive symptoms and the deeper fear of losing the love of the mother. The compulsive neurotic symptoms had arisen by the familiar mechanism of introjection of the mother and internalization of the conflict. The rôle played by the mother in the deeper conflict was now played by the patient's conscience and in place of the fear of loss of the mother's love there appeared guilt reactions with an anal-erotic coloring, feelings of being impure. By means of this internalization of the conflict the patient protected herself from the danger of an acute estrangement from the mother. The patient developed asthma attacks for the first time in the analysis only after this compulsive neurotic purity complex had been interpreted and worked through.

Summing up our findings: We have concluded that the asthma attack is precipitated as a rule by a temptation which threatens to estrange the patient from a mother figure and our discussion of the numerous possible defenses against this conflict would suggest that the common feature in the otherwise divergent personalities of our asthma patients is the fact that the personality of these patients is built up in large part around the task of master-

ing by one means or another the patient's fear of being separated from the mother.

We may perhaps best conclude with a very few observations that have a bearing upon the relation between allergic and psychological factors in the etiology of bronchial asthma.

First we have seen that asthma attacks tend to be precipitated by emotional conflict situations of a rather uniform and typical character. It would seem extremely improbable that these emotional conflict situations should in every case have coincided with an allergic stimulus which was absent during the often rather prolonged periods during which the same patient's emotional withdrawal from the analysis was marked and during which he also was free from asthma attacks. Moreover a few patients experienced an initial relief from asthmatic attacks upon starting the analysis due apparently to the reassurance resulting from the analyst's interest in them and most of our patients developed an aggravation of the frequency and severity of their attacks as the analysis, after working through the defensive mechanisms, approached the fundamental conflict that we have been describing. In a few cases whose attacks had been confined to a particular season, asthmatic attacks appeared during the analysis also during other seasons of the year.

In some cases the substances to which the patients were allergically hypersensitive proved also to play a significant rôle in their psychological material. A patient of Dr. McLean's whose skin test was strongly positive for ragweed recalled that at the age of six he had lost his brother in a hay field where the brother remained all night before he was found. During the analysis the memory of this incident was frequently accompanied by severe wheezing. Other patients who showed marked hypersensitivity to cat hair also dreamed frequently of temptation situations in which the temptation was symbolized by a cat. Still another patient who expressed a violent conscious resentment of children displaced the greater part of her normal maternal interest upon cats and upon test developed an extreme allergic reaction to cat hair.

Finally, in a number of patients as a result of analytic therapy the asthmatic attacks were greatly relieved and we could observe during the analytic sessions the gradual replacement of the asth-

matic attacks by crying and then by a diminution of the fear of sexual temptation.

One patient of Dr. Wilson's who had previously shown a marked sensitivity to horse dander and in whom horses regularly elicited attacks of asthma became later able to ride horseback without any trace of asthma.

All these observations would suggest that psychological and allergic factors probably stand in a somewhat complementary relationship to each other in the etiology of bronchial asthma, that in some cases asthma attacks may be precipitated by allergic factors alone, in others by emotional factors alone and that in still other cases cooperation of allergic and emotional factors may be necessary to produce the attacks.

DISCUSSION.

Dr. Bernard Berliner (San Francisco, Calif.).—It has been stated by Fenglinger and other authors, that asthma is a pre-genital conversion neurosis and we heard from Dr. French the importance of the Œdipus situation in these cases, and we heard nothing about the importance of pre-genital factors.

I had the opportunity to analyze two similar cases. One was that of a young girl of sixteen who suffered from a very severe asthma and couldn't be helped in any way except by adrenaline injections which would give her temporary release. This asthma started when the girl was twelve years old. She was entirely healthy until then. She suddenly entered the living room and found her father in an intimate situation with a maid. The girl cried, almost fainted, was very much shocked, and since then she suffered from asthma. It took a long time of analysis until this scene became conscious to her, under great resistances, and then the asthma was cured, and during ten year's observation after this time, the young woman remained healthy.

The other case was that of a manifest homosexual man about the age of thirty, who, when he was a little boy, suffered from the lack of love from his mother, who was chronically ill, suffering from a disease, probably carcinoma, connected with a bad odor, and the boy had to attend the mother, do domestic services, and so forth, and had no love at all from his sick mother. He became manifestly homosexual later on, and the analysis showed his attacks were reminiscences of this scene with the sick and bad-smelling and unloving mother.

Dr. Franz Alexander (Chicago, Ill.).—I should like to say only one word regarding Dr. Berliner's remarks. The patients show again the importance of the mother relationship. He mentioned the pre-genital material, and I must say that there is no lack of pre-genital material in these cases.

You remember that Dr. French spoke of a conflict between a very dependent attitude upon the mother and sexual temptation. This dependence on the mother is the essence of every pre-genital relationship, so that the pre-genital conflict situation is there, and if Dr. Berliner missed it, this is probably because it is not a typically oral dependent attitude towards the mother but something which we like to describe as prior to the receptive attitude toward the mother, the help-seeking, the protection-seeking attitude; not the oral receptive attitude which we find in gastro-intestinal cases, but not in asthma.

It was interesting that Dr. Bernfeldt, who made his formulations entirely independently from ours, remarked the conspicuous absence of oral character trends in these patients and even anal character trends; and we find that this lack of oral material also is quite characteristic in these cases. It is not an oral dependence but an even more primitive, dependent attitude upon the mother's help, protection-seeking attitude, to hide by the mother, to lean on the mother, but not to receive from her.

As to the lack of anal material, we must make some qualification, because during the asthma-free periods there is much anal material, but usually those cases who suffer from acute asthma, with very frequent attacks, have no anal character trends nor anal material, because the anal material seems to be a defense; so when there is an abundance of anal material, usually there is no asthma, but it should not be understood that they never have any anal material or any anal characteristics.

Dr. Clarence P. Oberndorf (New York, N. Y.).—There is only one point which I should like to make and that is that the mechanisms which are described for various syndromes have considerable similarity in that repressed hostility seems to be accountable for most of them. Dr. Alexander has made some very fine discriminations between the type of hostility and the repression type of dependency, and I think that is very often a matter of judgment of the individual analyst.

If one might comment about the work which has been reported here by the Chicago school, one might possibly say this: that when one takes a series of diseases, asthma, mucous colitis, thyroid, eye troubles, and investigates them as single groups, one is very likely to place upon that particular group particular emphasis, to emphasize the factors which seem to be particularly pertinent to that group, and, in the investigation of any personality, one can find all these factors.

When one goes from bed to bed in a general hospital, one finds pretty nearly the same type of mechanisms which have been described here today as being specific for specific disease. My impression is that the underlying anxiety is the essential element and I would further say that when these anxieties are relieved, one often gets a striking relief of symptoms.

I am thinking of a case of mucous colitis which I was called to see after the patient received five blood transfusions, and was nearly dead. She was a Catholic girl of 25, unmarried, and she said there was nothing

wrong with her and, after I talked with her a little while, during the second interview, I said, "But there must be something wrong."

She said, "Why?"

I said, "Because you are biting your lip."

She said, "Yes, that is so."

Then came the whole flood of repressed hostility against the mother and the church. The mucous colitis ceased. All the bacteriological evidence of colitis had been carefully excluded. The girl began to take on weight and recovered after 10 interviews.

I could recall from an experience of over 20 years, innumerable such cases, but I will say that the underlying mechanisms in them all seemed to be very similar to those described by Dr. Alexander, possibly including those for mucous colitis also.

I should say that is possibly one of the main difficulties with the work which Dr. Alexander has so carefully elaborated over the period of years. He knows my viewpoints. We have discussed them on these occasions quite often, and I must say as the years have gone on in my own clinical experience, even with the idea of tending to corroborate what Dr. Alexander has pointed out, it seems to be that we are still very far from determining any specificity of emotional disorder and disease symptoms.

Dr. Thomas M. French (Closing).—I have just one or two points to make with reference to the discussion. Dr. Berliner's remark about the rôle of the pre-genital drives or urges, I would answer, in addition to what Dr. Alexander said, in the following way: That here we seem to see a pre-genital dependent attachment manifesting itself primarily in the ego attitude, while the Œdipus complex, the sexual problem at the early phallic phase seems to be the disturbing element.

This is a type of distinction which we in psychoanalysis, or in the literature previously have not been accustomed to dwell upon so much, but in the asthma cases I think it is quite an important one. A pre-genital attitude, clinging dependence to the mother, forms the basis of the ego's defenses, whereas the phallic urges are the disturbing situation.

To show the importance of that I might just remind you of the contrast between this and the formulation which Alexander made with reference to the gastric ulcer cases. Here we have similar forces in conflict with each other, but the difference is between which is on top and which is below, and that leads me directly, of course, to Oberndorf's question about specificity, and I think the point he makes is one that could be made with reference to all of psychoanalysis so far, that the conflicts we find in every patient, no matter what he is suffering from, are to some extent similar. One might say that they are the permutations and combinations of the same infantile conflicts, and that is what we would expect. We all have mothers and fathers and, basically, the same biological drives, and the differences between us are the quantitative differences in the nature of the drives, and also differences in the way they are organized.

Here, contrasting the asthma patients with the gastric ulcer cases, we have a very similar set of drives in action, but an entirely different organization. The asthma patient in his general attitude organizes his ego around the dependent tendencies and is threatened by the phallic impulses; in the gastric cases you have the ego organized around the competitive phallic impulses and the oral dependent wishes threatening.

I think it is impossible ever to attempt to arrive at some sort of specificity unless we turn away from just the enumeration of the types of impulses the person has, and turn to the questions of quantity and the questions of organization, organization even more important than quantity.

PATHOLOGIC CONSIDERATIONS ON INSULIN TREATMENT OF SCHIZOPHRENIA.*

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Although the biochemical approach is likely to offer the conclusive evidence for the explanation of the mechanism of action of insulin in the treatment of schizophrenia, there can be little doubt that pathologic investigations may add significant data. However, reports on brain changes of insulin treated patients are few in number.[1a, b, c, d] Therefore, the nature of the morphologic alterations that underlie the hypoglycemic symptoms related to the central nervous system is still little understood. It is the purpose of this paper to attempt an interpretation of the pathologic changes of hyperinsulinism and to comment upon certain aspects of the mechanism of action of insulin on the nervous system. The material was afforded by the histologic study of the brain of five schizophrenic patients who died during insulin shock treatment. A detailed report of the clinical course, and a comprehensive illustration of the pathologic findings of four of these cases have been published elsewhere [2] (*Psychiatric Quarterly*, 13: 207, 1939).

The most significant histologic alterations may be summarized under the two headings of neuron cells and blood vessels.

1. *Neuron Cells.*—Nissl's method disclosed that the majority of the nerve cells in the cerebral cortex appeared altered. A large variety of changes was observed ranging from chromatolysis and shrinkage to severe colliquation and formation of cellular shadows. Homogenizing alterations and Spielmeyer's "ischemic nerve change," were also frequently seen. Lipoid infiltration of the neuron cells was also found in four cases, particularly marked in the Ammon's horn.

There were marked alterations of the normal cyto-architecture of the cortex. Areas of almost total loss of nerve cells were observed extending at times over a centimeter in length. Their distri-

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bution was irregular; areas of relatively intact lamination were observed near areas of marked cellular destruction, the boundaries between the two zones being often abrupt. The glia at times had actively proliferated; at other times showed a slight reaction. These areas of "cortical devastation" were seen especially in two cases. In all cases, however, dropping out of cortical neurons was observed, either diffusely throughout the six laminæ of the cortex or involving more markedly the superficial layers. The glia showed some hyperplasic reaction and ameboid changes were occasionally present. There was apparently slight increase of microglia cells while the oligoglia showed acute swelling. Another type of cyto-architectural alteration consisted of small, roundish, more or less sharply circumscribed areas of cellular waste, involving one or more layers. The majority of neuron cells had disappeared, the few remaining showed severe changes, usually of the ischemic-homogeneous type.

There was also complete "dropping out" of the nerve cells of the lamina pyramidalis of the Ammon's horn.

2. *Blood Vessels.*—These showed striking changes. There were areas in which a large number of proliferated small vessels and capillaries were present either in the whole width of the cortex or in some layers of it. High power examination of the altered vessels showed that the vascular walls were considerably thicker than normal, due to proliferation of cellular elements. Both endothelial and adventitial cells appeared enlarged and actively proliferating. The nuclei were swollen and the granuli of chromatin were numerous and darkly stained. Varying phases of karyokinesis were occasionally noted. It was not infrequent to observe pictures which could be interpreted as budding formation of new capillaries. The proliferative changes of the vessel walls resulted frequently in narrowing and eventually in obliteration of the lumen. In the areas of major vascular proliferation the neuron cells had disappeared; however, "productive" vascular changes were occasionally noted also in areas of relatively well preserved neuron cells.

Two of our cases showed these areas of vascular proliferation and neoformation irregularly diffuse throughout the cortex. In another case, proliferation of adventitial and endothelial cells was seen occasionally but no areas of vascular neoformation could be detected. Finally, two cases showed no morphologic changes of

the blood vessels. The case showing the most marked blood vessel proliferation had reecived 11,400 units of insulin in 44 injections, a much higher dose than the one received by the other patients.

From this summary description it appears that severe lesions of the neuron cells are an outstanding feature in hypoglycemia, thus confirming the findings of other investigators.[1a, b, c, d] It will be noted that severe cellular changes were also found in the brain of diabetics who died in hypoglycemic coma following over-dosage of insulin (Wohlwill,[3] Bodechtel[4]) and in spontaneous hyperinsulinism (Terbrüggen,[5] Moersch and Kernohan[6]). To be sure, such cellular changes might be due to comatose conditions preceding death and lasting, occasionally, several days. However, that hyperinsulinism plays a certain rôle appears from a comparison of two of our patients who died following a coma of the same duration; in the one patient who received over 10 times more insulin the lesions were markedly more pronounced. The conclusion, therefore, that severe insulin shocks destroy a considerable number of cerebral nerve cells appears well grounded.

The mechanism by which these cellular changes are brought about remains a problem for investigation. Such widespread and severe degenerative changes of the neuron cells as seen in numerous sections of the present cases are commonly regarded as histologic evidence of a toxic action. In fact, the opinion of a direct toxic damage of the nerve cells by insulin has been expressed by Kobler.[1b] However, it seems preferable to replace the rather vague notion of "toxicity" by a more clearly defined concept indicating impairment of some specific cellular function brought about by the toxic substance. As far as insulin is concerned, there is much evidence in favor of the opinion that the respiratory activity of the brain cells is depressed in hypoglycemia, the effects of glucose deficiency on the nervous system being similar to those of oxygen deficiency.[7] Hence, insulin might be considered toxic inasmuch as it interferes with the oxidative mechanism of the nerve cells.

A further question arises as to what factors are responsible for oxygen deficiency. It is likely that anoxia is partly due to inability of the nerve cell to utilize oxygen in the absence of glucose which is essential to the oxidative activity of the neuron cells (Holmes,[8] Wortis[9]). The investigation of such chemical factors is obviously beyond the possibilities of morphologic pathology.

The study of our pathologic specimens offers strong evidence in favor of an additional mechanism which appears to play a rôle in the causation of anoxia. Some of the histologic features above referred to, such as focal areas of cellular waste in the cerebral cortex, dropping out of the cells of the Sommer sector, "ischemic" and homogenous changes of the neuron cells may be considered as indicative of vascular disturbances. As a matter of fact, on histological evidence of this type, Spielmeyer and his school base the diagnosis of "vascular" disease of the central nervous system. Similar lesions, in fact, have been reported in a variety of brain conditions which have as a common pathogenic mechanism, vascular disturbances. There is considerable evidence in support of the opinion expressed by Ricker,[10] and Spielmeyer [11] that damage of the nervous system of this type may occur without morphologic alterations of the blood vessels. Functional vascular disturbances are regarded as the pathogenic factors playing a causal rôle. It is well grounded, therefore, to assume that alterations of the vascular function might explain satisfactorily important pathologic features of our cases. It should be noted that a similar, though less marked "vascular" type of brain pathology has been found in patients who died in hypoglycemic coma (Bodechtel,[4] Morsier and Mozer,[12] Leppien and Peters [13]) and in experimental hyperinsulinism (Stief and Tokay [13]). An interpretation based on the hypothesis of vascular disturbances, functional in character, has already been offered by these investigators.

The study of the present material, however, while confirming the indirect evidence of functional changes based on the type and distribution of the nerve cells changes, offers direct evidence of the vascular origin of the lesions based upon actual morphologic alteration of the blood vessels. The striking pathologic changes of the brain blood vessels above described, seem to indicate a direct damaging action of insulin or some other substance formed in hyperinsulinism, upon the cells of the vessel walls. The reaction of the adventitial and endothelial elements appears to be mainly hyperplasic and, to a lesser extent, degenerative in character. Neoformation of capillaries may be explained partly as a secondary vascularization of an altered tissue that is undergoing new organization; partly, however, as a direct effect of proliferative stimuli, since cellular proliferation of vessel walls was also observed in the

absence of major parenchyma destruction. The pathologic features of the vascular changes bear striking similarities to the "productive type" of vascular reaction found in lead "encephalitis" (Bonfiglio,[14] Hassin[15]). This type of vascular changes has not been described as yet in insulin treated patients, although in experimental hyperinsulinism Dunner *et al*,[16] and Accornero[17] have mentioned the finding in the central nervous system of proliferation of vascular endothelium and neoformation of capillaries.

It seems reasonable, in conclusion, to assume that the cellular waste of the brain tissue in severe hypoglycemia is brought about by a mechanism of anoxia in which not only biochemical factors but also vascular alterations appear to play a rôle. In the majority of cases of hypoglycemic shocks, the vascular reaction is likely to be functional in character at the beginning. However, high doses of insulin may produce proliferative changes in the wall of the vessel, leading to narrowing and obliteration of the lumen. In both functional and anatomical vascular alterations, the quantity of oxygen conveyed to the tissue by the blood vessels is forcibly reduced and cellular anoxia, eventually leading to destructive changes of the nerve cell, is the ultimate result.

Functional disturbances and productive changes of the blood vessels might be regarded as two successive stages of the same process; evidence has been offered, in fact, in favor of the hypothesis that angiospastic states of long duration and frequently repeated may determine endarteritic proliferation.

BIBLIOGRAPHY.

1a. Leppien, R., and Peters, G.: Todesfall infolge Insulinshockbehandlung bei einem Schizophrenen. Ztsch. Neur. u. Psychiatr., 160: 444, 1937.

1b. Kobler, F.: Histologischer Gehirnbefund nach Insulinkoma. Arch. f. Psychiatr., 107: 688, 1938.

1c. Kastein, G. W.: Insulinvergiftung. Ztsch. Neurol. u. Psychiat., 163: 322, 1938.

1d. Cammermeyer, J.: Über Gehirnveränderungen enstanden unter Sakelscher Insulintherapie bei einem Schizophrenen. Ztsch. Neur. u. Psychiat., 163: 617, 1938.

2. Ferraro, A., and Jervis, G. A.: Brain pathology in 4 cases of schizophrenia treated with insulin. Psychiat. Quarterly, 13: 207, 1939.

3. Wohlwill, F.: Über Hirnbefunde bei Insulinüberdosierung. Klin. Wchnschr., 7: 344, 1938.

4. Bodechtel, G.: Der hypoglykämische Schock und seine Wirkung auf das Zentralnervensystem. Deutsch. Arch. Klin. Med., 175: 188, 1933.
5. Terbrüggen, A.: Anatomische Befunde bei spontaner Hypoglykämie. Beit. Path. Anat., 88: 37, 1936.
6. Moersch, F. P., and Kernohan, J. W.: Hypoglycemia. Neurologic and neuropathologic studies. Arch. Neurol. and Psychiat., 39: 242, 1938.
7a. Dameshek, W. A., and Myerson, A.: Insulin Hypoglycemia. Mechanism of the neurologic symptoms. Arch. Neur. and Psychiat., 33: 1, 1935.
7b. Himwich, H. E., Bowman, K. M., Wortis, J., and Fazekas, J.: Brain metabolism during the hypoglycemic treatment of schizophrenia. Science, 86: 271, 1937.
7c. Glickman, N., and Gellhorn, E.: The effects of oxygen deficiency on the sensitivity of rats to insulin. Am. J. Physiol., 121: 358, 1938.
7d. Ingraham, R. C., Moldavsky, L., and Gellhorn, E.: On the relation between hypoglycemia and anoxemia. Am. J. Physiol., 119: 341, 1937.
8. Holmes, E. G.: Oxidation in central and peripheral nervous tissue. Biochem. J., 24: 914, 1930.
9. Wortis, S. B.: Respiratory metabolism of excised brain tissue. Arch. of Neurol. and Psychiat., 33: 1022, 1935.
10. Ricker, G.: Pathologie als Naturwissenschaft. Berlin, 1924.
11. Spielmeyer, W.: Kreislaufstörungen und Psychosen. Ztsch. Neurol. u. Psychiat., 123: 536, 1930.
12. Morsier, G., and Mozer, J. J.: Lésions cérébrales mortelles par hypoglycemie au cours d'un traitement insulinque chez un morphinomane. Ann. de Med., 39: 474, 1936.
13. Stief, A., and Tokay, L.: Beiträge zur Histopathologie der experimentellen Insulinvergiftung. Ztsch. Neur. u. Psychiatr., 139: 434, 1933.
14. Bonfiglio, F.: Circa le alterazioni della corteccia cerebrale consequenti ad intossicazione sperimentale da carbonato di piombo. Nissl Alzheimer Histol. Arb., 3: 359, 1909.
15. Hassin, G. A.: Contrast between brain lesions produced by lead and other inorganic poisons and those caused by epidemic encephalitis. Arch. Neurol. and Psychiat., 6: 268, 1909.
16. Dünner, L., Ostertag, B., and Thannhauser, S.: Klinik u. pathologische Anatomie der Insulinvergiftung an Tieren. Klin. Wchschr., 12: 1054, 1933.
17. Accornero, F.: Experimental histopathological researches on insulin shock. Am. J. Psychiat., 94: (Suppl.), 130, 1938.

CEREBRAL DAMAGE IN HYPOGLYCEMIA.

A Review.*

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That hypoglycemia can produce cerebral damage has been fairly well demonstrated by observations both on human material and in the experimental animal. These reports are widely scattered throughout the literature and some of them, no doubt, have escaped the attention they properly deserve. Certainly with the increasing use of insulin shock therapy as a therapeutic agent in so many conditions (morphinism, schizophrenia, affective disorders, neuroses, etc.) a review of these studies seems very much indicated.

It is well known that when the blood sugar is lowered sufficiently a syndrome is provoked which is marked by a most varied, severe and complex symptomatology. The neurological and psychiatric phenomena suggest an extensive disintegration of cerebral function. The profound clinical manifestations make one wonder how these patients are able to withstand such severe reactions without some ill effect upon the nervous system. And, as a matter of fact, damage to the nervous system is not as uncommon as the sporadic reports would lead one to believe. Fatal outcomes from hypoglycemia have filtered into the literature for years, but the slow recoveries and the permanent functional impairments have not been emphasized or possibly not reported, although such cases must occur. From a review of the literature and a study of our own cases it seems very apparent that continued repeated hypoglycemia may definitely produce a depression of the cerebral function and even an irreversible degeneration of the brain tissue and cells, resulting in slow recovery or even in permanent functional damage. These cases in our experience are even more numerous than the fatal ones and are of extreme importance because they tend to indicate the borderline in the reversibility of the cerebral changes. Some of these cases will be reported as supplementary studies in this report.

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The clinical picture of the cerebral disorder during hypoglycemia is extraordinarily varied and often fantastic, and has been the subject of numerous reports (Wilder *et al.*,[1] Bowen and Beck,[2] Moersch and Kernohan,[3] Morsier and Bersot,[4] etc.). The prodromal symptoms usually consist of nervousness, perspiration, pallor, tremor, sleepiness and salivation. These may clear up or progress in severity. The neurological manifestations begin with restlessness, diplopia and muscular twitchings of the face and limbs, and then pass through various stages of motor involvement with the appearance of hemiplegias, Jacksonian attacks, epileptiform crises and clonic and tonic convulsions. Numerous other focal findings may occur such as aphasia, disorders in sensation, visual disturbances (hemianopsia, complete blindness), auditory disorders and disturbances in coordination. If the symptoms deepen, the patient passes into a stupor. The reflexes are variable in the extreme. Occurring alone or in association with these neurological manifestations, there frequently appear psychic disturbances. Early these consist of fears, anxieties, mild depressions and difficulty in concentration. The speech becomes thick and there result peculiar grimacing, unmotivated laughing and crying, hebetude, garrulity, compulsions, fugues and hallucinatory states. Eventually, after varying degrees of confusion and clouding of the sensorium, consciousness is lost and a complete amnesia concerning the attack often ensues.

It seems that hypoglycemia or hyperinsulinism, regardless of the cause, produces similar cerebral symptoms and brain tissue alterations. Four sources of material are now available for summarizing the recorded cases of brain damage resulting from a lowered blood sugar. These are as follows:

1. Insulin shock in diabetic individuals.
2. Spontaneous hypoglycemia secondary to neoplasm or functional overactivity of the islands of Langerhans.
3. Insulin shock therapy in schizophrenia, morphinism, etc.
4. Experimental production of hypoglycemia in animals.

"Insulin Shock" in Diabetics.

Ever since the introduction of insulin as a therapeutic agent in diabetes, the danger of overdosage has been very evident. The fluctuation of the patient's tolerance to the drug, individual care-

lessness in the administration of the insulin, and often the lack of recognition of an insulin reaction, have resulted in severe hypoglycemia and in definite cerebral damage or even death. Joslin [5] was the first to report such fatalities in four cases but the cerebral lesions were not discussed. Many subsequent deaths have been recorded (Heimann-Trosien and Hirsch-Kauffmann,[6] Campbell and Macleod,[7] Pemberton,[8] Dahl [9]), but in only a few have the brain changes been studied.

The first detailed study of the brain changes in diabetics dying from insulin shock was reported by Wohlwill [10] in 1928. His first case was a 62-year-old female, while the second was a 29-year-old male. The nervous system revealed primarily ganglion cell injury. This consisted of a tigrolysis, fragmentation of the cell cytoplasm, irregularity of the cell outline and pyknosis of the nuclei. Some diffuse swelling of the axis cylinders was also observed. Glial proliferation was only moderate but a definite vacuolization of the glial cells as well as pyknosis of their nuclei had occurred. The changes were uniform in all sections of the brain.

Somewhat similar changes were recorded by Terplan [11] in 1932 in a 16-year-old boy with diabetes. The patient passed into insulin shock and died in three days in spite of well-regulated treatment. The autopsy revealed an extreme edema of the brain with a marked injection of the cerebral and meningeal vessels. Sections showed very severe changes in the cortex consisting of colliquation of the ganglion cells, especially of the third layer. The greatest alterations were observed in the occipital and frontal lobes, the island of Reil and hippocampal gyrus. The glial cells presented severe regressive changes of ameboid type. There was swelling of the capillary endothelium.

In the following year Bowen and Beck [2] described the cerebral changes in a 16-year-old diabetic who was brought into the hospital in hypoglycemic shock and within a period of two hours had had 50 convulsive seizures, cyanosis and a rigidity of extremities. In spite of the administration of large doses of glucose this patient remained unconscious and died in four days. The meninges and brain tissue were very hyperemic. There were small hemorrhages scattered throughout the leptomeninges of the cerebellum and spinal cord. No microscopic changes were described. These same authors quoted a case of White and Smith,[12] in which an 8-year-old diabetic

received an overdosage of insulin and in spite of glucose administration became irrational, developed convulsions and died in 24 hours. The brain contained scattered foci of degeneration with edema and scattered lymphocytes and oligodendroglia. In many places a marked ependymal edema had occurred with foci of dislodgment of the ependyma.

Lindsay *et al.*[13] described even more striking changes in a 50-year-old diabetic woman who had had repeated mild attacks of hypoglycemia over a period of six years. She was found in coma and died eight days later. At autopsy punctate hemorrhages and a cystic space 1.5 cm. in diameter were observed in the left cerebral hemisphere. The cyst was lined by a fine fibrinous material. Similar hemorrhages and smaller cysts were present in the right hemisphere. There was also a moderate perivascular glial increase.

In 1938 Bodechtel [14] again emphasized the acute cell changes in hypoglycemia. He studied the cerebral damage in a 41-year-old diabetic. Hardly a normal nerve cell was visible. The cell fragments and degenerated cells presented a complete chromatolysis and homogeneity. Glial elements also showed regressive changes while the endothelial cells of the blood vessels were swollen and pyknotic. The axis cylinders were swollen. Even the Purkinje cells of the cerebellum had undergone a definite homogeneity.

In a previous publication [15] we also described the cerebral changes in a diabetic male aged 64 years who because of a varied sugar tolerance was extremely difficult to regulate and had suffered repeated mild attacks of hypoglycemia. Prior to his last admission this patient was erroneously given 80 units instead of 40 units of insulin and immediately passed into a deep stupor with convulsive movements. He failed to respond to intravenous sugar and died eight days later. Grossly the brain revealed petechiæ and an occasional cyst formation. Microscopic studies showed moderate nerve cell alteration, many scattered petechiæ, areas of encephalomalacia and cyst formation and finally diffuse generalized glial proliferation and additional extensive localized gliosis around the areas of softening. The glial proliferation was one of the most striking changes observed and had replaced large regions of brain tissues.

In this group of cases of insulin shock in diabetics it was often difficult to determine just how numerous the hypoglycemic reactions had been. Most of the investigators were impressed by the nerve

cell alterations which no doubt were the result of the terminal cerebral insult. In a few cases, including our own, definite attention was given to changes other than those in the nerve cells, and it soon became apparent that myelin and glial alterations did occur very prominently. These latter changes do not occur acutely and hence would be present only some time after the onset of the cerebral insults.

To this group can now be added three more cases observed in our clinic. Two are still alive although handicapped by varying neurological and psychiatric disabilities. If we can draw any conclusions from our own experience, there seems to be no question that many diabetics yearly must either die or develop varying disabilities as a result of hypoglycemic reactions, but for some reason these cases have not found their way into the literature. Reports of such cases would be of extreme value in helping to understand the action of hypoglycemia upon the brain tissue.

Case I.—A white male, aged 39 years, had been a known diabetic for 20 years. He was fairly well controlled with insulin although he occasionally manifested mild reactions. About one year prior to his death he was placed on protamin insulin. Following one of these injections he suddenly collapsed and expired a few hours later. The brain was negative on external examination. Coronal sections revealed numerous petechiæ scattered subependymally along the lateral ventricles. In the left occipitoparietal region there was a circumscribed clump of hemorrhages. Histologically the brain changes were relatively mild and consisted primarily of a glial proliferation, chiefly in the white substance. This proliferation was diffuse without formation of glial nodules. The vessels showed some bleeding with an occasional large destructive extravasation scattered here and there with no tendency to localize. Throughout, there was a tremendous distention of the perivascular spaces and mild vacuolization of the white substance with small cavity formation. The nerve cells showed some shrinking, but this change was generalized and probably of questionable significance.

Case II.—H. E. (H. N. 643612), a 15-year-old female, had been a diabetic for 11 years and had had mild insulin reactions from time to time. She was admitted to the hospital primarily for regulation on protamin insulin. She was started on 40 units, which was increased to 50 units and resulted in a mild insulin reaction which seemed to respond to orange juice and glucose. The following day, however, the patient seemed hard to awaken. By noon she was semi-comatose, roused only with difficulty and developed peculiar jerky movements of the right side of her face and body. She was given glucose but did not respond very well. The jerking movements increased in severity. The blood sugar was 35.5 mg. per cent. Neurological examination revealed the patient to be in a deep stupor. Her reflexes were slightly in-

creased on the right side, and the Babinski's sign was positive on the right. Glucose was administered steadily and the patient gradually improved over a period of days. As improvement ensued it was observed that the patient had a marked mixed aphasia and a right-sided paresis. Improvement continued although at the time of discharge three months later, she still showed the aphasia and paresis. She was seen again six months after the original hypoglycemic reaction. There was still a slight weakness of the right upper extremity with a slightly hyperactive biceps reflex. Lower extremities were now normal. The abdominal reflexes on the right side were reduced. There had occurred a definite improvement in speech although even at this time the patient was able to use and understand only the simplest words and phrases. Reading was not improved.

CASE III.—V. C. (H. N. 672417), a white male, aged 39 years, was a known diabetic for seven years. He was taking his insulin irregularly and had not adhered strictly to his diet. About 10 days prior to his admission he did not feel well, and since this was ascribed to his diabetes he was given greater amounts of insulin by his family. He received 120 units over a period of 12 hours. He then retired, and shortly afterwards his sister discovered the patient throwing pillows about his room. He was very restless but mute and gradually passed into a stupor. On admission he was semistuporous, his eyes were open, and he mumbled unintelligibly. To all queries he nodded his head in assent and grinned foolishly. Neurological examination at this time was unsatisfactory because the patient was uncooperative and resistive. Pupils were equal, regular and reacted to light. His mouth was drawn to the left. All deep reflexes were reduced. During the next few days he continued in about the same state. Glucose intravenously succeeded in quieting his motor restlessness but at first did not seem to help his mental state. After 10 days he began to improve slowly although he remained more or less noisy, uncooperative, incontinent and disoriented. After a number of weeks, contact could be established with the patient. He appeared brighter and more cooperative but continued to be confused and had a tendency to be euphoric. There was a definite paucity of ideas and his speech was somewhat irrelevant. The patient was discharged after two months of hospitalization. At the time of discharge the neurological examination revealed a reduction of the deep reflexes and some generalized weakness of all the musculature, although no definite paralysis was present. He complained of a numbness of both hands and lower forearms; however, no objective sensory disturbance could be elicited. There was still some definite evidence of mental aberration. He was able to talk slowly and engage in the simpler occupational and recreational activities. Psychometric studies revealed on the Stanford-Binet scale a rating of 55. He was discharged to his family as it was believed that he had improved sufficiently to continue outside of an institution under proper supervision.

Comment.—These last two cases tend to demonstrate the borderline in the reversibility of the cerebral alterations in hypoglycemia. Whether complete return of function will ever occur is questionable,

but certainly in view of the long persisting neurological and psychiatric disturbances one suspects very strongly that some irrevocable damage has occurred to the cerebral tissues.

Spontaneous Hypoglycemia Secondary to Neoplasm or Functional Overactivity of the Islands of Langerhans.

Hypoglycemic attacks appear spontaneously when the pancreas shows an increase in island tissue, a diffuse hypertrophy or circumscribed neoplasms either benign or malignant. They have also been reported in cases showing no visible evidence of increased islet tissue. Harris [16] in 1924 suggested that a clinical condition of hyperfunction of the islands of Langerhans ought to produce the same symptoms as those observed after an overdose of insulin. It remained for Wilder and his coworkers [1] to record completely the first case in which the clinical data, chemical studies and pathological examinations were correlated. Their patient was a 40-year-old physician who at autopsy showed a carcinoma of the islet cells of the pancreas. Since this study many similar cases of hypoglycemia have been reported. However, in most of them the primary interest has centered about the possibilities of therapy and surgical treatment, rather than upon the occurrence and character of the cerebral damage. These earlier observations have done much to establish the identity of this new disease syndrome which, in later reports only, was described as presenting definite subjective and objective symptoms and anatomical findings of cerebral damage. This series of cases is of special importance since in most of them the hypoglycemic reactions had extended over a known period of time and the pathological changes were due, at least in part, to a chronic process.

Before proceeding with a description of the cerebral alterations it might be well to mention briefly some of the non-fatal cases in which functional impairment and slow recoveries followed some of these hypoglycemic insults. Howland and his coworkers [17] described a case of a 52-year-old female who had suffered from frequent attacks of coma and convulsions often associated with a left-sided hemiparesis and a thick slurring speech that persisted for days. At operation a carcinoma of the pancreas was removed.

Best and his coworkers [18] studied a 44-year-old male who complained of frequent convulsive seizures. In one such attack the patient remained rigid and unresponsive in spite of glucose therapy, and it was not until the ninth day that the patient became oriented. Here also a carcinoma of the pancreas was found at autopsy. Wolf *et al.*[19] recorded a rather acute onset of so-called spontaneous hypoglycemia in a young male who was admitted to the hospital confused and dazed. During his hospital stay he presented transient recurrent attacks of right-sided hemiplegia which persisted for days. Blau *et al.*[20] reported a rather striking case which occurred in a 49-year-old male who had had numerous severe hypoglycemic and convulsive seizures over a period of some 16 months. During his stay in the hospital this patient's symptoms became progressively worse and he developed a stiff gait, retropulsive tendencies, tremors and a dysarthric, thick, slow whining speech so characteristic of an extrapyramidal involvement. His mental condition was one of a progressive intellectual and emotional deterioration in spite of the maintenance of a normal blood sugar. Anderson [21] also described a very tragic case of a 33-year-old male with an adrenal tumor who had had two attacks of hypoglycemia prior to his admission to the hospital in severe shock. In spite of glucose therapy, four days later the patient's speech was still slow and deliberate, and he had a great deal of trouble in remembering recent events. After discharge this patient again went into shock and expired. A similar type of case was recorded by Smith and Seibel [22] in a patient with a circumscribed adenoma of the pancreas. This individual, for more than two years, had had frequent dizzy spells with an occasional loss of consciousness. After recovery from the last of these spells he noticed that his speech was slower than formerly and that his mental ability had decreased. He eventually became very unstable emotionally. Five years after the onset of his illness he was again admitted unconscious and died four days later from pneumonia.

The above cases are of special significance because they demonstrate, except for the last two, the non-fatal type of reaction where the threshold of irreversibility in the cerebral function is so closely reached that the disabilities resulting from the hypoglycemia persist for days, weeks or even become progressive, resulting in definite permanent disabilities. In many cases, as in the last two listed, the

cerebral threshold of reversibility is later exceded and there results a fatal termination.

Besides these non-fatal cases, many reports have appeared describing the actual brain changes in spontaneous hypoglycemia secondary to neoplasm or functional overactivity of the islands of Langerhans. One of the first of such studies was recorded in 1929 by McClenahan and Norris [23] in a 41-year-old male who for 1.5 years had had attacks of amnesia lasting a short time and disappearing spontaneously. He was finally admitted in coma and died of a bronchopneumonia. A pancreatic adenoma was found at autopsy. The brain revealed evidences of meningeal and cerebral irritation manifesting itself in the form of slight perivascular round-cell accumulations.

Terbrüggen [24] in 1931 studied a patient with frequent hypoglycemic reactions, who passed into a deep coma during one of her reactions and died in spite of therapy. The cause of the hypoglycemic attacks was numerous pancreatic adenomas containing island tissue. The brain was hyperemic and contained an occasional perivascular hemorrhage. The nerve cells presented a complete chromatolysis with intact nuclei but an extensive infiltration of the cell body by fat droplets. The glial elements were free of fat droplets but did show a few mitotic figures and a pyknosis of their cell bodies.

More severe cerebral damage was observed by Wolf and his coworkers [19] in a young child with an adenoma of the pancreas. This patient had suffered from atypical convulsive seizures for 18 months, occurring three to four times weekly. Each seizure persisted for about seven minutes. The patient was admitted to the hospital in a semicomatose state and began having clonic convulsions which terminated fatally in 72 hours. The brain tissue contained a scattered loss of cells throughout the second and third layers of all cortical areas. There was a thickening of the external glial membrane and a mild scattered gliosis of the zonal layer. The pons showed a mild early granular ependymitis and a subependymal gliosis.

Moersch and Kernohan [3] in 1938 reported the cerebral changes in two individuals dying in spontaneous hypoglycemia. The first was a 35-year-old farmer who had had periodic attacks of confusion over a period of a year. He was admitted semistuporous

and died of a terminal pneumonia. The second patient, aged 47, had had hypoglycemic spells for four years. In both cases the brain changes were predominantly limited to the nerve cells which showed a widespread degeneration especially in the regions of the cerebral cortex and diencephalon. There were practically no normal nerve cells in the cerebral cortex. The cell dendrites were swollen, fragmented or absent, chromatolysis was complete and many ghost cells were observed. The cell nuclei were swollen and eccentric. In certain devastated areas no cells remained and there was a degeneration of the underlying white substance.

That same year there appeared a most significant report by Malamud and Grosh [25] of a 30-year-old female who had suffered repeated "fainting spells" for six years. These gradually increased in severity and after four years had produced in the patient a definite personality disturbance and mental retardation. It was finally necessary to commit the patient to a state hospital where she later died from a bronchopneumonia. At autopsy an adenoma was found in the pancreas. The cerebral cortex of both hemispheres showed a distinct parenchymatous degeneration. Some of the nerve cells were swollen, homogeneous and pale while others were completely destroyed leaving areas of acellular cortex. There was a demyelinization of certain cortical areas with a reduction of the tangential and radial fibers. Only a slight rarefaction in the myelin sheaths of the white substance was observed. Microglial proliferation was marked in certain areas while glial proliferation was moderate but definite. The left cerebral hemisphere was the most extensively involved. This report is most important because it illustrates the actual clinical effects of the chronic brain damage resulting from these repeated hypoglycemic shocks. Moreover, it was possible to demonstrate pathologic alterations associated with this clinical change.

In a series of publications [15, 26] we also have reported four cases of spontaneous hypoglycemia (two with adenomas of the pancreas, one with a carcinoma, and one with a functional overactivity of the islands) in which the cerebral tissues were studied in an attempt to observe the chronic effects of hypoglycemic insults upon the brain substance. These patients ranged in age from 27 to 60 years and manifested hypoglycemic symptoms over a period of one to four years. The frequency and severity of the hypoglycemia varied

greatly from case to case. One of the patients merely complained of spells of nervousness for a year prior to the onset of her terminal illness, while some of the other patients had suffered very severe and repeated reactions with convulsive seizures and deep stupor. The cerebral alterations in all cases were fundamentally alike although of a varying severity. Cerebral petechiæ were prominent and ranged from small discrete extravasations to large hemorrhages that destroyed the surrounding brain tissue. There were also observed large areas of encephalomalacia, demyelinization and cyst formation. Many small glial-lined cavities were found as well as huge gross areas of softening. The most significant change, however, seemed to be the diffuse, generalized glial proliferation and the extensive gliosis around the areas of softening. In one of the cases this glial proliferation was so extensive that it had replaced large areas of brain tissue. Although some atrophy and disappearance of nerve cells was observed, this cell change was overshadowed by the others described.

This entire group of cases of hypoglycemia secondary to tumors or functional overactivity of the islands of Langerhans is of special significance because as a whole they present a beginning in the study of the more subtle and chronic alterations that might result from these cerebral insults. Most of the patients in this group had suffered repeated attacks of hypoglycemia long before the identity of their ailment was recognized. Those which were followed for some time and were studied pathologically revealed cerebral changes that to us seemed even more significant than the acute and chronic nerve cell damage that was so prominent in those cases of insulin shock in diabetics. These changes consisted of hemorrhages, myelin damage and glial proliferation. It is these alterations that must influence the terminal picture in those long-standing cases of repeated hypoglycemia which terminate in clinical and pathological lesions within the central nervous system.

Insulin Shock as a Therapeutic Agent.

The final group of cases providing suitable material for a study of the cerebral lesions in hypoglycemia has appeared only recently in the literature and is the result of the increasing use of insulin shock as a therapeutic agent. With such a procedure it was in-

evitable that certain fatalities should occur, since at present our knowledge concerning the threshold of the individual variations in tolerance to large doses of insulin is limited. These most interesting reports have aided materially in confirming one's impression concerning not only the possibility of the damage that might be produced to the nervous system, but also the necessity of extreme caution that ought to be used in the administration of this drug in large doses.

Membrez and Razdoljsky [27] reported two cases in which insulin was used to improve the patient's general health. Both individuals showed persisting neurological findings many days after recovery from the insulin shock, and both succumbed to a later injection of the drug. The autopsy findings were very incompletely described and no mention was made of the cerebral changes.

In 1936 Morsier and Mozer [28] described the cerebral changes in a 41-year-old physician who had been a morphine addict and who was using insulin injections as a part of the cure. He took an overdose of the drug and passed into coma from which he did not recover. The cerebral vessels showed a marked congestion with an occasional petechial hemorrhage. The nerve cells of the brain were shrunken and had lost their Nissl substance. The most striking pathological finding was a glial proliferation that occurred especially around the vessels.

The first description of the cerebral alterations in schizophrenia following insulin shock therapy was recorded by Salm [29] in 1937. His patient during the course of her treatment suddenly went into coma and could not be aroused. Death occurred on the eleventh day of coma. The brain at autopsy contained hemorrhages about the third ventricle, within the substantia nigra and in the vegetative nuclei. In the cortex the nerve cells showed widespread ischemic changes. Moderate gliosis was also observed.

A similar case was described by Kobler.[30] His patient was a 24-year-old female, a schizophrenic who was given insulin shock therapy according to the method of Sakel. After a period of treatment the patient suddenly went into severe coma and expired. Her spinal fluid was bloody. Gross examination of the brain revealed an extensive hyperemia with some petechiæ. The ganglion cells were altered and showed some tigrolysis and vacuolization of the cell body. There were many sclerotic cells as well as many structureless ones.

Finally Freed and Wofford [31] in 1938 reported a non-fatal case of a 30-year-old schizophrenic who, two weeks after the onset of a course of insulin shock therapy, passed into coma and did not respond to the usual types of restorative therapies. A spinal puncture performed five hours later showed a bloody fluid. The following day the patient was still comatose and presented a hypertonus of the left arm with absent deep reflexes in the left leg, positive Babinski on the left and bilateral positive Kernig's sign. Six days later the spinal fluid was still bloody. The patient gradually recovered during the next three weeks.

This group of cases is still limited but this is probably due to the failure to study and report the brain changes in some of the resulting fatalities. That fatalities do occur and remain unpublished is proved by the author's knowledge of some of the local cases. It must be kept in mind that a large number of individuals are receiving this type of therapy and although many appear to have no untoward effects, still it seems very probable that some of them must terminate with functional damage although the patients actually survive the effects of the hypoglycemic reaction. One such case has recently come to our attention and will be briefly recorded.

E. M., a 30-year-old white female, was admitted to the hospital with a diagnosis of schizophrenia. Three years previously she had had a similar attack from which she recovered in about six weeks. At the time of admission the patient had been ill for about one year. She would seldom speak unless spoken to and believed that everyone was against her. She was careless about her appearance and very suspicious of her food. The patient was given insulin shock therapy according to the method of Sakel. She did not go into coma until 180 units had been given. In her eighth period of coma she could not be aroused in spite of intravenous glucose and adrenalin. She remained in coma for about 24 hours and then gradually improved, although for weeks following she had unequal pupils, difficulty in swallowing, involuntary urination and a high grade confusion which was different from anything which she had previously manifested. The most characteristic feature of this confusion was the misidentification of persons.

Her confusion and overactivity continued and one month later it became necessary to transfer the patient to a state hospital.

Experimental Hypoglycemia.

The production of hypoglycemia in animals has proved to be a very convenient method of investigation because the procedure is

quite simple and the dosage of the drug is easily controlled. Most investigators have used either dogs or rabbits and, over a period of years, have described the most varied and the most extensive changes in the brain tissue in these animals. These investigations have been carried out by Schereschewsky *et al.*,[32] Dünner,[33] Stief and Tokay,[34] Gani,[35] Schmid,[36] Weil *et al.*,[37] and Accornero,[38] and their results were recently reviewed in some detail in a paper by the author. In that review an article by Accornero was overlooked. He used dogs and gave some of them a few shocks at close intervals allowing the animals to die in coma. These showed very acute diffuse alterations of the nerve cells. Some cells were pyknotic, fragmented and appeared to be irreversibly damaged. The astrocytes in some areas were hypertrophic and formed glial tufts. The blood vessels were dilated. Other dogs were given 30 shocks at daily intervals and were allowed to live from 2 to 15 days after the last insult. These showed less diffuse changes, but areas of cellular rarefaction were present. There were signs of marked hypertrophic and hyperplastic activity on the part of the glia.

The author[39] also recently reported a study of the cerebral changes in rabbits exposed to a series of hypoglycemic reactions. In this investigation 26 rabbits were used, 13 of which were subjected to insulin shock at weekly intervals, the number of insults varying from 7 to 19. The rabbits were then allowed to live from 1 to 100 days after the last treatment in order to allow for the appearance of all possible types of chronic or permanent brain damage. In these animals the nerve cell damage was not conspicuous although some shrinking and destruction of cortical cells did occur. In some cortical areas the cells had undergone complete fragmentation, liquefaction and actual disappearance. More prominent, however, were the glial and myelin sheath changes. The glia showed a proliferation producing a subpial, perivascular and diffuse gliosis with the formation of scattered glial nodules. There was also a focal and diffuse demyelinization with actual cavity formation. Hemorrhages were observed in every case and often were so extensive that they tore through the cortex into the subarachnoid space.

Comment.

A comparison of the cerebral alterations in all the groups of cases recorded (insulin shock in diabetics, spontaneous hypoglycemia secondary to neoplasm or functional overactivity of the islands of Langerhans, insulin shock as a therapeutic agent, experimental hypoglycemia in animals) must impress one with their great similarity. These changes consist of nerve cell alterations, cerebral petechiæ, diffuse and focal glial proliferation and areas of demyelinization. The only constant factor in each of these various cases was the low blood sugar, which obviously must be at the basis of the brain damage. It seems inescapable that such hypoglycemic insults have occasionally produced cerebral disasters leading to temporary or even permanent brain damage and functional impairment, and it behooves us at this time to pause and briefly consider the possible mechanisms responsible for the damaging effects of hypoglycemia.

The pathological picture described for hypoglycemia does resemble from an anatomical point of view the results of an anemia upon the cerebral tissue. The effects of a decreased circulation upon the brain have been described by many investigators in both man and animal (Spielmeyer,[40] Ford,[41] De Buck and de Moor,[42] Gomez and Pike,[43] Gildea and Cobb,[44] Kammy,[45] and Döring [46]). In the experimental animal most investigators have studied the nerve cell changes and have described a large range of alterations varying from swelling with pale staining, rounded, partly injured cells to cell liquefaction and coagulation producing small shrunken, fragmented bodies which in many areas entirely disappear. Gildea and Cobb [44] attempted to produce the more chronic alterations with cerebral anemia and were able to show in their animals "areas of devastation" where all cells had disappeared and necrotic brain tissue had resulted. Even more closely do the human cases of cerebral anemia resemble the findings in hypoglycemia. Döring [46] and Kammy [45] described four cases in which the patients died days following a severe cerebral anemia. These investigators observed in the brain, nerve cell damage, vascular congestion, many areas of demyelinization which in some regions, had transformed the brain into a spongy tissue and finally both diffuse and local glial reaction often resulting in many small glial nodules. Because of

this similarity of the pathological changes in hypoglycemia to those on anemia, one wonders whether it is not likely that the damaging effects of the hypoglycemia is also due to a circulatory disturbance which in the latter is of a qualitative rather than a quantitative nature, the blood reaching the brain, because of its deficient glucose, containing insufficient nourishment to support the proper oxidative processes within the brain tissues. Probably the greatest factor in the more severe hypoglycemic accidents is the different individual responses to such a disturbed nutritive process, some individuals reacting much more severely and readily. When one considers these uncertainties of individual response together with the degree and extent of the pathological changes just reported, the implications that these studies might have for the internist in the treatment of diabetics, for the internist and surgeon in the early diagnosis and treatment of adenomas of the pancreas, and especially for the psychiatrist in the use of insulin shock therapy in mental illnesses, become obvious. Each must always keep in mind the possibility of occasional untoward sequels or even of disasters from severe or prolonged hypoglycemic reactions. One certainly assumes great responsibility when using such a mechanism as a therapeutic agent, especially if there are available less dangerous and more controllable procedures. This is of special importance at the present stage of our knowledge since one is unable to predict or adequately control the actual reactions, because the factors behind them are still unknown. At present there is great need for more detailed studies of the unfavorable outcomes so that perhaps in the near future a better understanding may be obtained concerning the basic mechanisms of the beneficial results or the permanent devastations that are seen.

Summary.

1. The literature on those studies describing cerebral damage in hypoglycemia is reviewed.

2. Four sources of material underly this study—namely, insulin shock in diabetics, spontaneous hypoglycemia secondary to neoplasm or functional overactivity of the islands of Langerhans, insulin shock therapy in mental disorders, and experimental hypoglycemia in animals.

3. Four personal cases are reported, three of whom are still alive but handicapped by varying neurological and psychiatric disabilities.

4. From a review of the literature and a study of our own cases it seems very apparent that continued repeated hypoglycemia may definitely produce a depression of the cerebral function and even an irreversible degeneration of the brain tissue and cells, resulting in long continued or permanent functional damage or even death.

5. The factors behind the different individual responses to hypoglycemic insults are at present unknown, hence making it impossible to predict or adequately control the more severe reactions or the resulting brain damage.

BIBLIOGRAPHY.

1. Wilder, R. M., Allan, F. N., Power, M. H., and Robertson, H. E.: Carcinoma of the islands of the pancreas. J. A. M. A., 89: 348-354, 1927.
2. Bowen, B. D., and Beck, G.: Insulin hypoglycemia. Ann. Int. Med., 6: 1412-1425, 1933.
3. Moersch, F. P., and Kernohan, J. W.: Hypoglycemia: Neurologic and neuropathologic studies. Arch. Neurol. and Psychiat., 39: 242-256, 1938.
4. de Morsier, G., and Bersot, H.: Cerebral disorders in provoked hyperinsulinemia. Am. J. Psychiat. (supp.), 94: 143, 1938.
5. Joslin, E. P.: Treatment of diabetes mellitus. Philadelphia, Lea and Febiger, 1928.
6. Heimann-Trosien, A., and Hirsch-Kauffmann, H.: Hypoglykämie mit tödlichem Ausgang. Klin. Wchnschr., 5: 1655-1657, 1926.
7. Campbell, W. R., and Macleod, J. J. R.: Insulin. Medicine, 3: 195-308, 1924.
8. Pemberton, H. S.: Hypoglycemia. Brit. M. J., 1: 1004-1005, 1925.
9. Dahl, B.: Et dodsfold ved insulinforgiftning, med bemerkninger om overdosering av insulin. Norsk mag. f. laegevidensk., 89: 40-55, 1928.
10. Wohlwill, F.: Über Hirnbefunde bei Insulin Ueberdosierung. Klin. Wchnschr., 7: 344-346, 1928.
11. Terplan, K.: Changes in the brain in fatal insulin shock. Arch. Path., 14: 131, 1932.
12. White, P., and Smith, B.: Quoted by Bowen, B. D., and Beck, G. Ann. Int. Med., 6, 1412-1425, 1933.
13. Lindsay, J. W., Rice, E. C., Selinger, M. A., and Mish, K. W.: Protamine insulin as a contributing factor in the death of a diabetic patient with cerebral arteriosclerosis. Ann. Int. Med., 10: 1892-1900, 1937.
14. Bodechtel, G.: Der hypoglykämische Shock und seine Wirkung auf das Zentralnervensystem. Deutsches Arch. f. klin. Med., 175: 188-201, 1933.

15. Baker, A. B.: Cerebral lesions in hypoglycemia. II. Some possibilities of irrevocable damage from insulin shock. Arch. Path., 26: 765-776, 1938.
16. Harris, S.: Hyperinsulinism and dysinsulinism. J. A. M. A., 83: 729-733, 1924.
17. Howland, G., Campbell, W. R., Maltby, E. J., and Robinson, W. L.: Dysinsulinism. J. A. M. A., 93: 674-678, 1929.
18. Best, T. H., Schmidt, E. R., and Sevringhaus, E. L.: Pancreatic tumor with hypoglycemic status epilepticus. Acta chir. Scandinav., 71: 82-102, 1932.
19. Wolf, A., Hare, C. C., and Riggs, H.: Neurological manifestations in two patients with spontaneous hypoglycemia. Bull. Neurol. Inst. New York, 3: 232-251, 1933.
20. Blau, A., Reider, N., and Bender, M. B.: Extrapyramidal syndrome and encephalographic picture of progressive internal hydrocephalus in chronic hypoglycemia. Ann. Int. Med., 10: 910-920, 1936.
21. Anderson, H. B.: A tumor of the adrenal gland with fatal hypoglycemia. Am. J. M. Sc., 180: 71-79, 1930.
22. Smith, M. G., and Seibel, M. S.: Tumors of the islands of langerhans and hypoglycemia. Am. J. Path., 7: 723-739, 1931.
23. McClenahan, W. U., and Norris, G. W.: Adenoma of the islands of langerhans with associated hypoglycemia. Am. J. M. Sc., 177: 93-98, 1929.
24. Terbrüggen, A.: Anatomische Befunde bei spontaner Hypoglykämie infolge multipler Pankreasinseladenome. Beitr. z. path. Anat. u. z. allg. Path., 88: 37-59, 1932.
25. Malamud, N., and Grosh, L. C.: Hyperinsulinism and cerebral changes. Arch. Int. Med., 61: 579-599, 1938.
26. Baker, A. B., and Lufkin, N. H.: Cerebral lesions in hypoglycemia. Arch. Path., 23: 190-201, 1937.
27. Membrez, G., and Razdoljsky, I.: Über die Gefahren der intravenösen und subkutanen Insulineinführung. Zwei Fälle mit tödlichem Ausgang. Zentralbl. f. Chir., 58: 1627-1631, 1931.
28. de Morsier, G., and Mozer, J. J.: Lésions cérébrales mortelles par hypoglycémie au cours d'un traitment insulinque chez un morphinomane. Ann. de. méd., 59: 474-487, 1936.
29. Salm, H.: Benommenheitszustände in Auschluss an die Insulinshockbehandlung von Schizophrenen. München. med. Wchnschr., 84: 1046-1048, 1937.
30. Kobler, F.: Histologischer Gehirnbefund nach Insulinkoma. Arch. f. Psychiat., 107: 688-700, 1938.
31. Freed, H., and Wofford, C. W.: Subarachnoid hemorrhage during shock therapy for schizophrenia. Arch. Neurol. and Psychiat., 39: 813-816, 1938.
32. Schereschewsky, N. A., Mogilnitzky, B. N., and Gorjaewa, A. W.: Zur Pathologie und pathologischen Anatomie der Insulinvergiftung. Endokrinologie, 5: 204-224, 1929.

33. Dünner, L., Ostertag, B., and Thannhauser, S.: Klinik und pathologische Anatomie der chronischen Insulinvergiftung an Tieren. Klin. Wchnschr., 12: 1054, 1933.
34. Stief, A., and Tokay, L.: Weitere experimentelle Untersuchungen über die cerebrale Wirkung des Insulins. Ztschr. f. d. ges. Neurol. u. Psychiat., 139: 434-461, 1932.
35. Tani, N.: Experimentelle Beiträge zum Insulinkrampf, mit Berücksichtigung histopathologischer Befunde. Zentralbl. f. d. ges. Neurol. u. Psychiat., 80: 30, 1936.
36. Schmid, H.: Histopathologic changes associated with insulin shock. Ann. méd.-psychol. (pt. 2), 94: 658, 1936.
37. Weil, A., Liebert, E., and Heilbrunn, G.: Histopathologic changes in the brain in experimental hyperinsulinism. Arch. Neurol. and Psychiat., 39: 467-481, 1938.
38. Accornero, F.: Experimental histopathological researches on insulin shock. Am. J. Psychiat. (supp.), 94: 130-133, 1938.
39. Baker, A. B.: Cerebral lesions in hypoglycemia. III. Experimental investigations. Arch. Path. (in press).
40. Spielmeyer, W.: Vasomotorisch trophische Veränderungen bei zerebraler Arteriosklerose. Monatschr. f. Psychiat. u. Neurol., 68: 605, 1928.
41. Ford, F. R.: An experimental investigation into the effect of asphyxia on the brain. Bull. Johns Hopkins Hosp., 42: 70, 1928.
42. De Buck, D., and de Moor, L.: Lésions des cellules nerveuses sous l'influence de l'anemie aiguë. Le Nevraxe, 2: 2, 1901.
43. Gomez, L., and Pike, F. H.: The histological changes in nerve cells due to total temporary anemia of the central nervous system. J. Exp. Med., 11: 257, 1909.
44. Gildea, E. F., and Cobb, S.: The effects of anemia on the cerebral cortex of the cat. Arch. Neurol. and Psychiat., 23: 876-903, 1930.
45. Kammy, E.: Beitrag zur Histopathologie und Histogenese der Sauerstoffmangelschädigung der Gewebe mit besonderer Berücksichtigung des Gehirns. Beitr. z. path. Anat. u. z. allg. Pathol., 100: 248-261, 1938.
46. Döring, G.: Cerebral changes in delayed death after hanging and ligation of the carotid artery. Virchows Arch. f. path. Anat., 296: 666, 1936.

JEAN-ETIENNE-DOMINIQUE ESQUIROL.

His Work and Importance for Modern Psychiatry.*

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Exactly one hundred years ago, in June, 1838, two important events took place in France that left their trace in psychiatry the world over: (1) The publication of the first *modern* textbook on psychiatry by Esquirol, in two volumes, under the title "Des Maladies Mentales, Considerees sous les Rapports Medical, Hygienique et Medicolegal," that was soon translated into many languages including English. (2) The passage in France of the first in the world law, the so-called "Law of June 30, 1838," designed to protect the rights and interests of the mentally ill. It was Esquirol who began the ground work of framing and drafting of this "Law" that served as a model for several other countries, where similar laws thereafter were adopted.

It is surprising, therefore, that the name of Esquirol is not as well known as it should and deserves to be. For instance, one does not find his name even mentioned in a book, published recently, that aspires to be considered as a text-book on the history of psychiatry.

It seems appropriate indeed that The American Psychiatric Association should note Esquirol's work at this time, as it is only too true, as Bernard states that "we stand upon the intellectual shoulders of those medical giants of by-gone days and, because of the help they afford us, we are able to see a little more clearly what they were able to do." One wonders at times if the last part of Bernard's statement is correct.

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Jean-Etienne-Dominique Esquirol (1772-1840) was born at Toulouse, France. There he graduated from the medical school. Like the great Pinel, he pursued further post-graduate studies in medicine at Montpellier. In the seventh year of the French Revolution, he arrived at Paris in rather straitened financial circumstances. He continued his post-graduate work by attending lectures of the celebrated Corvisart, the future court-physician to Napoleon. While paying a visit to Salpétrière, he met Pinel. This meeting decided his fate. Pinel soon recognized the talents of the young physician and employed him in the preparation of his book—"Medico-Philosophical Tractate" (1802). Daily intercourse with Pinel defines finally the scientific character of Esquirol. In 1805 he finishes his dissertation—not of course without some help from his teacher—entitled "Les Passions Considerees Comme Causes, Symptoms et Moyens Curatifs de L'alienation Mentale."

At this time Esquirol is 33 years of age. His professional standing has already reached a high level. He continues to study and carries on research uninterruptedly. In 1814, he is on the verge of making the greatest discovery of the nineteenth century, namely, that of dementia paralytica. He writes: ". . . . When paralysis complicates a mental disorder, then one after another there become manifest the following symptoms: at first, the articulation of speech becomes more difficult, then there appears a certain clumsiness in movements, later involuntary loss of urine, and so forth. . . . . ." In a later work he adds ". . . . that such a paralysis should not be mistaken for a consequence of cerebral hemorrhage, cancer or tuberculosis." And he goes on ". . . . at first the paralysis is a limited one; later it becomes general, and its appearance, regardless of the delusions of the patient, is an invariable precursor of dementia. The disease lasts no longer than three years; and almost always its later stages are accompanied by convulsions, inflammatory manifestations of the brain, intestinal sluggishness and, ultimately, by gangrene affecting those portions of the skin on which the motionless bodies of the patients rest. . . . ."

Esquirol, however, merely indicated a fact. But his own interpretation of this fact was still far from the actual delineation of a disease entity. Here one does not find as yet even a hint of dementia paralytica *per se,* but only a presentment of it. Still one might

suppose that it was Esquirol who led Bayle to his final discovery of dementia paralytica in 1822.

In 1817 Esquirol had begun the presentation of a course of clinical psychiatry which he continues throughout his lifetime. His clinical demonstrations, lectures and ward-rounds soon acquired international renown. Salpétrière, where he worked at this time, became the European center of new psychiatric ideas. In the same year, 1817, he delivered before the Parisian Academy of Science his remarkable thesis, "Des Hallucinations Chez Les Alienées." The aged Pinel had greeted this work with enthusiasm and stressed the importance of recognition of the difference between hallucinations and illusions.

In 1818 Esquirol made his first extensive trip through several provincial cities, inspecting the institutions where the mentally ill were kept. His impressions were desolate. His report achieved wide publicity. A special committee was created to undertake and carry out a program of improvement of the existing institutions and construction of special new hospitals. Esquirol and his coworkers opened an energetic fight with the old deep-rooted opinions that psychotics are incurable. Due to him, everywhere there were introduced daily medical rounds, records and progress notes.

Soon thereafter Esquirol suggested the organization of colonies for the mentally ill and of a boarding-out system for chronic mental patients. In 1821 he visited the celebrated and unique village of Gheel, Belgium, where for centuries hundreds of mental patients had been boarded out, and where no physician, up to Esquirol's visit, had ever penetrated.

Indeed Esquirol took an amazing interest in everything occurring in the field of psychiatry anywhere. In 1820 a new mental hospital is being opened in Schleswig, Germany, and Esquirol is interested in it. In 1824 a psychiatric department is started in the remote Obuchov's Hospital, St. Petersburg, Russia, and Esquirol writes about it.

From his extensive travels, Esquirol brings back new ideas and new plans. He is even interested in the question, in which we all are still quite interested, namely: is there an increase in the number of the mentally ill? And the basic problems of future psychiatric statistics were taking shape before his eyes.

As an aged man, in the late thirties of the last century, he traveled throughout Italy, inspecting hospitals and attending lectures in medical schools. During one lecture, while he modestly occupied a seat among the listeners, he was recognized and enthusiastically greeted. Before he left Italy, he was invited to inspect the new, just completed, mental hospital in Turin. He found many important defects, and, as a result, the hospital, by order of the King, was turned into barracks, and a new construction was started immediately, in accordance with his plans.

In 1825, Esquirol left Salpétrière and accepted the chair at Charenton. There he began the ground work of framing the original legal act, designed to protect the rights and interests of the mentally ill. The "Law of June 30" (1838) established the important principle of medical testimony; without it no one should be deprived of his liberty. Analogous laws were adopted in England in 1845, and in Scotland in 1857. The "Law of 1838," which was drafted with the active participation of one of Esquirol's pupils, Ferrier, was a most important achievement of French psychiatry.

Such was Esquirol: an energetic reformer of the psychiatric hospital system, a creator of social-legal psychiatry, a founder of psychiatric statistics, but no less great as a clinician.

He was the first to introduce into psychiatry the terms "remissions" and "intermissions"; he, too, was the first to point out the importance of the patient's weight in diagnosis and prognosis. He lived in intimate contact with his patients, and his artistic descriptions of individual cases, scattered throughout his text-book, have lost none of their vividness. His text-book on "Mental Diseases," published as mentioned in 1838, is the result of four decades of psychiatric activity, and must be regarded as the foundation of the entire scientific psychiatry of the following periods.

In the general chapter "De La Folie," Esquirol states that we must take into consideration not only the mental but also the somatic symptoms of mental diseases. Here is thus expressed the basic principle of psychiatry as an empiric science and is outlined a whole program for future observations. Indeed, French psychiatry was not slow in applying these recommendations directly to the description of dementia paralytica. Throughout the entire text-book a multitude of very important theoretical and practical points are scattered. Of particular interest are the first hints as to depres-

sions in epilepsy, the indication that those patients while suffering from occasional mental (spiritual) pain are driven "to drink wine and other liquors"; attention is also called to their characteristic irritability which leads them "into rages and conflicts." The chapter on "Suicide" is still of great interest, as are many other chapters.

His study on "Monomanias" is probably his most original one. According to him, in monomania the intellectual disorder is confined to a single object, or a limited number of objects. The patients seize upon a false principle, which they pursue without deviating from logical reasonings, and from which they deduce legitimate consequences which modify their affections, and the acts of their will. He further differentiates intellectual monomania, affective monomania and instinctive monomania.

As a vivid illustration of monomania, he mentions the morbid craving for alcoholic beverages. He does not use the term "dipsomania," but describes all its symptoms: the change of the person's character, the periodicity of the attacks, the return to normalcy after the attack, etc.

He says: "There is sometimes also a disordered condition of the system, which leads certain individuals to the abuse of fermented drinks. There are cases in which drunkenness is the effect of the accidental disturbance of the physical and moral sensibility; which no longer leaves to man liberty of action. The sick, who are thus affected, had formerly gentle manners, and habits of sobriety, but have suddenly changed. Some physical or moral cause has provoked this change; certain precursory signs have announced it. When the attack has terminated, the patients return to their former temperate habits. Relapses are frequent; are provoked by the same causes, and announced by the same phenomena. They sometimes recur at fixed periods."

The chapter on "Homicidal Monomania" contains many points of interest, but time does not permit us to discuss them.

Of all the numerous practical attainments of Esquirol's psychiatric clinic, one must point out the artificial tube feeding. The invention of the stomach tube was ascribed to English psychiatry. Pinel, in his work, tells that for five months he fed a female patient by the tube method, not mentioning the source of it. Probably the initiative was Esquirol's, who stated very definitely: "I was the first to use the stomach tube in such conditions; usually this oper-

ation is free of danger and has saved the lives of many of my patients."

In the care for psychotics, Esquirol persistently recommended fresh air, calisthenics, physical work. By his initiative in 1832, there was organized, in the suburbs of Paris, the first colony for the mentally ill, the celebrated "Farm of St. Anne," which served later as the model for similar undertakings.

Around Esquirol was organized the first psychiatric school of physicians. At his rounds at Salpétrière and Charenton he was always surrounded by a number of pupils; several of them later became the leaders of psychiatry all over Europe.

One would like to raise the question: How much have we progressed in psychiatry since Esquirol's time? Should we now, in 1938, repeat the words of William Ellery Channing, uttered in November 1837: "We have indeed little cause of boasting. The great features of society are still hard and selfish. The work of a human being is a mystery still hid from an immense majority, and the most enlightened among us have not looked beneath the surface of this great truth. Still, there is at this moment an interest in human nature, a sympathy with human suffering, a sensibility to the abuses and evils which deform society, a faith in man's capacity for progress, a desire to carry to every human being the means of rising to a better condition and a higher virtue. Amidst the mercenariness which would degrade men into tools, and the ambition which would tread them down in its march towards power, there is still a respect for man as man, a recognition of his rights, a thirst for his elevation. . . . . We see in the common consciousness of society, in the general feelings of individuals, traces of a more generous recognition of what man owes to man."

Only recently someone has said that we all can "find help in reaching out to closer constant contact with the minds of those great physicians, whose bodies are dust long since, but whose thoughts live on and are powerful still."

In honoring today the memory of Esquirol, and recalling his great work in the interest of the mentally ill, we dedicate ourselves anew to the task of improving the conditions of our patients in every way possible. The memory of Esquirol, whose "body has been dust" for almost a century, will live on forever.

BIBLIOGRAPHY.

Esquirol: "Mental Maladies," Treatise on insanity. (Translated from the French by E. K. Hunt, M. D.) 1845.

Kannabich, Amdur, and Messinger, E.: Text-book on history of psychiatry. (In preparation.)

Deutsch, Albert: "Social factors in psychiatric progress." Mental Hygiene, *22*: 265, April, 1938.

DISCUSSION.

DR. OSKAR DIETHELM (New York, N. Y.).—I would like to underscore what Dr. Amdur stressed, that the great work of Esquirol was the reorganization of the French hospital system. To some extent it is a parallel of what we have today when we think of Dr. Hamilton's work in connection with this Association.

Psychiatric thinking has advanced more than the hospital. In a few places at that time, reform had taken place but there is a striking contrast when one reads what Pinel thought he had done in Paris, and look at what actually was going on in the state or county hospitals all over France. That is probably why this exceedingly energetic and forceful personality with a great mind and great organizing ability was able to do something which no other man at that time was able to do.

The work was, however, vigorously attacked, especially by scientific workers, and by an outstanding German psychiatrist of that time. His textbook was translated, but at the same time it was not as far reaching as it might seem. In his book, which is still one of the most interesting contributions of today, are no doubt statistics which compare well with modern state hospital statistics, and are therefore to be used as a comparison.

Jean Etienne Esquirol's work and publications have always been referred to in modern statistics of continental countries and also in this country. As a clinician he probably was less successful. He was a great clinician, but he got on the wrong track. He probably was the forerunner in forming the concept paranoia, but he got the French psychiatry on that angle into a corner from which it took a long time to emerge.

Even now we still have monomania playing a rôle in France, while a more dynamic viewpoint has been achieved for more than 50 years in other countries.

The textbook is really not a textbook. It is merely a compilation of all his articles which had appeared previously and now brought together in two volumes. It would therefore not be right to call this work the first textbook of modern psychiatry.

That credit should go to Gerucci 50 years before this time, who has never received enough consideration. Also, Pinel's book was really to a large extent a repetition of what the great Italian had produced before, but he was an exceedingly forceful organizer, with an exceedingly forceful personality, especially in the field of organization of mental hospitals and the legal aspects of psychiatry.

PSYCHIATRIC DISORDERS IN FIFTY SCHOOL TEACHERS.*

By JAMES H. WALL, M.D., White Plains, N. Y.

In an approach to the problem of psychiatric disorders in teachers, the writer has selected for study a group of 50 women teachers who were patients in the New York Hospital, Westchester Division, between 1920 and 1935, a period of 15 years. They were in the fifth and sixth decades of life; 47 was the average age at the time of admission. This age group was chosen because it was from that life period that the majority of this professional class came. They were from different localities and from both public and private schools. The study was also made to correct a false impression on the part of psychiatrists and others, that the prognosis for such a group was not good. Our experience has shown that half of these patients fully recovered and were able to resume their work with efficiency. It should be noted that statistical studies have shown that the rate of first admissions among teachers to the New York state hospitals was lower than the average for other occupations in the major psychotic groups, and the admission rate for this professional group at our hospital has been relatively low. Although there is no psychiatric disorder which is characteristic of any professional group in the community, this study of 50 women teachers does reveal a few interesting features which make up a composite picture of their problems. The family history, personality development and the illness of the patients are reviewed, illustrative case material is presented, treatment and outcome are discussed, together with some preventive measures which suggest themselves.

There were 29 patients with a positive family history for mental disease. The mentally sick relatives were parents or siblings. In all instances our patients were at some time intimately

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associated with these sick relatives, a definite source of strain and worry in the majority of cases. There was a type of father who played a dynamic rôle in the life of 24 of the patients; he was the stern, rigid, dictatorial individual. As children the patients were intimidated and frightened by him, and stood in awe of paternal authority. There was also an unconscious attachment, as revealed by later trends, but the obvious effect on their development was one of fear and inhibition. The later prudishness and intolerance of their personalities seemed to be associated with this relationship. There were 37 over-solicitous mothers who were inclined to keep their daughters, the patients, under their domination.

The birth and early development of these patients were normal. They came from large families. There were as many as 10 children in some of them; the average size of family for the group, including the patient, was five. In 20 cases the patient was the eldest sibling and in 10 others the second born. They held a position of respect in the home and were looked up to by the other children. They were serious-minded and inclined to take their responsibilities seriously. The families were closely knit in 46 instances. The early physical health of the patients was good; menses began at the average age of 13. The psychosexual development showed autoeroticism as the chief sexual outlet. Only 12 of them were seriously involved with members of the same sex and in all these cases the conflict over this instinctive problem contributed to the development of the illness. Only six were married and all but one, who married late in life, adjusted well to marriage. Among those who did not marry, there was a noticeable lack of interest during their teens and twenties in members of the opposite sex. They were considered prudish. On the other hand, there was an economic factor of great importance: the families of 37 of the single women were dependent, more or less, on the patients' earnings, a situation partly responsible for the choice of, and continuation in, the teaching profession.

All of the patients were college or normal school graduates. The average age at which teaching was begun was 21 and all were active in the profession when the illness began. By their relatives and associates they were described as having been tense, energetic and lacking in the ability to relax. As a group their range of interests was narrow and absorbing hobbies were rarely encountered. Only

10 had any interest in the domestic arts. There was a tendency to be hypochondriacal in 30 instances and these individuals shopped from one physician to another and oftentimes patronized unconventional healing cults.

One-half of the patients changed their faith or philosophy of life from time to time, and showed a leaning toward occult and mystical interests. Periods of working under tension on too many school projects, with irregular hours of sleep and rest, were frequently reported. Only one of the patients indulged in alcohol to excess.

The average duration of illness prior to admission was seven months. The climacterium was a factor in 34 of the patients, with the associated symptoms of irregular menses, hot flashes and other unpleasant physical sensations. Of greater importance were the psychological factors involved—the dread of retiring, fear of various physical incapacities, and increasing emphasis on economic security. The increased sex tension with a recurrence of autoerotic activity or worry over earlier masturbation were important factors. An accentuation of these psychological difficulties plus precipitating factors of death and illness in the family or circle of acquaintances constituted the etiology in the 34 patients who developed agitated depressions. There were 14 patients with dementia præcox and the development of the illness was insidious, though pronounced symptoms were manifest only during the year prior to admission. A careful study of this latter group revealed an increasing sensitiveness, suspicion, and withdrawal over a period of many years. Two patients had manic attacks and there was in both a history of mood swings and previous psychotic episodes.

Thirty-seven of the patients came voluntarily to the hospital and 13 were committed patients. At the time of admission, the physical condition was generally good. No serious physical pathology was found.

Before discussing the mental trends the following representative and illustrative case histories of four patients are presented:

CASE I.—The patient, a single woman of 45, was admitted in a tense, anxious, depressed state, after an unsuccessful attempt at suicide. Her family history showed a stern father and a solicitous mother. One brother had a depression. She was the oldest of five children; her birth and early development were normal. She was a bright child, did well in school, and throughout her life she was outgoing, social and had many friends. She matured at 13,

the menses were regular and without complication. Masturbation was without apparent conflict up to the time of her illness. Toward her siblings she took a motherly attitude and was fond of playing school in the rôle of teacher. Her plan for a career was definite and upon finishing college with honor she entered the profession in which she achieved success as a teacher of English. She had many interests and diversions. She had friends of the opposite sex but never cared to marry and was apparently honest in saying that she had no regrets or misgivings concerning this decision.

Two years prior to admission, her menses became irregular and after a few months ceased. She had no hot flashes but she did tire easily. With increasing sex tension, the masturbation became more frequent, with guilt and conflict. About this time her younger brother became depressed but recovered after a short illness. This illness together with the death of a close friend caused worry and insomnia. During the month prior to admission, she became fatigued easily and her agitation and restlessness were noticeable. On the day of admission she attempted suicide by trying to jump from a window. On admission she was in good physical condition but she was depressed, agitated, indecisive, and expressed a wish to die. She believed that she had brought disgrace on her family. Within a few weeks her agitation subsided and she was placed on a full schedule of activities. She was frank in the discussion of her problems; the illness of her brother and the death of her friend had accentuated a feeling of insecurity. The worry over masturbation and the climacteric adjustments had added their toll to the lowering of her resistance. After six months in the hospital she fully recovered and resumed her work. She has been well for nine years and during that time she has been promoted.

Case II.—A single woman of 50 came to the hospital in an agitated, depressed and hypochondriacal state. The family history showed a strict, serious father, who was a minister, and a quiet reserved mother. The patient was the second of five children. Her birth and early development were normal. She was a robust child and she did well in school. She matured at 13 and her periods were without complication. She was devoted to her parents and had many friends of her own sex. Upon finishing college she entered the teaching profession in which she was successful. Outside of her work she was considered impractical with no domestic interests and with a tendency to be indifferent in the care of her person and clothes. She had no hobbies or recreational outlets. She worked under tension and never took sufficient time for meals. Her father and mother lived with her and were dependent upon her. When she was 25 she lost her mother but continued to support her father and some of her siblings. From then on she was troubled by gastric discomfort and gaseous eructations following her meals which were invariably eaten hurriedly. She consulted various physicians and mysterious healers.

Three years prior to admission her father died. This loss was keenly felt and she became interested in religious work in which she felt she was emulating her father. One year later she passed through the menopause and

for the next two years she overworked by engaging in an excessive amount of exhausting religious work. She lived alone and took insufficient nourishment and gradually became reduced physically, with increasing restlessness and insomnia. One month prior to admission, after completing a successful series of lectures, she developed the idea that she had failed and immediately became agitated and depressed. On admission she was undernourished but no other physical pathology was found. She was extremely self-depreciatory, thought her body had changed and that her bowels did not function, so that she ate only with urging. She spoke of unpleasant thoughts concerning her father; she believed that she was hurting him with her thoughts, mentioned dreams about him, but would not discuss them except to say that he was being tortured by her evil thoughts. She believed that she heard the mournings of her parents and thought that there were instruments of torture in the hospital for her punishment. On a régime of rest, proper diet and a gradually increased schedule of hospital activities she improved. She learned many hobbies in occupational therapy and discovered the value of play for the first time in her life. With her physician she was frank and unburdened a resentment toward her family who had expected too much of her. After two years of hospitalization she resumed her work and for 12 years she has done well. In a recent letter to the hospital she stated that she had seemed to function and to feel better since her illness.

Case III.—A single woman of 44 became depressed and fearful and was brought to the hospital following a suicidal attempt. In the family history there was an aggressive and demanding father and a mother who had a depression at the menopause. The patient was the oldest of six children. She was a healthy child who was called a tomboy as she grew older. She matured at 13, her periods were regular and without complication. Masturbation was a life problem of intense conflict. She did well in school and became a teacher of outstanding ability. She enjoyed her friends but insisted on doing most of the talking and planning. She had a strong temper and was frequently ashamed of her outbursts and scenes. She was not fastidious about her person. At 30 she became a Christian Scientist in order to have purer thoughts. She became narrower and more intolerant, joining a group who considered Jews and Catholics a menace. She had no absorbing hobbies or relaxing recreations. Her friends noticed that she spent much time and energy in avoiding what she termed unpleasant things in life, and she commented shortly before her illness that she wished she could escape the menopause as she had the other "worldly curses of life."

The beginning of her illness was associated with the cessation of her menses. She lost weight, complained of fatigue and insomnia, and feared she would have the same illness as her mother. Over a period of six months the depression and agitation increased and on the day of her admission she attempted to jump from the roof of a building. On admission she was undernourished and she spoke loudly of her troubles while wringing her hands. She complained of tensions in her neck and abdomen and of severe constipation. She spoke most frequently of hell fire and expected to be burned and

tortured; she called herself an arch devil in a fiery furnace and believed all those around her to be in heaven. She thought that prolonged baths would save her from burning and gradually developed the delusion that she was made of metal. She attempted suicide on many occasions and identified herself with Christ in conception, birth and crucifixion. For nine months she continued noisy and agitated. During the next three months she began to project her ideas and to be quieter, expressing the belief that the treatment had changed her. She developed an elaborate delusional trend with paranoid ideas about hospitals and physicians. She was critical of her relatives and at the end of a year she left the hospital to live in the country with friends. She has never resumed her work and after 10 years has become more withdrawn and seclusive.

CASE IV.—The fourth patient, a single woman of 51, was admitted in a highly delusional state, believing that she must die for her sins and those of the world. Her father was an impractical visionary school teacher who had died 16 years before of anemia. The mother, a mild mannered woman, kept her children under her control. The patient was the oldest of seven children. As a child she had temper tantrums and night terrors. She was very much upset by her first menstrual period at 12; she thought it disgusting. After a short period of masturbation in adolescence, she became prudish, intolerant, and decided early in life never to marry in order to shun sex completely. She was a hard worker in school and became an outstanding teacher. Her range of interests was narrow and she worked under tension. She took no interest in her personal appearance. She had no male admirers and at 30 when another women attracted her very much, she immediately dropped this friend. She was inclined to be envious and critical of her younger siblings. She was fond of her father and blamed her mother for his death. She was born into a Presbyterian family but changed her faith many times and in her illness she became deeply interested in the Catholic religion. Her family considered her erratic, unstable and impractically religious.

There was a marked change in her personality one year before admission. She was passing through the menopause, sex tension increased, she was troubled by sex obsessions, with an annoying curiosity about the genitals of male relatives. She felt flooded by instinctual urges and indulged in orgies of autoerotic activity, while at the same time she became more interested in religion as a means of controlling herself. Six months prior to admission she became disturbed over the death of an associate and she developed the idea that her mother and sister should die. In an ecstatic state she cut down a tree which was near a tree her sister had planted, and soon afterward mutilated one of her fingers, necessitating its amputation. She was in a general hospital for a few days.

On admission here she was in good physical condition. She was restless and suicidal, almost succeeding in an attempt at hanging herself. She was aggressive and critical, expressing frequently an admiration for "purity of thought and high-mindedness." She said that she had bitten her finger for its offense of masturbation. She thought her family and the hospital staff

were persecuting her and that her death would save the world. She continued delusional and after a year was transferred to another hospital. On the day after arrival there she deliberately slammed a door on her finger and calmly spoke of the good effect of self-inflicted pain. She believed she was sent by the Almighty to help the other patients, in whose treatment she often interfered. For the past five years she has been in the other hospital and a recent letter describes her condition as unchanged.

These patients' histories show the influence of personality equipment and of conflicts on the form of the mental reactions and the outcome. The common factor in the trends of the 50 patients, as illustrated by four histories, was the feeling that they were being flooded by instinctual urges which they were unable to accept or manage. This was apparent to some degree in all of them. Associated with this state were agitation, anxious depression and hypochondriacal notions in the 34 who had agitated depressions. These symptoms were also present in the 14 with dementia præcox, although the splitting, projection and scattering became more manifest as the disorder progressed.

In the two patients with manic reactions a similar conflict was seen, although expressed in uninhibited behavior and productions for a time. The conflict was dramatically portrayed by a fear of burning and consuming fire in the trends of 20 of the patients. The instinctive forces appeared to be rebelling against a life of little or no gratification. Suicidal trends and attempts were present at some time in most of the group. It was noticed that a fear of hurting others was present in 15 of the patients. None showed disturbances of sensorium and the intellectual level was high.

TREATMENT.

During the first period of hospitalization attention was directed toward securing rest, sleep and a proper state of nutrition. Prolonged baths, packs, other hydrotherapy and massage were used to combat the restlessness and agitation. Although starting on a limited schedule, the majority of these patients were soon participating in a full program of occupational therapy, physical education and social activities. Psychotherapeutic interviews were frequent, and the intelligence and psychological interest of the group contributed a great deal to the careful study and modification of their personality difficulties. The acquiring of a more tolerant attitude toward instinctual drives and problems was the most difficult but

most helpful to achieve. Those who recovered were able to understand that they had turned aside or avoided inner needs without facing and making some sort of peace with them. In some instances the recovered patients spoke of their illnesses as an explosive release from a smoldering conflict of years' duration; as something which cleared the atmosphere. Some of these patients remarked years after discharge that after the illness they had seemed to function better than before.

Many of them learned to play for the first time in their lives, having received instruction at the hospital in many indoor and outdoor sports. Lasting hobbies were acquired in occupational therapy. Some of the patients placed great value on the benefit derived from close daily contact with members of their own age in the social and recreational aspects of their treatment. A better organization of their time for work, rest and sleep was stressed by some.

Of the 50 patients, 25 recovered and were able to resume teaching. The other 25 did not return to work and most of them have been in mental hospitals. The average length of hospital residence of those who recovered was 12 months. The average length of hospital residence for those who did not recover was 10 months, an average of 11 months for the entire group. The 14 patients with dementia præcox did not resume teaching. The two patients with manic reactions had subsequent attacks. The nine patients who did not recover from their agitated depressions were individuals with narrow rigid personalities, and their trends and behavior displayed a prolonged drawn-out battle in which instinctual gratifications, though in regressed expression and form, were a little stronger than the personality forces for normal adjustments.

Some indications in the way of prevention were suggested by the study. The family relationships might have been managed differently. The narrowed interests and the failures to adopt and cultivate wholesome hobbies and recreations were remediable, particularly for those who thought such adjustments could wait until the time to retire. The shifting philosophies and faiths, and the shopping from one healing cult to another could have been modified. The earlier achieving of some solution and harmony out of the energy-consuming conflict between unfaced inner demands and too rigid ideals was possible. The importance of sufficient rest, relaxation and sleep, has been stressed by some teachers who were

interviewed after passing through the crises of their lives, including retirement, without a breakdown. It is adjustments such as these which psychiatrists are equipped to deal with, and one can think of no group more worthy to receive such help than this professional group who play such an important rôle in the community.

BIBLIOGRAPHY.

Nolan, William J.: Occupation and dementia præcox. The State Hospital Quarterly, February, 1918.

Nolan, William J.: Occupation and manic-depressive psychosis. The State Hospital Quarterly, November, 1918.

Mason, Frances V.: A study of seven hundred maladjusted school teachers. Mental Hygiene, July, 1931.

LIFE SITUATION TESTS AS AIDS IN PSYCHIATRIC PROGNOSIS.*

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This report is presented not as a finished study but as an indication of possibilities contained in a method of studying patients by standardized procedure designed to give some indication of the presence or absence of functions that involve the total personality.

The term "life situation" is a misnomer. Every situation in which men act is a "life situation." What is really meant is *spontaneous* life situation, as opposed to *artificial test* situation. No attempt will be made to define the word "spontaneity." Suffice it to say that the phrase "life situation" is here used in the sense of a spontaneously emerging setting in contrast with the "test situation" which is artificially arranged.

There are degrees in the spontaneity of reactions. Persons become less spontaneous the more they think they are being watched. The more a situation has the mark of strangeness and unfamiliarity the less the spontaneity. On the ward, a patient who has retained some measure of contact with his surroundings is likely to display spontaneity with nurses and attendants but hardly with the physician, who is essentially a stranger in the situation. He is official, and does not share the patient's daily routine. With nurses and attendants, on the other hand, the patient is immersed in a common, familiar medium. When, in 1932, we decided to devise a set of life situation tests, it was clear that the tests must be applied

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by nurses, not by physicians. About a year was spent on a series of trials, and by the end of 1933 a battery of tests had been elaborated which has since been in use. The battery consists of six verbal and six pragmatic tests. This report is limited to a description of three verbal tests which, thus far, have proved most informative and have shown correlations with subsequent adjustment of the patients.

Description of Tests.

1. *Insinuations.*—In the forenoon of the day after admission a nurse asks the patient casually: "Jane, did you hear about Marshall Field?" This question is intended to arouse curiosity and to elicit such replies as "What about them?" "What happened to them?" "Did they have a sale?" or "Did they burn down?" If any of these answers are returned the test is ended and the score is 100. If the patient, however, merely answers "yes," "no," or "I don't know," "I didn't hear anything," the nurse repeats the question with such modification in wording and intonation that there is implied that some important event has happened. This is the second step of the test and runs: "Jane, didn't you hear about Marshall Field?" If now the patient replies: "What about them?" or "What happened to them?" the test is ended at the second step, and the score is 66.7. If the answer again was "yes," "no" or "I don't know," still more emphasis is given in wording and intonation: "Why, Jane, *didn't* you hear about Marshall Field?" If a correct answer is given the score is 33.3. If no such answer is forthcoming the score is zero.

False Time Statements.—In the afternoon of the same day the nurse says to the patient: "You certainly look much better than you did when you came here two weeks ago." If the patient replies "I came yesterday," the test is completed at the first step and the score is 100. If not, the nurse says: "Do you think you gained weight since you came here two weeks ago?" If necessary, the third step is taken: "Do you like it better now than you did when you came here two weeks ago?" Scores of 66.7 and 33.3 are given for correct replies to the second and third steps respectively.

Absurd Time Statement.—This test is given on the third day. The steps are: (1) "Will you go home yesterday?" (2) "I thought

you will go home yesterday." (3) "How is it you will not go home yesterday?" The manner of scoring is the same as in the preceding tests.

During the fourth and fifth day after admission, the three tests are given again in exactly the same wording and sequence of steps, except that substitutions are entered in the last part of the sentence spoken by the nurse. In the "insinuations" the name of the ward physician is substituted for Marshall Field; in the "false time statements," the nurse mentions "two months" instead of "two weeks," and in the absurd time statements, the question, "Will you go home yesterday" is changed to "Did you go home tomorrow?" The first three tests, given during the first two days, comprise what we call the first quarter, the second three tests, the second quarter. In the third and fourth quarters, the substitutions are of a similar nature. The "insinuation" of the third quarter mentions the name of the closest relative, that of the fourth quarter inquires about President Roosevelt. The "false time statements" of the third and fourth quarters substitute "two years" and "two days." In the corresponding quarters of the "absurd time statements," the questions are: "Will you go home last week?" and "Did you go home next week?" Tables 1, 2, 3 and 4 illustrate the nurse's questions, the patient's answers and the scores obtained in the four quarters of the three tests.

Final Score.—The final score is calculated from the aggregate performances in the three tests. In the right lower quadrant (D) of Chart I a graphic analysis is given of tests of 145 patients. The "insinuation" scores are plotted under I, the "false time statement" under II, the "absurd time statements" under III. The chart also plots the scores in two other tests (IV and V) which will not be considered here. The solid black line represents the scores of the first quarter, the double broken line those of the second, the single broken line those of the third, and the dotted line those of the fourth quarters. The chart demonstrates a close similarity of performance in all four quarters. The scatter is 9.4 in I (insinuations), 8.1 in II (false time statements) and 3.0 in III (absurd time statements). It is evident from the chart that the first and second quarters occupy a median position. Hence it seemed legitimate to consider them representative of the final

TABLE 1.

I. INSINUATIONS.

First Step: Jane, did you hear about Marshall Field?
Second Step: Jane, didn't you hear about Marshall Field?
Third Step: Why, Jane, didn't you hear about Marshall Field?
Second, third and fourth quarters: *Ward Physician, Relative, President Roosevelt.*

II. FALSE TIME STATEMENTS.

1. You certainly look much better than you did when you came here *two weeks ago.*
2. Do you think you gained weight ever since you came here *two weeks ago?*
3. Do you like it better now than you did when you came here *two weeks ago?*

Second, third and fourth quarters: *Two Months, Two Years, Two Days.*

III. ABSURD TIME STATEMENTS.

1. *Will* you go home yesterday?
2. I thought you *will* go home *yesterday.*
3. How is it you *will* not go home *yesterday?*

Second, third and fourth quarters: *Did—Tomorrow, Will—Last Week, Did—Next Week.*

TABLE 2.

I. INSINUATIONS.

Date.	Nurse's insinuation.	Step responded to.	Patient's response.	Score.
12–10–34	Marshall Field.	3d	(1) No. (2) No. (3) No, I didn't. What about them?	33.3
12–12–34	Miss K. (nurse).	3d	(1) No. (2) No, I didn't. (3) No one told me. What about her?	33.3
12–14–34	Mother.	None of three.	(1) No, I didn't. (2) No, I didn't. (3) I didn't hear.	0
12–17–34	President Roosevelt.	3d	(1) No. (2) No, I didn't. (3) What about him?	33.3

TABLE 3.

II. False Time Statements.

Date.	Time mentioned by nurse.	Step corrected.	Patient's response.	Subsequent Inquiry.	Score.
12-10-34	Two weeks.	None of three.	(1) Yes. (2) Well, I lost. (3) Yes.	I came today.	0
12-12-34	Two months.	3d *Comment.*	(1) Yes. (2) I don't know. (3) You know I've only been here two days.	.....	33.3
12-14-34	Two years.	1st	(1) I just came last Monday.	.....	100.0
12-17-34	Two days.	3d	(1) Thank you, I feel fine. (2) I don't think I gained. (3) I am here longer than two days.	Over a week.	33.3

TABLE 4.

III. Absurd Time Statements.

Date.	Time absurdity mentioned by nurse.	Step corrected.	Patient's response.	Score.
12-11-34	Did—tomorrow?	3d	(1) No, not before Friday. (2) The doctor said Friday. (3) You mean: will I go home tomorrow?	33.3
12-13-34	Will—yesterday?	2d	(1) Dr. B. said I'll be here a month. (2) You mean: did I think I am staying?	66.7
12-15-34	Will—last week?	1st	(1) What do you mean? Did I go home last week?	100.0
12-18-34	Did—next week?	None of three.	(1) Dr. B. said I'll have to stay here awhile. (2) I didn't. I was home for Christmas. (3) No, I didn't.	0

score. This procedure cut in half the number of partial scores to be calculated and made for simplification.

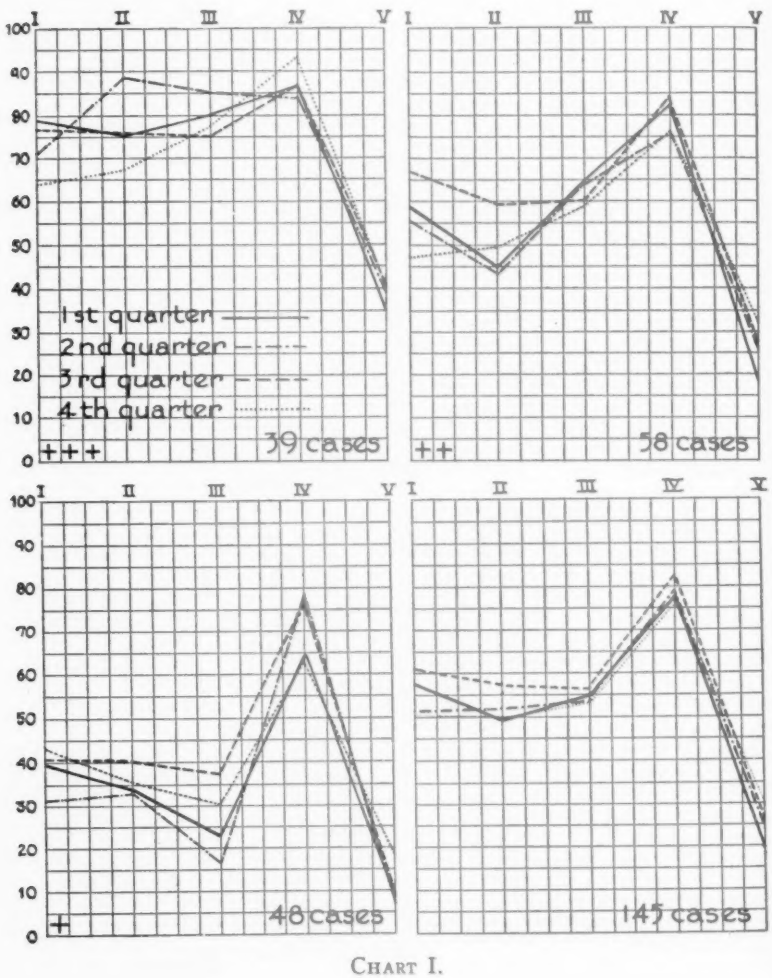


CHART I.

In the quadrants A, B and C of Chart I the scores in the 145 cases were subdivided into three separate subgroups. In the quadrant A, 39 patients are represented; their combined scores rise about 50 per cent in all three tests; the sign + + + at the bottom of the quadrant symbolizes the three higher than 50 per cent

partial scores. In quadrant B, 58 cases are plotted in which the combined scores attained more than 50 per cent in two tests only (++). This is true only if the first and second quarters alone are considered. Quadrant C presents 48 cases in which the combined scores rose to or above 50 per cent in one test only (+). This relationship is not plainly visible in the tracing, which records a lower than 50 per cent score for all three tests. The explanation for this discrepancy is: Of the 48 patients, 20 scored well in the "insinuations," and poorly in the other two tests; 20 other patients had good scores in the "false time statements;" only eight in the "absurd time statements" only. Each group, then, had poor scores in two tests. These poor scores tended to suppress the final score of the total group in all three tests.

Two subgroups were tabulated but not charted. The one consisted of patients who scored less than 50 per cent in all three scores; the other of patients who in the course of testing, usually during the first and second quarters, gave evidence of having discovered that they were being tested. When the nurse addressed them they replied: "Oh, you just want to test my memory," or "You have orders from the physicians to ask these questions," or "You want to find out whether I am insane." Once such a discovery was made the setting of the "life situation" was no longer considered existent and the tests were discontinued.

Material of the Study.

Since September 1933, each patient admitted to the Psychiatric Institute was given the three tests in the manner described. Selection was thus precluded. The present report comprises a total of 233 cases, 108 of which were classified as dementia præcox, 54 as manic-depressive conditions, 5 as involutional psychoses, 14 as psychoneuroses. A miscellaneous group of 52 cases, variously classified as psychoses with somatic disease, psychoses with mental deficiency, psychoses due to other metabolic disorders or primary behavior disturbances, were grouped together under the heading "other psychoses." Twenty-four persons with psychoses associated with gross lesions of the brain and nearly 150 patients treated with insulin and metrazol shock were tested but not included in this series.

Results.

Age and Sex Distribution.—This is shown in Table 5. There was a preponderance of female patients among those in the manic-depressive group and in the "other psychoses." In the total series the percentage of male patients was 43.8 and of female patients, 56.2. The average age was 30.5 years for the total group. Except for the patients with involutional psychoses, the subgroups showed a fairly even age distribution, ranging from 27.2 years in the cases of dementia præcox to 34.9 years in the "other psychoses."

TABLE 5.

AGE AND SEX DISTRIBUTION OF TWO HUNDRED AND THIRTY-THREE UNSELECTED PATIENTS.

	No. of cases.	Average age on admission, in years.	Sex.			
			Male.	Per cent.	Female.	Per cent.
Dementia præcox	108	27.2	53	49.1	55	50.9
Manic-depressive psychosis	54	31.9	20	37.1	34	62.9
"Other" psychoses	52	34.9	18	34.6	34	65.4
Involutional depressions	5	51.2	2	40.0	3	60.0
Psycho-neuroses	14	27.3	9	64.3	5	35.7
Total	233	30.5	102	43.8	131	56.2

Follow-up Investigations.—In February, 1938, *i. e.*, about four and one-half years after the first systematic application of the tests, a follow-up investigation was made of all patients who had been given the tests. A questionnaire was sent concerning patients who were known or supposed to live in the community, and the remainder were studied from the records of the state hospitals to which the patients had been sent. The questionnaire, addressed to the parents or nearest relative, requested an answer to two questions: "Your.....(relative) was discharged from the Psychiatric Institute on..... (1) How many months has he (she) since spent at home? (2) How many months has he (she) since spent in hospitals for mental disease?" Of 248 patients investigated, information was obtained concerning 233. Of these, 18 had died, 106 were in the community, either with or without supervision, and 109 were in hospitals. Of the 106 patients living in the community, 49 appeared for a personal interview.

Subsequent Adjustment, Clinical Classification and Test Score.—Table 6 analyzes the relationship of subsequent adjustment to original test score in the group of cases classified as dementia præcox. Of the 108 patients, 14 made a "discovery," 22 scored better than 50 per cent in all three tests, (+++) 30 in two tests (++) 32 in one test only; (+) 10 scored less than 50 per cent in all three tests (---). The table indicates that at the time when the tests were given the score would have furnished a basis for prediction as to the prospect of the patient returning to the community and maintaining life there, either with supervision or independently. The patients who made a "discovery" had a 35.7 per cent chance of unsupervised, and a 21.4 per cent chance of supervised life in the community subsequent to discharge. The prospects for unsupervised communal activity diminishes progressively in direct proportion to the lowering of the score. The prospect of community life, without regard to the need for supervision, shows a progressive decline in the first three test groups (discoveries; +++; ++), but then maintains a steady level of about 20 per cent. The length of residence in the hospital increases in uninterrupted progression from 35.7 per cent among patients who made discoveries to 80 per cent among those who scored poorly in all three tests. It is of interest that, as the totals at the bottom of Table 6 indicate, only 15.7 per cent of schizophrenic patients could be rated as fully recovered when studied from about one year to four and one-half years after admission.

Of the 32 patients who are listed as residing continuously in the community, 5 were readmitted for one brief intermittent stay in the hospital. Of the 70 patients listed as "continuously in the hospital," 8 had brief intermittent residences in the community.

Table 7 analyzes the correlation of subsequent adjustment and test scores of 52 patients, grouped together under the heading of "other psychoses." Of this group, 7 cases were carried in the record as "unclassified," 18 were classified as "psychoses with somatic disease," 9 as "psychoses due to other metabolic diseases," 7 as "psychoses with mental deficiency," 5 as "primary behavior maladjustment," 2 as "paranoid state," 2 as "psychoses with convulsive disorders" and 2 as "family incompatibility." It is again evident from the table that the probability of subsequent adjustment in

the community bears a positive correlation with the test score; it is highest in patients who made a "discovery" and declines, although not progressively, in the other test groups. The column "without regard to supervision" shows a more progressive decline of the

TABLE 6.

SCHIZOPHRENIC CONDITIONS.

	No. of cases.	Continuously residing in community.						Continuously in hospital.	Per cent.	Died.	Per cent.
		Without supervision.	Per cent.	With supervision.	Per cent.	Total (without regard to supervision).	Per cent.				
Discoveries...	14	5	35.7	3	4.4	8	57.1	5	35.7	1	7.1
+ + +.....	22	5	22.7	4	18.2	9	40.9	11	50.0	2	9.1
+ +.....	30	4	13.3	2	6.7	6	20.0	22	73.3	2	6.7
+.....	32	3	9.4	4	12.5	7	21.9	24	75.0	1	3.1
- - -.....	10	..	....	2	20.0	2	20.0	8	80.0	..	....
Totals...	108	17	15.7	15	13.9	32	29.6	70	64.8	6	5.5

TABLE 7.

"OTHER PSYCHOSES."

	No. of cases.	Continuously residing in community.						Continuously in hospital.	Per cent.	Died.	Per cent.
		Without supervision.	Per cent.	With supervision.	Per cent.	Total (without regard to supervision).	Per cent.				
Discoveries...	7	4	57.1	1	14.3	5	71.4	1	14.3	1	14.3
+ + +.....	16	3	18.8	5	31.3	8	50.0	7	43.8	1	6.3
+ +.....	17	4	23.5	3	17.6	7	41.2	10	58.8	..	....
+.....	8	1	12.5	1	12.5	2	25.0	4	50.0	2	25.0
- - -.....	4	1	25.0	..	....	1	25.0	2	50.0	1	25.0
Totals...	52	13	25.0	10	19.2	23	44.2	24	46.8	5	9.6

percentage figures and provides a fair basis for prediction; similarly, the column "in hospital."

A comparison of the totals at the bottom of Tables 6 and 7 suggests that schizophrenic cases as a group have a significantly less favorable prognosis than cases ranged under the heading "other

psychoses." The probability of maintaining an unsupervised life in the community is 15.7 in the former as against 25.0 in the latter. The corresponding probability figures for maintenance of community residence regardless of supervision are 29.6 as against 44.2. A similar differential obtains in the probabilities of retention in the hospitals.

Of the 23 patients "residing continuously in the community," 2 were readmitted for brief intermittent hospitalization. Of the 24 patients "continuously in hospital," 4 had brief residences in the community.

In the manic-depressive group of patients (Table 8), predictability on the basis of initial test scores is possible only with regard to subsequent unsupervised activity in the community. The scores decreased in fairly direct proportion with test performance, from about 56 in patients who made "discoveries" or scored above 50 per cent in all three tests, (+++) to 20 in the group with a less than 50 per cent score in all three tests, (---). With reference to continued residence in the community, with or without supervision, the differential between the higher and lower score groups is relatively insignificant. Of the 36 patients who "continuously resided in the community," 5 were readmitted for brief intermittent hospitalization. Of the 12 patients "continuously in hospital," 2 had brief residences in the community. A glance at the totals at the bottom of tables 6 to 8 reveals that the probabilities of maintaining residence in the community, unsupervised or without regard to supervision, is lowest in the schizophrenic group, higher among the "other psychoses," and highest among the manic-depressive cases.

Of the 14 psychoneuroses, 8 patients made "discoveries;" 3 scored better than 50 per cent in all three tests, and 3 had a better than 50 per cent score in two tests. All maintained continued existence in the community after discharge; 1 only required supervision. This suggests a conspicuous tendency to spontaneous remissions, sustained for years, even in patients whose condition is sufficiently severe to necessitate commitment. Of the patients with involutional psychoses, 1 made a "discovery," 3 scored well in all three tests, 1 in two tests. Two only maintained continued existence in the community, one supervised, the other without supervision.

Evaluation of Tests.

The value of a test is measured by its (1) validity and (2) reliability. According to McBride,[1] validity implies that the test "actually tests what it purports to test." In this sense, validity has perhaps never been demonstrated in mental tests. It would, for instance, be impossible to prove that any intelligence test actually tests intelligence, since no agreement exists as to what intelligence is.

TABLE 8.

Manic-Depressive Psychoses.

	No. of cases.	Continuously residing in community.						Continuously in hospital.	Per cent.	Died.	Per cent.
		Without supervision.	Per cent.	With supervision.	Per cent.	Total (without regard to supervision).	Per cent.				
Discoveries...	18	10	55.6	2	11.1	12	66.7	4	22.2	2	11.1
+ + +.....	14	8	57.1	3	21.4	11	78.6	2	14.3	1	7.1
+ +.....	9	4	44.4	1	11.1	5	55.6	3	33.3	1	11.1
+.....	8	3	37.5	2	25.0	5	62.5	1	12.5	2	25.0
- - -.....	5	1	20.0	2	40.0	3	60.0	2	40.0	..	....
Totals...	54	26	48.1	10	18.5	36	66.7	12	22.2	6	11.1

When the tests here used were first elaborated in 1932 and 1933, little thought was given the question of what precisely they were intended to test. Both selection and application were empirical. One might conjecture that the "insinuations" tend to arouse the patient's curiosity about what is insinuated by the question. Similarly it may be assumed that the false and absurd statements about the time elements test the patient's intellectual alertness. However these are mere assumptions, the correctness of which cannot be demonstrated. It was only after investigation of patient's subsequent adjustment that a plausible answer was secured to the question of what was tested by the battery. Obviously it was an index to the ability to maintain life in the community. This factor correlates fairly well with the test scores and the latter may be said to measure the former. If the ability for unsupervised life in the community is considered exclusively, the correlation is rather rigidly

consistent with the test scores in the group of schizophrenic patients; its consistency is conspicuous for the manic-depressive patients, but only relatively so in the group of patients with "other psychoses." It may be legitimate to claim validity for the tests on the basis of this varying degree of consistency in the various groups of patients.

Reliability.—Terman [2] gave two main criteria for reliability. One refers to the reliability of applying the test, the other to its dependability when repeated with the same subject. A test is reliably applied if: (1) the person who gives the test masters its technic; (2) the subject's cooperation is secured. Since the tests here described consist of simple questions and statements the technic is easily mastered. We had occasion to study the cooperation of the patients. The nurses gave the tests to patients on the neurology and general medicine wards, but failed almost from the start. They asked a test question, and the patient in the adjoining bed exclaimed; "She is kidding you." When the nurse appeared on the following day she was greeted with the remark, "Hello, Miss Sunshine, what do you want to ask me today? Yesterday you told me I looked much better when I came here two weeks ago. Say, what kind of work do you do around here? I hear you work in the nut house." After a few days the effort had to be abandoned. The patients either refused to answer or became facetious and sometimes abusive. They had noticed they were being tested, and the fact of being tested was a current topic of conversation on the wards. On the psychiatric wards, reactions of this kind occurred twice only, when hypomanic patients who had made a "discovery" made it a point to call the attention of other patients to the fact that they were being tested. Even then, the effect on the other patients was negligible. Obviously the tests are unreliable and even impossible of application when given to nonpsychotic patients. Among them, the situation assumes soon or instantly the character of the "test setting."

We had a different but equally disturbing experience when the tests were given to a group of patients at the Chicago State Hospital. Although the patients were selected with a view to approximate the average ages and classifications of the group tested at the Psychiatric Institute, the results were disappointing. The

majority of the subjects returned laconic answers ("Yes," "no," "I don't know") or were evasive and abusive. It was soon apparent that the setting was not that of a "life situation." Due to a shortage of nurses at the hospital, the tests were given by student nurses who had enrolled for a three month course at the Psychiatric Nursing School of the institution. The students went to the ward for the period of testing and then left. The setting was again that of a test situation, and the unsatisfactory replies were probably the result of awareness on the part of the patients that the

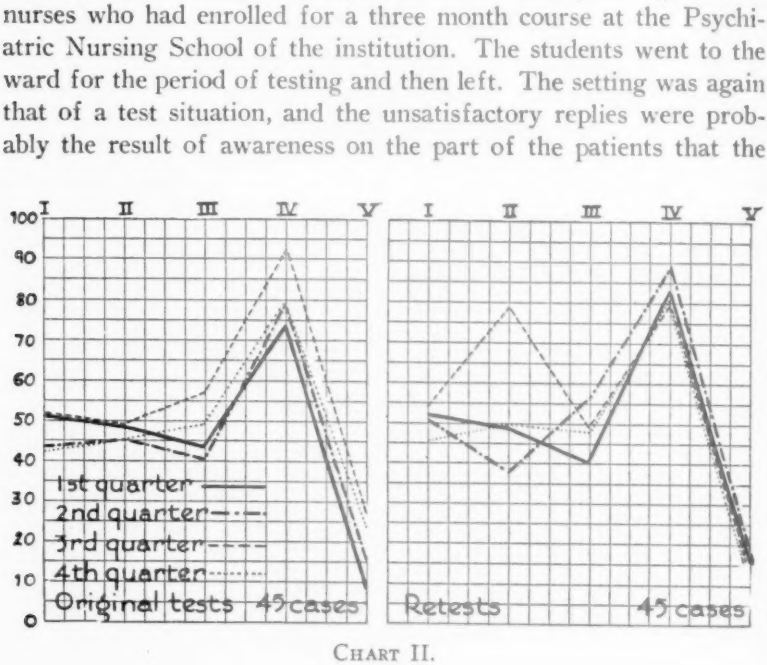


CHART II.

nurses were sent to the ward for the purpose of addressing statements and questions to them. The testing nurses were not an integral part of the daily environment, but persons specially delegated to exercise some official function. Due to this circumstance, the setting at the state hospital was not that of a reliable "life situation."

Terman's second criterion for reliability, the dependability of a test when repeated on the same subject some time after the original test was given, had already been applied when the three tests were automatically repeated with every patient in four successive quarters. As was shown in Chart I, the scores corresponded relatively closely, except that the scores improved generally in the third quarter. This constituted a fair evidence of dependability. A further check on dependability was obtained when 45 patients, after being

given the four quarters of the three tests during the first two weeks of admission, were subjected to exactly the same sequence of tests after the lapse of from three to six months. Chart II contrasts the original tests with the retests of the forty-five patients. The original tests show approximately the same correlation as demonstrated in Chart I. There is little variation in the scores of the first, second and fourth quarters, and improvement of performance in the third. As for the retests, the result coincides with those of the original tests in the first and fourth quarters and diverges from them in the second and third quarters. The precise mode of correspondence and divergence is clearly brought out in Chart III. The chart shows a well nigh point-to-point correspondence between originals and retests in the first and fourth quarters, a divergence but parallelism of results in the second quarter, and greater divergence, particularly in the "false time questions" (II), in the third quarter. Why the correspondence should be limited to the first and fourth quarters we cannot explain.

Conclusions.

1. The scores obtained with a set of three "life situation tests" in an unselected group of 233 patients correlate closely with the average length of residence in the community. No correlation was found in the manic-depressive group of patients.

2. The high degree of correlation suggests that validity may be claimed for the tests.

3. The tests may be said to be reliable and to yield similar or parallel scores if repeated from three to six months after the original application.

4. The test scores seem to afford a fairly valid basis for predicting the probability of continuous residence in the community after discharge from the hospital.

BIBLIOGRAPHY.

1. McBride, K. E., (in Th. Weisenburg and K. E. McBride): Aphasia, a clinical and psychological study. New York, The Commonwealth Fund, 1935, p. 133.

2. Terman, Lewis M.: The measurement of intelligence. Boston, Houghton, Mifflin Company, 1916.

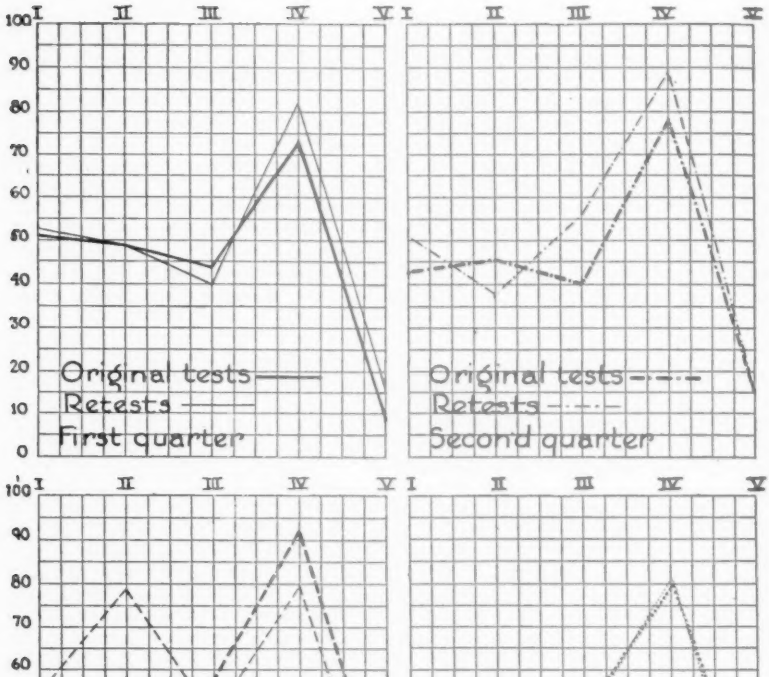

I II III IV V
100
90
80
70
60
50
40
30
20
10
0
Original tests
Retests
First quarter
Original tests
Retests
Second quarter
Original tests
Retests
Third quarter
Original tests
Retests
Fourth quarter

CHART III.

DISCUSSION.

DR. LLOYD H. ZIEGLER (Wauwatosa, Wisc.).—The writers of this interesting paper are to be commended for their attempt to throw light on prognosis. The estimation of prognosis has proven to be hazardous even for the most experienced of physicians when an individual patient has been under consideration. Statistical estimates of it offer splendid retreats in time of need.

With tests such as the authors present, I have had no personal experience. It has occurred to me that they may be testing a certain degree of alertness or awareness which is an asset in recovery, or which may even be an attribute of patients less seriously ill. Whether spontaneity can be tested by such methods would be difficult to decide.

If a large number of recovered patients were studied for a group of common attributes, a battery of tests might be devised that would elicit or demonstrate them, or correlate with a large number of such attributes. As an aid in such analyses, Karl Pearson's equation of correlation or modifications of it would be most helpful. By this equation one could correlate the reactions to a battery of tests with ability to get along outside a hospital, ability to earn one's living, or relative freedom from symptoms and signs.

Until prognosis gets beyond the realm of prediction by "hunches," I fear we will continue to allow our hopes to triumph over our experience.

It is gratifying to know that the authors are presenting an unfinished product and will continue to direct their efforts to these vital problems.

DR. H. DOUGLAS SINGER (Chicago, Ill.).—This series is presented with very definite objects in view. Some of you may not be familiar with the fact that I have had the impression for a long time that we have neglected very much, clinical studies of patients. We have been relying on laboratory methods and efforts to do things, rather outside the realm of medicine.

The thought of establishing tests of this kind was based on the hope that one might be able to establish in some such objective way, the fact that something is lost and something remains in the patient.

This method has evolved from the studies that Dr. Low and I were making some time previously, in relation to aphasia. It seemed to me that the methods which were adopted in the study of aphasia could be adapted, although we feel it is a much more complex problem, to the study of patients presenting other forms of mental illness.

The tests have been very different. Some were dropped at once because they had no significance at all, but certain tests remain, and undoubtedly there will be others.

This work is quite tedious. It requires a tremendous lot of work with the personnel who are carrying out these tests. The nurses have to be trained and checked all the time so that we know they are living up to the standards that are being developed. But I have the impression that observations of this kind are far more valuable than anything one can get from a daily record of a nurse as to what the patient has been doing. We get some information

which really gives us an insight into the kind of person we are dealing with, and that is an important matter.

The fact that the information we get correlates with what happens later is one of the things that we have been looking for, though we hardly expected to secure it; and this work stands out rather prominently in that respect as I think the figures will show.

I believe it is a method that is well worth following and developing. It does not require special laboratory facilities or training, but it does mean actual, daily contact and study of the patient himself, which I believe is going to give us a great deal more in the end.

DR. ABRAHAM A. LOW (Closing).—I merely make the remark that this is, of course, only a presentation of the quantitative side of the study. There is something to be said on the qualitative side of this information, and Dr. Singer has just mentioned the fact.

CONVULSIVE STATES WITH EVIDENCE OF BRAIN HEMIATROPHY.*

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In studying a syndrome as ill defined as the one we call epilepsy one is constantly trying to delimit the condition and to define the term. The conditions which crowd themselves under this broad term are many and varied and only some of them are characterized by convulsions. The basic pathological alterations that are responsible for the clinical picture have been the subject of much speculation throughout the ages. Gross and microscopic brain changes have been described in certain of the cases studied but with not enough uniformity to establish any definite pathology to explain all of the cases. It is most likely that there is no one pathological change nor series of changes that will explain even a majority of convulsive cases. Probably convulsions are symptoms which can be caused by a number of pathological states—anatomical, physiological and psychological.

The present paper deals with a group of convulsive cases which showed definite gross pathological changes in the brain that were demonstrated during life by roentgenogram and pneumo-encephalogram. The basic change was a mal-development of one side of the cerebrum with concomitant changes in the bones of the skull. Prout and Clarke writing in Spratling's [1] study of epilepsy report various thickenings of the skull in certain of their cases. They also quote Binswanger and others as responsible for the statement that "deformity (of the skull) is undoubtedly most frequent in the epilepsies engrafted upon an infantile cerebral hemiplegia and allied conditions."

Dyke, Davidoff and Masson [2] in 1933 described a roentgenological syndrome of cerebral hemiatrophy with homolateral hyper-

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trophy of the skull and sinuses that added reality to the previous statement. All their nine cases suffered from convulsions and hemiplegia that started in the early years of life—some of them even from birth. The syndrome described by these authors consisted of a uniform roentgenographic and encephalographic picture which they described as follows: "The roentgenograms reveal a thickening of the cranial vault on the same side as the cerebral lesion and also an overdevelopment of the frontal and ethmoid sinuses and of the air cells of the petrous pyramid of the temporal bone. On the encephalograms there is an enlargement of the lateral ventricle on the side of the cranial lesion and sometimes of the third ventricle and both of these structures are displaced toward this side." The etiology of the condition was either traumatic or infectious. Of the nine cases reported by Dyke *et al.*, three had their origin in a severe infectious disease, four dated from a birth which followed a difficult labor, one from a definite head trauma at one year of age and in one the immediate etiological factor was unknown. Frequently it is assumed that cerebral symptoms which have their onset immediately following a difficult labor are due to birth trauma. While this assumption is undoubtedly accurate in a number of cases it is often impossible to state with accuracy that the trauma is causative and that the condition did not exist before birth in a cerebral mal-development.

The material on which the present paper is based consists of nine cases of convulsive and other epileptic phenomena in children under 15 years of age who showed the roentgenographic and encephalographic syndrome described above. Of these, three were definitely traumatic in origin, one was due to the so-called birth trauma, four owed their beginning to an attack of encephalitis and one may well have been due to a cerebral hemorrhage that resulted from a severe paroxysm of coughing in the course of an attack of pertussis.

Clinical Cases.

TRAUMATIC.

Case I.—Female. Age 15 years. No. 26622. Admitted 11/19/35. This child developed normally up to her 15th month. She was talking and using words at 11 months of age. At 15 months she was struck on the forehead by an iron pipe and was unconscious for a number of hours. On return to consciousness she had repeated generalized convulsions which

lasted about a week. Following these series of convulsions the patient was paralyzed on the right side of her body—the arm more than the leg. No further convulsions; but the paralysis of the right arm and leg remained. She gradually acquired use of these limbs but they never were as good as those on the opposite side and she used her left hand for all skilled movements. Her speech has always been somewhat indistinct with a tendency to stammer.

At 10 years of age the patient began having a new type of attacks. These would begin with a dull aching pain around the navel which was variable—at times becoming quite severe and sharp. Coincident with this pain the patient's eyes and head would turn involuntarily to the left. She would become very tense and tremble all over but there were no convulsive movements and no loss of consciousness. Her appendix was removed but this did not relieve the pain nor the attacks. The attacks have occurred at irregular intervals—sometimes three in one day and again she would go for a week or so without an attack. It was thought that these attacks were precipitated by emotional excitement and indiscretions in diet. In September, 1935, she had an episode while at school during which she was confused, disoriented and had no memory of the attack after recovery. She wandered about in a dazed, confused state for a few minutes. She did not completely lose consciousness nor have any convulsive movements. In the two and a half months between this time and her admission the child had three of these attacks. She has always been backward in school—is now in the seventh grade at 15 years of age.

The positive neurological signs were a weak, rather spastic right arm and leg with atrophy and shortening of the right arm. There was some ataxia of the right arm and leg but none in the left. All her deep reflexes were much greater on the right than the left. The abdominals were less on the right. There was a positive Babinski on the right. Her optic discs showed slight pallor and the right side of her face was somewhat smaller and weaker than the left. I. Q. was .68.

X-ray showed a left hemihypertrophy of the bones of the skull and of the sinuses. Encephalogram showed the left ventricle to be dilated. The sulcus markings over this hemisphere were unusually coarse and the entire ventricular system was displaced to the left. The cortical markings on the right were normal in size and distribution. The entire left ventricle was enlarged and there appeared to be only a thin shell of tissue peripheral to it.

CASE II.—Female. 11 years of age. No. 32075. Admitted 2/9/37. This child was the result of a second pregnancy, born at full term after a precipitate labor. The baby is said to have breathed and cried spontaneously but had a convulsion shortly after her birth from which she recovered quickly. She refused to take the breast and was fed from a bottle. She was kept in an incubator for a while and seemed to have trouble in breathing. The mother and infant went home from the hospital on the tenth day and, except for a peculiar whistling aspirant note in her breathing, she seemed to be quite normal. At about three months of age the mother noticed that the

child did not use her left arm and leg as well as the right. She hardly moved them at all.

Following this she was treated in a number of hospitals for this condition. For a while she wore a brace on both arm and leg.

In August, 1934, while the patient was in the West Haverstraw Hospital the mother noticed that the patient was having some peculiar episodes of momentary lapses. In these she would turn and walk away from her mother apparently not hearing her mother call her. Her face was pale and her eyes staring and her pupils were dilated. These attacks lasted for a few seconds. First these spells came about once a week, later they came every day and then several times a day.

In December, 1935, the patient had a grand mal seizure which was characterized by a sudden stiffening of the body, twitching of the eyelids and marked rolling upward of the eyes, extension of the head, jerking of the arms, legs and trunk with loss of consciousness. At first such attacks came about at two and a half month intervals and later she was having three or four every week. In the later attacks she was incontinent of urine and bit her tongue. The child was never very bright in school but the mother did not feel that she had undergone any intellectual deterioration.

Physical examination showed a child whose left arm was definitely shorter and weaker than the right. The arm reflexes were fairly active and somewhat greater on the left. The abdominal reflexes were active, slightly greater on the right. The left leg was considerably shorter than the right, smaller in circumference in both the thigh and the calf. All the leg reflexes were active—greater on the left, and the Babinski was positive on the left. The optic discs were negative.

X-ray showed a thickening of the right side of the skull. Encephalogram showed the right ventricle greater than the left. The entire ventricular system was displaced to the right (Fig. 1). The enlargement in the right ventricle was limited practically to the right frontal horn and body.

Case III.—Male. Age 15 years. No. 31418. Birth and development normal up to three and a half years of age. At this time he fell down a flight of stairs, striking his head in right parietal region. Unconscious for 12 days. No bleeding from orifices. X-ray of skull negative for fracture. Left hemiparesis for three months following injury. This cleared and patient was symptom-free until the age of nine. Then began to suffer from left-sided convulsive seizures. Onset of typical seizure characterized by a whistling noise in both ears, stomach then "feels upset" followed by "itching" sensation of the left side of upper lip, head turns to the left and twitches. Left arm and leg then involved in rotary clonic movements. Patient loses posture control, does not become unconscious until seizure has been under way for several minutes. No incontinence, no tongue biting. Seizures last three to ten minutes and are followed by a "numb feeling" in the left upper and lower extremities. For past six years attacks have become progressively more frequent, occurring now one to three times weekly. Following an attack there is a sharp headache in the right frontal region. The patient has done reasonably well in

school although he had been obliged to remain out of school between the ages of 10 and 12 because of seizures.

Positive neurological findings: slight clumsiness in succession movements on left, deep reflexes in lower extremities more active on left than right, Babinski positive on left. Irregularity palpable in the right parietal bone.

X-rays: skull films show calvarium to be moderately dense and irregular in thickness. Petrous ridge on right slightly higher than on left. Right frontal sinus slightly larger than left. Encephalogram: right ventricle larger than left—the latter being within normal limits in size. Entire ventricular system is displaced to right. Temporal horn and atrium of right ventricle markedly enlarged.

CASE IV.—Female. Age 14 years. No. 25556. Service of Dr. Riley. Admitted 8/28/35. Birth and development normal until age of ten. At this time fell backward while roller-skating and hit back of head. The patient was not unconscious and there was no bleeding. Two or three hours later she awoke from sleep, vomited and had a clonic convulsive seizure, limited to the left side with drawing of head, face and eyes to the left. This lasted for three hours and was followed by headache. Four months later fell from rumble seat of car. Unconscious for several hours. One hour after accident had a convulsive seizure similar to the above. Incontinence of urine, coma for 24 hours following. Since then attacks have occurred once every two months. The attacks begin by patient feeling dizzy, then seems to veer to left in walking and the head begins to twitch to the left, face and eyes then involved followed by left arm and leg. Seizure usually lasts three hours but occasionally it is over in 15 to 30 minutes. The patient is unconscious, bites her tongue on the left and is incontinent of urine. Following an attack usually sleeps six or eight hours, awakening with severe frontal headaches. For two years previous to admission the patient has noted that the left arm and leg are weak, left leg drags in walking and she has difficulty in using left arm in skilled movements. Following an attack the weakness in the left hand is noticeably increased.

On neurological examination the patient drags her left foot when walking. There is a hemiparesis on the left and a hypertonicity in the left musculature. There are diminished associated movements of the left arm. Non-equilibratory tests show dysmetria and ataxia on the left. Deep reflexes are somewhat increased on the left and there is a left Babinski. There is slight involvement of muscle-joint-tendon sense on the left. There is a left facial weakness of central type. Right palpebral fissure less than the left. There is a slight pes equinus on left. I. Q. 95.

Encephalogram shows the ventricular system to be displaced slightly to the right. Right lateral ventricle is larger than left. Third ventricle displaced slightly to right.

ENCEPHALITIC.

CASE V.—Male. Age 11 years. No. 32299. Measles at age of 14 months complicated by pneumonia and then meningitis. Ill for two months. During

this period status epilepticus for 24 hours, seizures involving primarily right side. Patient recovered and apparently well until seven years of age. Then began to have seizures characterized by head turning to right, eyelids fluttering, mouth pulled to right, right arm drawn up, staggering to the right, no falling, no consciousness of seizures. Between ages of 7 and 11 years this type of seizure became progressively more frequent. No personality change.

Positive neurological findings: weakness in right arm and leg, unsteady standing on right foot, deep reflexes in upper extremities equal, lower extremities slightly more active on right, double plantar flexion, right pupil 3 mm., left 5 mm. Right leg slightly shorter than left. In walking right heel does not touch floor. Muscle volume of right extremities slightly less than that of left.

X-ray findings: left parietal bone slightly thicker than right. Left frontal sinus larger than right. Encephalogram: left lateral ventricle slightly larger than right. Questionable shift of ventricular system to the left.

Case VI.—Female. Age 12½ years. No. 30297. Service of Dr. Riley. Admitted 9/22/36. Normal birth and development until an attack of acute encephalitis at age of 14 months. This attack was ushered in by convulsive seizures. The patient was immediately placed in the Jewish Hospital in Brooklyn where she was unconscious for one week. Several lumbar punctures performed and diagnosis of acute encephalitis made. Discharged after two weeks. During following month had four or five attacks a day consisting of blinking of eyes and deviation of jaw to the right, lasting about five minutes. Since then has had retarded development of right arm and right foot with varying degrees of paralysis. At age of five operated on at Flower Hospital and right Achilles tendon lengthened. At age of eight began to have attacks characterized by mental confusion and purposeless activity. Soon after began to have attacks characterized by involuntary movements of shoulders, blinking of the eyes, twitching of the mouth on both sides, rotation of the pelvis and unconsciousness. Attack would last three to five minutes, almost daily in occurrence. At age of 10½ began to have grand mal seizures during sleep characterized by clonic movements and incontinence of urine. One week before admission had 50 convulsive seizures in one night.

On neurological examination there was a hemiplegic gait on the right, musculature on the right less developed than that on the left. Paralysis of extensors of right hand and dorsiflexors of right foot. Unable to stand on right foot. Non-equilibratory tests impossible right hand. Marked dysarthria. Deep reflexes increased on the right, Babinski and Chaddock positive on right, nystagmus on upward and lateral gaze, tongue protrudes to the right. Thoracic kyphosis and scoliosis. Right homonymous hemianopsia.

X-rays: ventricular system displaced toward left. Left ventricle larger than right (Fig. 2). Left side of skull thicker than right. Left frontal sinus larger than right. Mastoid cells more developed on left than right. Left petrous ridge and cribriform plate higher than right (Fig. 3).

Case VII.—Female. Age nine years. No. 24776. Admitted 6/25/35. According to the history this child at eight months of age was treated in Mt.



FIG. 1.—Case II. Note enlarged right ventricle and entire ventricular system displaced to right.



FIG. 2.—Case VI. Note left ventricle larger than right and ventricles shifted to left.



FIG. 3.—Case VI. Note larger frontal sinus on left. Mastoid cells larger on left. Petrous ridge and cribriform plate higher on left. The latter is tilted definitely and the crista gallæ is bent to the left.

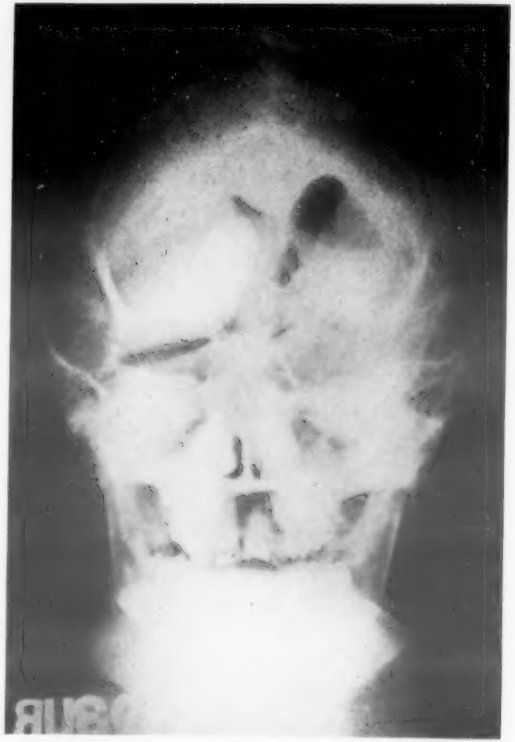


FIG. 4.—Case VII. Note larger left ventricle and entire ventricular system displaced to left. Skull is thicker on left.

and the crista gallæ is bent to the left.



FIG. 5.—Case IX. (Encephalogram at 5 years of age.) Note larger left ventricle. No displacement of ventricular system. No difference in thickness of skull on two sides.



FIG. 6.—Case IX. (Encephalogram at 10 years of age.) Note the great increase in size of left ventricle. Ventricular system now shifted to left and left side of skull thicker than right.

Sinai Hospital for some kind of seizures. The mother was told that she would outgrow them and apparently she did. At four years of age she had an isolated convulsive seizure of which no particular history could be obtained. From that time on the child appeared well until the onset of the present illness.

In June, 1933, when the patient was seven years of age she contracted measles and with this she had a severe convulsion which lasted 20 minutes. She was removed to the Willard Parker Hospital where she remained for 10 days. Following the convulsive seizure she had a right hemiplegia for which she was transferred to Bellevue Hospital where she remained two and a half months; when she left she was able to walk. Her convulsions continued. With these convulsions her head would turn to the right, her eyes upward, pupils dilated, face drawn to the right, left arm held out and she would fall to the right. There was jerking of both the right arm and leg and she would remain unconscious for about one-half minute. On one occasion she bit her tongue and within the past few months she has been incontinent of urine during the attacks. The attacks usually occur about once a month. The patient has petit mal seizures daily. In these she twitches her right arm a little but if she is talked to she comes right out of the attack. After these attacks she is usually very sleepy. The attacks appear to be decreasing in frequency and severity.

Neurological examination showed a child with a right hemiplegia. All the deep reflexes were greater on the right. The abdominal reflexes however were equal and there was no Babinski. There was a slight right facial weakness which was more marked on smiling. The I. Q. was 88.

Encephalogram showed the left lateral ventricle to be enlarged and the whole ventricular system displaced to the left including the third ventricle. The skull was definitely thicker on the left. The sinuses were not particularly enlarged (Fig. 4).

Case VIII.—Male. Age 14 years. No. 33141. Service of Dr. Pardee. Admitted 5/10/37. There are no data in the history of any etiological factor. This boy had a mild attack of scarlet fever about six months before the onset of his twitches. This was slight and uncomplicated and he recovered rapidly. In the spring of 1934 he began to notice that he was having occasional twitches of his face on the right. These occurred about every three to four weeks and gradually increased in frequency and severity. At first he was able to lessen his attacks of facial twitching by stuffing a handkerchief into his mouth or by blowing his nose. If he did not get his left hand up to his mouth at once he was unable to control the attack which usually lasted about two minutes. The boy is left handed. During the attack the right hand is held close to his side and is somewhat tremulous. About the middle of April 1937 he went to a church supper. There he had a slight attack with twitching of the right side of his face but he ate a hearty meal and went home early, did his homework and retired at 10 p. m. Shortly afterward an attack of twitching started in his face. He got out of bed, turned on the light and fell forward but he was able to put out his arm to

save himself. He did not lose consciousness. After a few moments his arm started to beat back and forth in a rigid fashion and he was unable to stop the movement. The entire seizure lasted about four minutes. He was helped back to bed and about five minutes later he was able to get out on the floor but he felt weak. Ten minutes after the attack he said that he felt perfectly well. There was no incontinence nor tongue biting and the consciousness was not really disturbed.

Neurological examination showed rather poor coordination in the right arm and likewise in the right leg. The right pectoral and triceps jerks were somewhat more active on the right but all of the others were equal on the two sides. The Babinski was negative. There was a slight volitional facial weakness on the right.

Encephalogram showed that the left lateral ventricle was considerably larger than the right. The other ventricles appeared normal. The left parietal bone was thicker than the right and the left frontal sinus was somewhat larger.

CASE IX.—Female. No. 36545. This patient was first admitted at the age of five years. Normal birth and development up to three and a half years of age when she contracted whooping cough. After she had been ill of whooping cough for six weeks and had been coughing continuously the patient "suddenly threw up and turned blue" following a paroxysm of coughing. Immediately she suffered a right-sided convulsive seizure which lasted for one and a half hours followed by unconsciousness for 24 hours. She was taken to Riverside Hospital where she remained for six weeks. Associated with this illness the patient had a total loss of speech which gradually returned. With recovery the patient gradually improved for the next one and a half years at the end of which time a second convulsion occurred. This was five weeks previous to her first admission here. During this five weeks' period the patient had had one to four grand mal seizures daily. These attacks were characterized by generalized clonic spasms, a fall with loss of consciousness, frothing at the mouth, incontinence of urine and rolling up of the eyes. Following an attack the patient was dazed and sometimes fell asleep.

Neurological examination at this time showed a right-sided weakness. Arm and leg reflexes more active on the right, abdominals diminished on right, right facial weakness of central type. The patient appeared intelligent but did not answer questions.

Encephalogram: left lateral ventricle considerably larger than right. No definite displacement of ventricular system but ventricles were not completely filled with air (Fig. 5).

The patient was admitted for the second time at the age of ten years. Interval history shows that right-sided convulsive seizures had continued with varying frequency in the five year interval. In addition the patient complained of "dizzy headaches" above the right eye which were not associated with convulsive attacks.

On neurological examination there were the following positive findings: muscle strength slightly decreased on the right, no muscular atrophy, deep reflexes more active on the right, right facial weakness, distinct dysarthria.

Encephalogram: there is a marked enlargement of the ventricles as compared to the previous encephalogram taken five years ago. There has been a disproportionate increase in the size of the ventricles compared to the growth of the skull. Left lateral ventricle considerably larger than the right. The latter is also somewhat larger than normal. Left parietal bone and left frontal sinus are larger than those of the opposite side (Fig. 6).

COMMENT.

In these nine cases the one point in common besides the roentgenographic findings was the presence of convulsions, but there was no uniformity in this regard. Of the four traumatic cases two (Cases I and II) had generalized convulsions at the time of the injury; one (Case II) had generalized convulsions starting nine years later; two (Cases III and IV) had unilateral convulsions. One patient (Case I) had no convulsion after the first week but periods of confusion starting 11 years later. Of the encephalitic and vascular hemorrhage cases three (Cases V, VI and VII) had generalized convulsions at the time of onset; one (Case IX) had unilateral convulsions at the start and one (Case VIII) had none. The later appearing convulsions were unilateral in four patients (Cases V, VI, VIII and IX) and generalized in one (Case VII).

All the patients showed evidences of weakness with increase of weakness on one side of the body which was the side opposite to the brain lesion. In none was the hemiplegia complete. Two of the children (Cases I and II) showed hypoplasia of the paralyzed arm and leg and one (Case VI) had hemianopsia on the side of the motor weakness.

As did the convulsions and hemiplegia, the roentgenographic and encephalographic findings showed marked variations in degree. While there was a definite tendency for the amount of cerebral hypoplasia to vary with the duration of the condition this was not a uniform thing. For instance Case V with a duration of 10 years showed slight findings while in Case VIII which had lasted but three years the cerebral hypoplasia was marked.

Discussion.

The nature of the change which has taken place in these brains is a matter interesting to contemplate. The basic fact is that in these children one side of the cerebrum did not grow as fast as the other and this lack of growth was the result of localized injury or disease. The normal brain half grew normally and with it the skull which maintained its external symmetry. This skull growth takes place by an increase of bone on the outer surface and a disappearance of bone on the inner to make room for the growing brain. Where the brain does not grow or grows more slowly on one side the process of bone disappearance on the inner surface does not take place and the skull is thicker. Likewise when the brain does not grow symmetrically the air spaces of the skull, including the accessory sinuses, on the side of less brain growth increase in size with the increasing size of the skull and are not restricted in size by the growing brain within the skull.

Thus one can explain the X-ray appearance of the skull but one is faced with the problem of what constitutes brain growth and why did one hemisphere stop growing in these cases? It is well known that no new nerve cells are formed in the human brain after about the first two years of life at which time the cells of the cerebral cortex have reached their maximum number.

Donaldson [3] is authority for the statement that "brain growth consists mostly of increase of myelin." Quoting Schlossber he states that the ether-soluble constituents of the brain, consisting mostly of myelin, make up 3.77 per cent of the brain at birth and 15.25 per cent of adult brains. The nerve cells and their processes increase but very slightly in size while the brain volume increase is considerable. The nervous system volume, according to Donaldson, is 376 c. c. in the child at birth and 1340 c. c. in the adult. The average weight of the male brain at birth is 372 grm. and of the male adult 1360 grm. This increase consists almost entirely of myelin.

The structure of brain growth then is the myelin which surrounds the nerve fibers and these children in their development have been deprived of the normal myelin increase in one cerebral hemisphere. Most likely the growth of all the other brain structures was likewise retarded. It was assumed at first that this was a pro-

gressive phenomenon but it remained for the evidence of Case IX to prove that this is true. Comparison of the encephalograms of this child at 5 and 10 years of age shows that the process has been a progressive one. The left cerebral hemisphere is shown to have grown very little in those five years. The left lateral ventricle has considerably increased in size and the thickness of the skull and the size of the sinuses have increased proportionately. One even gets the impression from the pictures that at 10 years there is less brain tissue on the left than there was at five years. Of this however one can not be sure.

That these obvious brain changes have something to do with the convulsions and other epileptic phenomena there can be little doubt. In most of the cases the convulsions were principally on the side of the body controlled by the atrophic or hypoplastic brain. In some of the patients the limbs of the convulsive side were smaller and shorter than the others. One might speculate on the relationship of all these findings to the convulsive state, but until more is known of the nature of the epileptic manifestations nothing concrete could be offered.

Conclusions.

1. Attention is called to a roentgenographic syndrome of hemilateral cerebral hypoplasia and atrophy with concomitant increase of thickness of the skull and enlargement of the head sinuses which occurs in certain convulsive states.

2. Nine cases are described which presented this syndrome. The etiological factors were traumatic, inflammatory or vascular.

3. The nature of the pathological process is discussed from the point of view of brain growth.

BIBLIOGRAPHY.

1. Prout and Clark: Chapter on pathology in Spratling: Epilepsy and its treatment, p. 313. Philadelphia, 1904.
2. Dyke, Davidoff and Masson: Cerebral hemiatrophy with homolateral hypertrophy of skull and sinuses. Surg. Gyn. and Obst., LVII: 588-600 (Nov. 1933).
3. Donaldson: The growth of the brain. New York, 1909.

INVOLUNTARY EYE MOVEMENTS AS A CRITERION OF DEPTH OF INSULIN COMA.

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For the past year, the authors have been using involuntary eye movements as one of the criteria of depth of therapeutic insulin coma and have found them a useful adjunct in the management of the treatment. The reactions begin early in the course of coma, undergo a series of characteristic modifications as shock progresses and only disappear when the deepest levels are reached. The steps seem to be regular, recognizable, constant in their order, and they appear to bear a definite relation to the depth of shock. The pattern is similar to that seen under ether and chloroform anæsthesia although differing in many details. A review of the literature fails to reveal any previous description of the sequence outlined above although several of the individual findings have been described. Benedek [1] gives an account of the exophthalmos and staring so often seen. He also mentions the presence of nystagmus and ocular paralysis in his case reports. Von Angyal [2] also describes pendular nystagmus as an occasional finding. Others only mention the nystagmus and deviation in passing, as for example De Morsier and Bersot.[3] It will be the purpose of this paper further to describe the eye findings under insulin and to attempt to show how these may be used in the management of therapeutic coma.

The sequence in a typical case may be described in six stages.

I. Latent Period.

A shock producing dose of insulin is injected at 7.30 a. m. and for a period of one to three hours no ocular changes take place. The patient may perspire or show somnolence but can speak and swallow.

II. Early Involuntary Eye Movements, Nystagmus on Attempted Fixation.

The earliest characteristic finding is a nystagmus which has a fast as well as a slow phase. It is best seen when the patient at-

tempts to fix an object off the midline and may occur when he shows only slight evidence of shock, such as mild confusion and slight perspiration. The presence of nystagmus with a quick phase is reliable evidence that deep coma has not yet set in. In this stage the patient can usually speak a few words, can swallow, will resist tube feeding and can localize tactile stimuli in a vague manner. Early clonic reactions may be present and states of excitement and respiratory spasms may occur. There are several less constant eye reactions at this time. Most prominent is lid retraction which gives the patient a startled exophthalmic appearance that may not disappear until coma is well advanced. This is most marked in cases with strong clonic movements. As noted by Benedek,[1] lid retraction is accompanied by a decrease in normal lid movements. Pendular nystagmus and conjugate deviation may also occur at this level but only in a patient who is at rest or sleeping. When he is roused they disappear. This grade of coma corresponds roughly to Braunmühl's "light coma,"[4] or Von Angyal's phase I and II.[2]

III. Spontaneous Pendular Nystagmus or Conjugate Deviation Which Do Not Disappear on Stimulation.

As consciousness becomes further impaired, the lid signs, if they have been present, tend to disappear. Attempts at fixation are no longer made, and a large majority of cases develop a pendular nystagmus with five to 30 excursions per minute. Less often there is a strong, fixed conjugate deviation, usually lateral, less often downward. In very rare cases a uni-ocular nystagmus may be seen. This last reaction has been described by Alfoudry,[5] in cases of contra-lateral brain damage. Stimulation does not abolish the pendular nystagmus or the deviation, although it may produce a short bout of irregular eye movements superimposed on them. The patient can no longer localize painful stimuli, but corneal and tendon reflexes remain active. The plantar response may be normal, pathological or absent. This stage corresponds to Braunmühl's deep coma or Von Angyal's phase III and IV.

During the height of severe extensor spasms or marked spastic respiratory difficulty, all eye movements tend to disappear and the eyes remain fixed, in mid-position, usually with somewhat dilated pupils.

IV. Nystagmus on Stimulation Only.

The next level is characterized by the fact that eye movements may be elicited although all spontaneous movements and deviations have ceased and the eyes remain at rest in or near the mid position. If the head is rolled in such a way as to carry the patient's face quickly from one shoulder to the other a short period of pendular nystagmus will follow. This level also corresponds to Braunmühl's deep coma. Patients are not allowed to reach this stage till they have had several comas, and are never left at this level for over one hour.

V. No Nystagmus on Stimulation.

With further increase of depth, the eyes become fixed in mid-position and respond very slightly, if at all, to stimulation. At this time one finds a maximally contracted, fixed pupil, decreased or absent corneal reflexes and decrease in the general muscle tonus. With regard to corneal reflexes, the authors agree with Leipmann [6] that they disappear entirely only when coma has passed beyond the limits of safety. The pupils dilate if there is any respiratory obstruction or a spasm of excitement. Tendon jerks may be normal or decreased and the plantar responses may be absent, normal or abnormal. This corresponds to Von Angyal's stage V.[2] Patients are not left at this stage for over 30 minutes.

VI. Paralytic Respiratory Phenomena.

If depth increases beyond stage five, an intercostal paralysis with respiratory depression sets in and the patient shows gasping, shallow irregular respirations and dilating pupils. (Von Angyal's stage VI.)

If a patient in one of the deeper stages of shock be given intravenous glucose one can observe a rapid but fairly exact reversal of the order of events already described.

I. Amelioration of respiratory difficulties: this occurs often after a few cc.'s of glucose.

II. Return of nystagmus on stimulation.

III. Spontaneous pendular nystagmus of increasing speed and amplitude of excursion.

IV. Marked conjugate deviation of the eyes, often downward.
V. Nystagmus on fixation.
VI. Awakening with a cough.

While the above described sequence obtains in a typical case, it must be emphasized that the description is schematic, and many variations occur. One often sees omission of certain phases and prolongation of others. In rare cases all spontaneous eye signs are lacking. For this reason the ocular reactions must always be considered in conjunction with other criteria of coma depth and do not necessarily take precedence when they seem to contradict these findings.

In spite of the above limitations, observation of the eye movements affords a type of information that is distinctly useful in the management of insulin coma. It helps one to recognize the earliest stages of coma in negativistic, uncooperative, catatonic individuals. It provides a rough quantitative measure of depth and serves to warn the physician when a patient needs special attention on this account. It is of particular use in following the reaction to gavage when treatment is terminated. One finds quite regularly an initial but transient deepening of coma. If this deepening is marked and fails to disappear within 15 or 20 minutes the authors feel that intravenous glucose is indicated. Especially in the early treatments of a given case evidence of marked increase of shock is not unusual and is particularly dangerous as Braunmühl [1] states.

In many cases the patient fails to wake completely, although he can speak a few words and cooperate to a certain extent. Examination of the eyes helps to distinguish between negativism and the persistent effects of hypoglycemia. Nystagmus and ocular deviation are often seen while such individuals are receiving intravenous glucose and precede their complete awakening.

Summary.

Nystagmus and other involuntary eye movements are a constant feature of insulin shock. During the course of shock these reactions undergo a constant and progressive series of changes which have a distinct relation to the depth of induced coma, as judged by other criteria. One first sees a nystagmus characterized

by a slow and a fast component. This is followed by a spontaneous pendular nystagmus or by conjugate deviation. Later, all spontaneous eye movements cease but nystagmus can be still elicited by stimulation. Finally, a depth is reached where no eye reaction can be elicited and this is shortly followed by signs of respiratory depression. The course, in general, seems to be one of stimulation of eye movements initially, followed by gradual depression. While the exact sequence of events is somewhat variable, successive observations afford a useful and delicate check on the progress of therapeutic coma if they are used in conjunction with other findings. One can judge with particular accuracy whether a given coma is becoming deeper or lighter.

BIBLIOGRAPHY.

1. Benedek, L.: Insulinshock Wirkung auf die Wahrnehmung. Berlin, S. Karger, 1935.
2. Angyal, L. v.: Über die verschiedenen Insulinshocktypen und ihre neuropsychopathologische Bedeutung. Arch. f. Psychiat., 106: 662-680, 1937.
3. de Morsier, G., et Bersot, H.: Les Troubles cérébraux dans l'hyperinsulinémie provoquée. Schweiz. Arch. f. Neurol. u. Psychiat. (Ergänz), 39: 101-108, 1937.
4. Braunmühl, A. v.: Die Insulinshockbehandlung der Schizophrenie. Berlin, Springer, 1938.
5. Alfoudry, I.: Du nystagmus provoqué dans les états comateaux. Rev. d'oto—neuro-ophthalmologie, 15: 161-169, March 1937.
6. Liepmann, W. v.: Über das Koma und Reflexanomalien bei der Insulinshocktherapie der Schizophrenie. Schweizer Archiv f. Neurol u. Psychiat., 40: 133-140, 1937.

ANOMALIES AND DANGERS IN THE METRAZOL THERAPY OF SCHIZOPHRENIA.*

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Since the original publication by L. von Meduna,[1] the convulsive therapy of schizophrenia and other psychoses has become extensive, and a large body of literature on the subject has been published. Results have been promising and sometimes spectacular, but in view of the drastic nature of the treatment and the possible complications it seems desirable that all adverse effects should be reported.

Prolonged apnea necessitating artificial resuscitation has been frequently mentioned.[2-3] Dislocations of the mandible are well known as a common complication of the seizure induced by metrazol.[1-7] Dislocations of the shoulder have been reported,[1-3, 5-6, 8] and as occasional accidents, fractures of the humerus,[3] the femur,[3, 9] the scapula,[10] and of other bones.[9]

Other complications occasionally mentioned include laryngeal spasm with bloody expectoration persisting for hours after the seizure,[11] avulsion of the tuberosity of the humerus,[6] temporary auricular fibrillation,[12] tearing of the masseter muscle,[2] loosening and breaking of teeth.[2]

Deaths have been reported [13-14] but these have been surprisingly rare. Five are described in the available literature. Autopsy reports one to have shown aortic insufficiency,[13] another hypernephroma,[13] two pulmonary embolism [13] and the fifth, fibrosis of the myocardium and mitral disease.[14]

Fatalities occurring from excessively severe or prolonged seizures *per se* have not been noted in the literature available to the writers. In fact the prevalent opinion seems to be that the seizure following intravenous metrazol is consistently regular in duration and severity.

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Friedman [4] states that although as many as six individual convulsions were noted after the intramuscular injections of camphor in oil, "only a single convulsion immediately ensues" after the intravenous administration of metrazol. Reese *et al* [7] state that in their series of over 600 metrazol treatments no instance of repeated convulsions or delayed reactions occurred and they conclude that the induced convulsion is always single and immediate.

The literature leaves one with the impression that only a single seizure is to be expected after intravenous administration of metrazol. McAdoo,[15] however, reports a typical major seizure occurring 16 to 20 minutes after an intravenous metrazol injection which had produced only mild clonic manifestations initially. Nightingale [9] mentions five cases among 44 treated which reacted with delayed or repeated seizures. One of these had a latent period of 10 minutes between the injection and the seizure. Another had three successive seizures after receiving 5 cc. of metrazol intravenously. Three other patients had secondary seizures 1 hour, 3 hours and 8 hours after the first induced seizure. No descriptive details concerning the type or duration of these anomalous reactions are given. Robards [16] mentions a delayed convulsion which occurred 1½ hours after an injection of metrazol.

Except for these instances no record of multiple or delayed seizures were found in the literature.[2-42] Western [43] who has reviewed extensively both the American and foreign literature informed the writers on January 15, 1939, that he had not discovered any reports of multiple seizures besides those mentioned.

In view of the widely accepted opinion that only one seizure follows the usual administration of metrazol and the rarity of reported observations to the contrary it is felt that the present case may prove of interest.

CASE REPORT.

No. 124472, N. B., white male, age 20 years. Admitted to the University Hospital October 17, 1938, with a history of vague personality changes during the previous year. Two weeks prior to admission he showed definitely psychotic symptoms, remaining mute whole days at a time, grimacing, murmuring to himself as if in response to hallucinations; but occasionally indulging in sudden, bizarre behavior. An example was his untimely emergence on the dance floor where he interrupted two professional dancers and attempted to go on himself with the female member of the team. A complete

physical examination, laboratory study and neurological examination showed nothing significant. Psychiatric examination presented an athletic body type, carelessness in dress, mannerisms, unresponsiveness or irrevelance, a flattening and inappropriateness of affect, unsystematized delusions and auditory hallucinations. During several days of observation the patient often lay for long periods with his eyes closed. He sometimes smiled with the typical glassy, artificial grin of the schizophrenic. He stated he was receiving messages from people in distant cities, and that a man named P——— was incessantly working to destroy him. Curious stilted postures were assumed and he often held his hand up with three fingers erect or made a circle with the forefinger and thumb, winking his eye in a strange deliberate gesture. Diagnosis: schizophrenia, catatonic type.

Metrazol therapy was begun October 19, when 3.7 cc. of 10 per cent solution I. V. produced, 30 seconds after injection, a typical major seizure with tonic and clonic phases. The seizure was more violent than the average in our experience and lasted 75 seconds. A large quantity of urine was voided just after the seizure; eyes were conjugately deviated upward and to the left at the end of the seizure.

The same amount on October 21st failed to produce a major seizure. An additional 1 cc.[13] one minute after the first also failed. Twitching of the facial muscles and fingers occurred and after the supplemental injection the patient raised the trunk to a 30° angle from the bed and held his arms rigidly before him in an apparent catatonic posture.

On October 22, 3.7 cc. with 1 cc. a minute later again failed to cause the seizure. An hour later 4.3 cc. caused a severe seizure lasting between 1½ and 2 minutes; the clonic phase occupied nearly all the time of the seizure, the tonic phase lasting less than 15 seconds. At the end of the seizure the eyes were again conjugately deviated upward and to the left. Pupils at the end of the seizure were contracted. Two minutes later when the patient passed into a dazed, active state the pupils were dilated. It was interesting to note that when the patient opened and closed his eyes the pupils could be definitely observed to dilate to the light stimulation as the lids opened.

October 24, injection 4.5 cc. failed to cause a seizure. One hour later 5.1 cc. produced a typical major seizure lasting slightly over a minute.

On October 26, 5 cc. were given; after 30 seconds the patient stiffened and very deliberately, the head and then the body began to twist towards the left. This rotation was continued until the body completed an entire turn of 360°. As the body turned the trunk rose from the bed as if the patient were attempting to arise while the spasm, simultaneously seizing him, took him on through his revolution. The rapidity of the turning motion increased as the body progressed. After completing this preliminary movement the head twisted slowly to the left and both eyes became fixed in extreme left deviation. A few twitches now occurred and the patient passed into rigid tonus, with right arm flexed, left arm extended. The mouth opened widely as usual.[7] The patient uttered a hoarse scream which lasted 15 seconds. Since the beginning of his torsion movement he had apparently been entirely unconscious. The tonic phase from the time the mouth opened lasted 20-25 seconds. Violent

clonic motions lasting 40 seconds succeeded the tonic phase. The patient became very cyanotic. At the end of the seizure the eyes were still deviated to the left.

On October 28, 5.4 cc. were given. The head again twisted to the left, and a typical convulsion lasting 65 seconds occurred.

On October 30, 5.2 cc. failed to cause a seizure. One hour later 5.4 cc. were given. Twenty seconds later the head twisted to the left in a slow torsion movement, the eyes again deviating left. After a few preliminary twitches the patient entered the tonic phase, the right arm again drawing up in extreme flexion with clenched fist on the chest, the left arm extending by the trunk, also with fist clenched. During the clonic phase, which was severe but less violent than during the seizure on October 26, the head remained twisted to the left. The entire seizure lasted between 1½ and 2 minutes. Respiration was regained spontaneously. The patient relaxed and passed into coma. The conjunctival vessels were markedly injected, so gorged that superficial inspection raised the question of hemorrhages. These, however, were not present. After lying thus in complete relaxation between 1½ and 2 minutes the right arm assumed rigidity in flexion. Tonus increased generally and deliberately, the patient finally assuming a position of marked opisthotonus with extremely severe muscular contraction. Distinct popping and cracking sounds were heard in the joints as the seizure molded the body. Respiration ceased and the patient became increasingly cyanotic. He remained thus, body arched and resting on head and heels, for a minute and a half. Meanwhile a nasal catheter was inserted and oxygen administered. Efforts at artificial respiration were altogether fruitless due to the rigid thorax which resisted compression. After 1½ minutes, without undergoing any clonic motions, the body slowly relaxed. As artificial respiration was instituted as soon as the chest relaxed, it is not known whether respiration would have occurred spontaneously.

After this rather alarming complication metrazol was discontinued, the patient having shown no real improvement. Nine periods of coma were then produced by the insulin hypoglycemic method. It is interesting to note that with these he also assumed a position of opisthotonus, and coma had to be interrupted each time because of severe convulsions.

No improvement resulted and he was dismissed December 3, in excellent physical condition, apparently having suffered no damage from his rigorous therapy.

COMMENT.

The patient presented here had no history of seizures prior to the present treatment. He was an athletic and particularly robust young male. His reactions could not therefore be readily ascribed to an inherent convulsive tendency or physical inferiority.

The most unusual feature of these reactions was the second and purely tonic seizure which occurred after the single injection of

metrazol. This was viewed by the writers with some alarm particularly because of its striking similarity to reactions seen in animals which were given lethal doses of metrazol.[44] A series of mice given metrazol subcutaneously, after varying numbers of seizures roughly similar to the typical seizures observed in patients, on receiving what proved a lethal amount of metrazol, almost invariably passed, after the usual seizure, into extreme and progressive hypertonus which persisted until death. The rigidity, in fact, persisted after death, the corpses remaining erect, hard as if frozen, their tails pointing aloft like flagpoles.

Published observations[45] of successive seizures with the similar drug triazol, which often causes a series of convulsions, might be taken to indicate that little danger lies in such reactions as the one reported here. Walk and Mayer-Gross [45] conclude that with triazol, despite repeated seizures, status epilepticus does not occur and suggest the possible therapeutic advantages of their induction. Since the elimination of triazol is much slower than that of metrazol [45] it is perhaps logical to expect more than one seizure and to interpret such reactions as being induced by the natural action of the drug. With metrazol, however, such an explanation is less reassuring. Because of the known rapidity with which its convulsive effects vanish [3, 7, 13] one cannot but suspect that, in case of metrazol, persistent or repeated convulsive manifestations may be due to some serious disorder induced in the organism by the drug which still threatens after the effects of the drug itself have passed. Hence we cannot feel that the reported practice [3] of repeating the injection after five minutes when a seizure does not occur is altogether a safe procedure.

Another interesting reaction in this patient was the tonic twisting movement, possibly similar to torsion movements described in insulin shock and sometimes ascribed to disorders of the extrapyramidal system,[46] but which in this patient completed a circuit.

This patient during insulin treatment had extremely severe seizures. It was in fact not possible to produce coma without obtaining seizures of such severity that treatment had to be interrupted. This further suggests that the patient's margin from seizure to death might naturally be very narrow rather than that these reactions be assumed outright as a complication of the specific drug metrazol. An assumption that this patient's second seizure with

metrazol was due to unusual susceptibility, as his reactions to insulin might imply, becomes illogical in view of the fact that he failed so often to have convulsions with moderate doses (5 cc. 5.2 cc.) which had previously been effective. We have had other patients who continued to have a seizure each time 3.5 or 4 cc. was given, having as many as 20 seizures without an increase in dosage. This would perhaps imply that instead of a high convulsive susceptibility this patient had a tendency, once the convulsive threshold was reached, to react anomalously.

The possibility of such unforeseen anomalous reactions leads the writers to suggest that not only barbiturates for intravenous administration, chloroform, etc., be kept at hand as advised,[27, 28] but that oxygen be ready at the bedside for immediate use. Since some patients after the metrazol seizure have difficulty in regaining respiration the possibility that barbiturates will further depress the respiratory center is to be borne in mind. Oxygen therapy would under such circumstances be a logical measure. This has been mentioned under the subject of insulin methods but not emphasized in the literature on metrazol methods. In cases where protracted hypertonus with apnea develops, mouth to mouth insufflation of the lungs should be attempted early if the thorax is too rigid for artificial respiration to be given by the usual methods.

This patient's seizures were always predominantly clonic unlike many other patients [17] whose seizures consisted almost entirely of tonic manifestations. It is surprising therefore that this patient and not others developed the second seizure consisting entirely of tonus.

CONCLUSIONS.

1. Various motor phenomena bearing little resemblance to the typical grand mal epileptic seizure or to the usual description of the seizure induced by metrazol occasionally occur.

2. The possible dangers from protracted tonic reactions secondary to the usual seizures should be kept in mind. The threat of persistent anoxia may under such circumstances become ominous.

3. When metrazol therapy is being used every available means should be at hand to combat such developments.

4. All anomalous reactions which suggest yet unrecognized complications of metrazol should be reported in detail. Warnings con-

cerning the possibility of such dangers have already been sounded.[48] The practice of repeating the metrazol injection within a few minutes, though apparently safe in most cases, is not recommended.

BIBLIOGRAPHY.

1. v. Meduna, L.: Die Konvulsionstherapie der Schizophrenie. Psych. Neurol. Wchsch., 37: 317, July 1935.
2. Winkelmann, N. W.: Metrazol treatment in schizophrenia. Am. J. Psychiat., 95: 303-316, Sept. 1938.
3. Goldstein, H. H., Dombrowski, E. F., Edlin, J. V., Bay, A. P., McCorry, C. L., and Weinberg, J.: Treatment of psychoses with pentamethylenetetrazol. Am. J. Psychiat., 94: 1347-1354, Sept. 1938.
4. Friedman, E.: The irritative treatment of schizophrenia. Am. J. Psychiat., 94: 355-372, Sept. 1937.
5. Finkelman, I., Steinberg, L., and Liebert, E.: The treatment of schizophrenia with metrazol by production of convulsions. J. A. M. A., 110: 706-709, Mar. 1938.
6. Low, A. A., Sonenthal, I. R., Blaurock, M. F., Kaplan, M., and Sherman, I.: Metrazol shock treatment of the "functional" psychoses. Arch. Neur. and Psychiat., 39: 717-736.
7. Reese, H. H., Vander Meer, A. H., and Wedge, A. H.: The effect of induced metrazol convulsions on schizophrenic patients. J. Nerv. and Ment. Dis., 87: 570-584, May 1938.
8. Cohen, Louis: The early effects of metrazol therapy in chronic psychotic over-activity. Am. J. Psychiat., 95: 327-333, Sept. 1938.
9. Nightingale, G. S.: Six months' experience with cardiazol therapy. J. Ment. Sc. 84: 574-580, May-July 1938.
10. Briner, O.: Results of the convulsion therapy in the psychiatric clinic in Berne. Am. J. Psychiat., Vol. 94 (Supplement): 155-172.
11. Barbato, L.: Metrazol therapy of schizophrenia. Texas State J. of Med., 34: 220-227, July 1938.
12. Dick, A., and McAdam, W.: Cardiac complications in cardiazol treatment: Observations in four cases. J. Ment. Sc., 84: 999-1001.
13. Kennedy, A.: Convulsion therapy in schizophrenia. J. Ment. Sc., 83: 609-629, 1937.
14. Hayman, M., and Brody, M. W.: Metrazol therapy in schizophrenia. J. A. M. A., 112: 310-311, Jan. 28, 1939.
15. McAdoo, H. W.: Metrazol convulsive phenomena in dementia præcox. J. A. M. A., 110: 528, Feb. 12, 1938.
16. Robards, E. M.: In discussion of Phillips, S. J.: Observations on insulin and metrazol therapy at Central Louisiana State Hospital. New Orleans Med. and Surg. Jour., 91: 135.
17. Bennett, A. E.: Convulsive (pentamethylenetetrazol) shock therapy in depressive psychoses.
18. v. Meduna, L.: New methods of medical treatment of schizophrenia. Arch. Neurol. and Psychia., 35: 361-363.

19. Reese, H. H., and Southoff, A.: Insulin and metrazol treatment in schizophrenia. Wisconsin Med. Jour., 37: 816-820, Sept. 1938.
20. Matthews, R. A.: An interesting result with metrazol. Arch. Neur. and Psychiat., 39: 862.
21. Wilson, D. C.: The treatment of schizophrenia by means of convulsions produced by the intravenous injection of metrazol. J. of Southern Med. and Surg., 100: 359-362, Aug. 1938.
22. Finiefs, L. A.: Induced epileptiform attacks as a treatment of schizophrenia. Lancet, 2: 131, July 17, 1937.
23. Gillies, H.: Convulsive therapy in schizophrenia. Lancet, 2: 131, July 17, 1937.
24. Bookhammer, R. S., and Saxe, E. J.: Preliminary report on metrazol therapy for the psychoses. Arch. Neur. and Psychiat., 39: 864.
25. Long, R. H.: Metrazol treatment in eight cases of mental disease. Jour. Southern Med. and Surg., 100: 363-366.
26. Owensby, N. M.: Convulsive therapy in mental disorders. Southern Med. Jour., 31: 1164-1166.
27. Phillips, S. J.: Observations on insulin and metrazol therapy at Central Louisiana State Hospital. New Orleans Med. and Surg. Jour., 91: 135-144.
28. Watters, T. A., Goldsmith, G. A., and Golden, L.: The treatment of schizophrenic reactions with metrazol. New Orleans Med. and Surg. Jour., 90: 601-604.
29. Goldsmith, G. A.: The use of metrazol in the treatment of schizophrenic reactions. New Orleans Med. and Surg. Jour., 90: 748-750.
30. Dennyssen J. A. F., and Watterson, D. J.: On the mechanism of the cardiazol convulsion. J. Ment. Sc., 84: 1002-1007.
31. v. Meduna, L.: The significance of the convulsive reaction during the insulin and the cardiazol therapy of schizophrenia. J. Nerv. and Ment. Dis., 87: 133-139.
32. Blair, Donald: Neurological manifestations seen during cardiazol and insulin treatments. J. Ment. Sc., 84: 776-780.
33. Sakel, M.: Significance of the epileptic convulsion as a therapeutic factor in the pharmacological shock therapy of schizophrenia. J. Nerv. and Ment. Dis., 87: 140-155.
34. Cohen, Louis H.: Observations on the convulsive treatment of schizophrenia with metrazol. New England J. of Med., 218: 1002-1007.
35. Maurer, S., Wiles, H. O., Marberg, C. M., Skorodin, B., and Fisher, M. L.: Blood chemical changes occurring in the treatment of psychogenic mental disorders by metrazol convulsions. Am. J. Psychiat., 94: 1355-1361.
36. v. Meduna, L.: General discussion of the cardiazol therapy. Am. J. Psychiat., Vol. 94 (supplement): 40-50.
37. Pfister, H. O.: Disturbances of the autonomic nervous system in schizophrenia and their relations to the insulin, cardiazol and sleep treatments. Am. J. Psychiat., Vol. 94 (supplement): 109-118.

38. Cook, C. L.: The range of mental reaction states influenced by cardiazol convulsions. J. Ment. Sc., 84: 664-667.
39. Russell, L. W.: Insulin and cardiazol: Experiences with the combined method. J. Ment. Sc., 84: 672-676.
40. Harris, Arthur: Cardiazol treatment of schizophrenia. J. Ment. Sc., 84: 735-767.
41. Cook, L. C., and Ogden, W.: Cardiazol convulsion therapy in non-schizophrenic reaction states. Lancet, 2: 885-887.
42. Pagniez: Le traitement de la schizophrenie par les crises d'épilepsie provquées, par le cardiazol. La Presse Medicale, 29: 551-553, April 9, 1938.
43. Western, F. B.: Personal communication, Jan. 11, 1939.
44. Cleckley, H. M., Bowles, L., and Mettler, F. A.: Histological changes following metrazol convulsions. (In Press).
45. Walk, A., and Mayer-Gross, W.: Observations on convulsive therapy with triazol 156. J. Ment. Sc., 84: 637-663.
46. de Morsier, G., and Bersot, H.: Cerebral disorders in provoked hyperinsulinemia. Am. J. Psychiat., Vol. 94 (supplement): 143-152.
47. Authors' unpublished observations.
48. Cobb, Stanley: Review of neuropsychiatry for 1938. Archiv. Int. Med., 62: 883-899, Nov. 1938.

METRAZOL SHOCK TREATMENT.

Pharmacological and Biochemical Studies.*

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The study of induced convulsions, both clinically and pathologically, has been made possible for the first time by metrazol shock therapy of psychotic patients. Comparison of the metrazol convulsion with the true epileptic attack raises many questions, the answers to which are important in that they will enable us to have a better understanding of the therapeutic effect of metrazol on the schizophrenic process and they may make it necessary to revise the biologic concept of epilepsy.

The object of the present investigation was to study the mechanism and localization of metrazol convulsions in man and to determine if metrazol convulsive therapy, as originally outlined, could be modified to obtain even better results. The following data were obtained from 15 patients and were collected before, during and after the convulsion.

There is little information in the literature on metrazol convulsive therapy as to its effect on various metabolic processes. Georgi [1] observed a consistent variation in the serum dextrose, serum calcium and potassium levels during convulsions. Sorger and Hofmann [2] made similar observations on the blood dextrose level, and von Meduna [3] studied the changes in the level of urinary chloride and phosphate before and after the attack. Speaking before the Association of Norwegian Psychiatrists, Jahn gave additional data on the metabolism of Na, K, Ca, P, Cl ions, urea and blood dextrose; he assumed that these changes were due to an increase in the secretion of adrenalin. A pharmacologic study of the effect of metrazol in convulsive doses in man has not yet been reported, although the effect of such doses in animals is voluminous. The literature on this subject was recently surveyed by Hildebrandt.[4] Schoen [5] made the original studies on the effect of metrazol on the brain and its antagonism to barbituric acid compounds. He

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assumed that metrazol convulsions had their origin in the midbrain because convulsions were produced in decorticized rabbits. Later, similar investigations were reported by Tartler,[6] Mehl,[7] Blume,[8] Kohn and Jacobi [9] and others. The latter investigators found a greater antagonism between metrazol and chloral hydrate or paraldehyde than the barbiturates. They believe that metrazol is an excitant of the brain stem. Similar results were reported by Kokoschka who agreed with Kohn and Jacobi [9] that such observations confirm the Pick-Molitor theory,[10] in which hypnotics are divided into two groups; one type exerts its main action on the brain stem (barbituric acid compounds, etc.) and the other on the cortex (alcohol, aldehydes, etc.). This theory has also been corroborated by the experiments of Lampel and Feitelberg [11] who studied the antagonistic action of metrazol to phenobarbital by the electrothermic determination of the site of action.

EXPERIMENTS.

In order to eliminate any possible antagonism between the ions which occur normally in the blood serum, the serum Ca, K, Na and Cl, as well as serum dextrose, were determined before, during and after the convulsion. In addition, the blood sedimentation rate was determined to help throw light on possible changes in the plasma proteins.

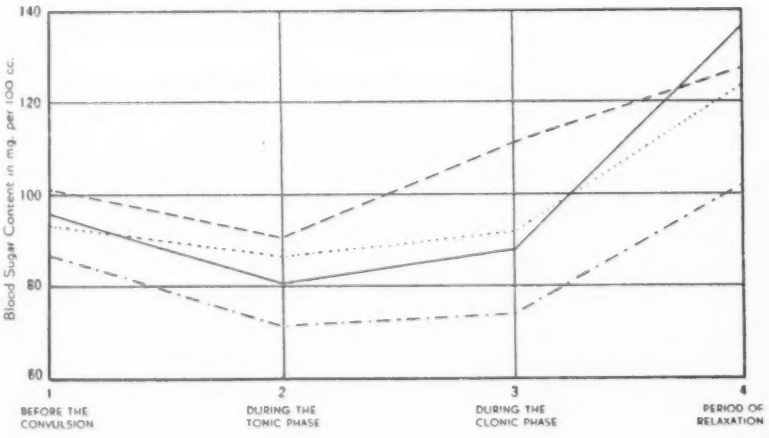
The pharmacodynamic influence of carbon dioxide, strychnine, ephedrine, pituitrin and thyroxin on the one hand and phenobarbital, sodium barbital, paraldehyde, the bromides and scopolamine on the other hand was also studied during metrazol convulsions in both man and animals.

The blood for the tests in Table I was withdrawn from the cubital vein immediately before and directly after the cessation of the clonic spasm while the patient was still in deep coma. An alteration in Ca and K values was not observed, but serum sodium chloride was diminished in the post-paroxysmal phase in most of the patients. It was not possible to determine any definite change in the serum dextrose level from two blood samples; in order to obtain a more accurate picture of changes in blood dextrose, four samples were taken: during the convulsion, during the tonic phase, during the clonic phase, and during the relaxation period after the convulsion. These results are presented in Graph 1.

TABLE I.

SERUM CONTENT OF Ca, K, NaCl, AND DEXTROSE IN MG./100 GM. BEFORE AND AFTER METRAZOL CONVULSIONS.

	Ca.		K.		NaCl.		Blood sugar.	
Patient.	Before metrazol.	After metrazol.	Before metrazol.	After metrazol.	Before metrazol.	After metrazol.	Before metrazol.	After metrazol.
Tu	10.4	10.7	....	....	807	789	...	...
El	9.4	10.5	21.6	21.8	778	690	...	...
Sn	14.0	14.0	25.3	25.8	655	631	...	...
Be	11.6	11.8	23.7	24.2	678	754	107	104
We	10.0	10.0	20.0	20.5	679	714	85	114
De	11.6	11.8	23.7	24.2	...	...	95	88
We	11.0	13.0	18.8	19.7	702	748	113	114
He	10.0	11.0	23.8	24.0	672	748	85	102
Gi	9.5	11.6	21.4	21.8	708	667	94	103
Ber	....	....	18.8	19.0	742	703	...	...
Re	9.0	12.0	19.0	18.6	703	748	...	...
Ku	9.9	9.8	16.1	16.0	707	690	...	...
Fa	11.1	11.0	19.7	21.0	737	713	...	...



GRAPH 1.

In eight patients, the blood sedimentation time, as determined by the Westergren Method, was slower. These results are given in Table II.

Metrazol was given intravenously except in a few cases. In the latter instances, intramuscular injection did not produce a convulsion even when a very large dose was used, but did cause a state of excitement which obviously blocked any therapeutic benefit. Injection into the jugular and femoral veins always produced a convulsion. According to von Meduna, metrazol should be injected rapidly at an optimum speed of 1 cc. per second. More rapid injection is not required to produce seizure and smaller doses cannot be used when injections are made more rapidly; there is also danger of injuring the vein. Metrazol 0.3 gm. was the smallest dose which caused a convulsion; the largest amount required was 2.5 gm. There was no difference in the seizure produced by the minimum or maximum doses. In general a course of metrazol treatment was started with 0.5 gm., which was usually sufficient to cause a seizure. If this dose did not produce a convulsion, it was increased by 0.1 to 0.2 gm., repeated in about 30 seconds. If necessary, a third dose, increased by the same amount was administered. It is desirable to avoid so-called psychic equivalents, furor or cloudy states. As the course of treatment progressed, it was necessary to increase the initial dose anywhere from 30 to 100 per cent. No sensitization to metrazol was observed. Ordinarily there was a latent period of 2 to 45 seconds between the injection and the beginning of the convulsion; the seizure lasted from 20 to 70 seconds, the tonic

TABLE II.

BLOOD SEDIMENTATION RATE IN METRAZOL CONVULSIONS.

(Westergren's method—results given in mm. after one hour.)

Patient.	Before the convulsion.	After the convulsion.
De	17	15
Re	7	3
Ber	10	5
Fa	25	19
Tu	15	9
Ku	14	8
Su	42	32
Be	11	8

phase taking up about one-third and the clonic phase two-thirds of the total time. The size of the dose and the speed of the injection apparently had no influence on the length of the latent period or the duration of the convulsion.

The idea for modifying the metrazol technique originated in the observation that it was necessary to give larger doses to produce convulsions in patients to whom barbituric acid compounds had been administered. In some of these patients, very large doses of metrazol did not produce convulsions. Similar observations have also been made in animal experiments. The effect of various medicaments on the sensitivity of patients to metrazol is summarized in Table III.

In combined insulin and metrazol treatment, the convulsive dose of metrazol was reduced 20 to 35 per cent when metrazol was given during the period of hypoglycemia.

In addition to these observations on the variation of human subjects to convulsive doses of metrazol when given in conjunction with other drugs, similar experiments were made on animals. For most of these experiments white rats were used, but in a few cases rabbits were used. Moderately sized rats with moderate convulsive reactivity were chosen. The minimal convulsive dose of metrazol was found to be between 4 and 7.5 mg./100 gm. rat weight. Lethal doses were over 8 mg./100 gm. Adrenalin in doses of 0.0001 gm. did not increase the sensitivity of the animal to metrazol. This was also the case with pituitrin when given in doses of 0.025 Voegtlin. The dose of metrazol required to produce a convulsion after strychnine nitrate 0.05 mg./gm. was reduced over 25 per cent. After caffeine sodio-benzoate 0.005 mg./gm., the minimal convulsive dose of metrazol was reduced to 30 per cent of that previously determined. After phenobarbital 0.1 to 0.2 mg./gm., the minimal convulsive dose of metrazol was doubled. The inhibitory action of rutonal was even more pronounced; experiments with this compound will be continued by Pisk and Redlich. The inhibitory action of paraldehyde was weaker than that of the barbituric acid compounds.

The effect of thyroxin on the convulsive dose of metrazol has been summarized in Table IV.

After the minimal convulsive dose of metrazol had been established, thyroxin 0.3 mg. per day was administered for five days and the minimal convulsive dose was again determined.

TABLE III.

Convulsive Doses of Metrazol Before and After Other Therapeutic Agents.

(Doses given in grams.)

	Patients.											
	Be.	Re.	We.	He.	De.	Fa.	Ku.	Wer.	Tu.	Ber.	Gi.	Ka.
Before CO_2	0.5	0.4	0.45	0.55	0.5	0.5	0.5	0.9	0.6	...	....	....
After CO_2 [1]	0.3	0.3	0.3	0.4	0.3	0.3	0.4	0.5	0.3	...	....	....
Before adrenalin	0.5	0.4	0.55	0.55	0.5	0.55	0.7	0.7	0.6	1.0	0.7	....
After adrenalin [2]	0.5	0.35	0.3	0.4	0.45	0.4	0.5	0.5	0.55	0.8	0.55	....
Before ephedrine	....	0.45	....	....	....	0.5	....	0.7	....	...	....	0.55
After ephedrine [3]	....	0.3	....	....	....	0.3	....	0.4	....	...	....	0.35
Before pituitrin	0.5	0.4	0.55	0.55	0.5	0.5	0.6	...	0.6	1.0	0.7	....
After pituitrin [4]	0.4	0.4	0.45	0.5	0.45	0.45	0.55	...	0.6	0.9	0.6	....
Before strychnine	0.5	0.45	0.7	0.55	0.55	0.5	0.5	...	....	1.0	0.7	....
After strychnine [5]	0.45	0.35	0.65	0.5	0.5	0.4	0.6	...	....	0.9	0.6	....
Before theophylline	0.5	0.45	0.7	0.6	0.6	0.5	....	...	0.6	1.0	0.7	....
After theophylline [6]	0.5	0.45	0.7	0.6	0.6	0.55	....	...	0.55	1.0	0.65	....
Before caffeine	0.5	0.45	....	0.6	....	....	....	...	....	1.0	0.7	....
After caffeine [7]	0.5	0.45	....	0.75	....	....	....	...	....	0.9	0.65	....
Before phenobarbital	....	....	....	0.5	0.5	0.5	....	0.9	....	1.0	....	....
After phenobarbital [8]	....	....	....	0.8	0.75	0.8	....	1.4	....	1.3	....	....
Before paraldehyde	....	....	....	0.55	....	0.5	....	0.9	....	0.9	....	....
After paraldehyde [9]	....	....	....	0.65	....	0.6	....	0.9	....	0.9	....	....
Before sodium bromide	0.5	0.5	....	....	0.6	....	....	0.7	....	1.0	....	....
After sodium bromide [10]	0.5	0.5	....	....	0.6	....	....	0.8	....	0.9	....	....
Before hyoscine	....	0.4	....	....	....	....	....	0.9	....	...	0.7	....
After hyoscine [11]	....	0.6	....	....	....	....	....	1.2	....	...	0.9	....

[1] Patients inhaled carbon dioxide for one minute causing about 100 per cent increase in the respiratory rate, after which metrazol was injected.

[2] 15 minutes after the injection of ¾ mg. adrenalin, at the height of its effect, metrazol was injected.

[3] Ephedrine sulphate 0.6 gm. was given hypodermically followed by metrazol ¼ of an hour later.

[4] 2 cc. pituitrin were given intramuscularly followed in ½ hour by metrazol.

[5] 5 mg. strychnine nitrate were injected subcutaneously and followed in ½ hour by metrazol.

[6] Theophylline with ethylene diamine 0.24 gm. was administered intravenously and followed in 10 minutes by metrazol. These patients vomited regularly after convulsions.

[7] Caffeine sodium benzoate 0.75 gm. was injected hypodermically and followed in 15 minutes by metrazol.

[8] Phenobarbital 0.22 gm. was injected during the early morning hours, followed in two to four hours by metrazol. When phenobarbital was given the evening before, it was usually not possible to obtain convulsions.

[9] Paraldehyde 4 to 7 gm. was injected four to five hours before metrazol.

[10] Sodium bromide 3 gm. a day was given for three days; on the fourth day 3 gm. were given two hours before the metrazol.

[11] Hyoscine 0.5 mg. was injected ½ hour before metrazol. When metrazol was given a few hours later, the effect of hyoscine was not well defined.

TABLE IV.

EFFECT OF PRELIMINARY TREATMENT WITH THYROXIN ON MINIMAL CONVULSIVE DOSE OF METRAZOL IN THE RAT.

Rat.	Minimal convulsive dose (M. C. D.) of metrazol.	M. C. D. after preliminary treatment with thyroxin.
1 C.....	5 mg./100 gm.—few twitches	3 mg./100 gm.—convulsions after 8 minutes
2 C.....	8 mg./100 gm.—few twitches	5 mg./100 gm.—twitches after 15 minutes
3 C.....	9 mg./100 gm.—few twitches	4 mg./100 gm.—twitches after 10 minutes convulsions after 15 minutes
4 C.....	7 mg./100 gm.—few twitches	4 mg./100 gm.—twitches after 7 minutes convulsions after 15 minutes
5 C.....	8.5 mg./100 gm.—few twitches	3 mg./100 gm.—twitches after 10 minutes convulsions after 15 minutes

DISCUSSION.

The present series of experiments corroborates the investigations reported by Hildebrandt [12] and his co-workers; those of Schoen,[5] Mehl,[7] Tartler,[6] Kohn and Jacobi,[9] etc.; and several reports from the Viennese school. The inhibitory action of sodium phenobarbital, sodium barbital and rutonal was found to be stronger than that of paraldehyde or chloral hydrate in animals and a similar antagonistic effect was observed during metrazol convulsions in man. Phenobarbital, sodium barbital and scopolamine increased the tolerance of patients to metrazol to the greatest extent, chloral hydrate and paraldehyde to a lesser extent, and the bromides had no inhibitory effect on the convulsion.

From this work, as well as that by Lampel and Feitelberg,[11] it can be concluded that metrazol acts on the centers of the brain stem. The antagonism which exists between the so-called "brain stem hypnotics" and metrazol increases the evidence tending to prove the validity of the Pick-Molitor theory. A similar antagonistic action was observed in man. It seems strange that the bromides which are of value in epilepsy do not exert any antagonistic effect

the evening before, it was usually not possible to obtain convulsions.
[9] Paraldehyde 4 to 7 gm. was injected four to five hours before metrazol.
[10] Sodium bromide 3 gm. a day was given for three days; on the fourth day 3 gm. were given two hours before the metrazol.
[11] Hyoscine 0.5 mg. was injected ½ hour before metrazol. When metrazol was given a few hours later, the effect of hyoscine was not well defined.

to metrazol. Apparently the bromides do not prevent epileptic attacks by the same mechanism as the paralyzing hypnotics, but act in some complex manner on the metabolic processes, lessening the irritability of the epileptic.

The fact that the convulsive dose of metrazol is reduced during insulin shock tends to show that unconsciousness does not necessarily mean a lowered sensitivity of the nerve centers, similar to that in sleep and in coma induced by hypnotics. Further investigations in this field may give us more information on the physiology and etiology of consciousness.

Analysis of the results obtained by using stimulating drugs in conjunction with metrazol shows that the sensitivity is increased by carbon dioxide, ephedrine and, to some extent, by adrenalin. The central irritating effect of ephedrine was especially marked. When ephedrine was injected at the same time as metrazol, 30 to 40 per cent less metrazol was required to produce a convulsion. Although excitant drugs, such as strychnine and caffeine lessened the minimal convulsive dose of metrazol in animals, they had very little effect on the sensitivity of man to such doses. Theophylline, which increases the permeability of the meninges, had no effect on the convulsive dose. Pituitrin also had no effect on the excitability but thyroxin increased it.

The use of carbon dioxide to interrupt catatonic stupor was first recommended by Loevenhart, Lorenz and Waters.[13] Carbon dioxide increased sensitivity more than any of the other drugs used in this series and its use in conjunction with metrazol markedly reduced the size of the convulsive dose. In some cases when metrazol alone did not produce a convulsion, a seizure was induced by injecting metrazol after the inhalation of carbon dioxide. When metrazol was given hypodermically during light carbon dioxide asphyxia no change in the blood pressure and respiratory rate was observed, showing that this combination apparently did not change the sensitivity of the nerve centers (Raab and Redlich [14]). In contrast to this finding, the administration of carbon dioxide during a metrazol convulsion resulted in an increase of the sensitivity of the nerve centers. This may be due to the different methods by which metrazol is administered, *i. e.*, small doses subcutaneously and large doses injected rapidly intravenously.

Technical difficulties prevented the author from determining the serum albumin fraction and limited this investigation to the determination of the blood sedimentation rate immediately before and after convulsions. The blood sedimentation rate is, *cum grano salis,* more rapid when there is a relative increase in the globulin fraction, slower when the albumin fraction is increased and may thus be used to help estimate this ratio when other factors are constant. In all patients the blood sedimentation rate was slower, so it was assumed that this retarded rate may correspond to an increase in the albumin fraction. Fried and later Fried and Frisch [15] observed an increase of serum albumin during periods of frequent attacks and considered that this change in the protein picture was an important factor in the pathologic physiology of epilepsy, and that it is responsible for the periodicity of attacks. However, the present study indicates that the attacks seem to be the cause for, rather than the result of, such a change. The other metabolic changes observed during metrazol convulsions are irrelevant. The antagonism between the calcium and potassium ions as reported by Georgi [1] was not observed. The slight increase in the level of serum sodium chloride corresponds to the decrease of urinary sodium chloride as observed by von Meduna.[3] The blood dextrose levels as observed corroborate the findings of Georgi [1] and Sorger and Hofmann.[2] Whether or not the changes in the level of serum dextrose are the result of an increase in the output of adrenalin during the tonic or clonic phase as Jahn assumed is still hypothetical. Studies on these factors which have been started by E. B. Tietz may prove whether or not the interesting hypothesis of Jahn is correct.

The bromides or paraldehyde are preferable to hyoscine or barbituric acid compounds for patients who become restless or violent following metrazol. From this experimental work it can be concluded that the minimal convulsive dose of metrazol can be decreased by the use of carbon dioxide or ephedrine in patients refractory to metrazol alone. Neither a high alkali ash diet nor variation of water intake changed the sensitivity of the patient to metrazol. The general stimulating effect of metrazol is not due to primary irritation of vasomotor fibers but to a direct action on the nerve substance. Its main site of action is the brain stem (Schoen,[5] Lampel and Feitelberg,[11] and others). The similar re-

sults obtained with convulsive doses of metrazol in human subjects corroborate the above-mentioned theory. All biologic phenomena following large dosages indicate that the effect of metrazol is on the brain stem. The theory of Angyal [16] that the action of metrazol is due to vasomotor waves affecting the frontal lobe is based on the observation of Stief and Tokay who believe that metrazol is primarily a vasomotor stimulant; metrazol is also especially useful in treating patients in a catatonic stupor accompanied by a frontal syndrome. Both of Angyal's assumptions cannot be definitely proved but we cannot say that these interesting theories have been contradicted in this study. A study by W. Birkmeyer on motor activity during metrazol convulsions points to a similar conclusion. Further studies on the changes of consciousness during metrazol convulsions will be made on both a pharmacologic and neurologic basis. There is some hope that the physiologic studies reported recently by Hall [17] on neurologic and psychologic observations may help to explain the epileptic syndrome and its relation to schizophrenia.

SUMMARY.

This report is concerned with a pharmacologic and metabolic study of metrazol convulsive therapy in human subjects. It was found that the level of serum calcium and potassium remained almost unchanged before and after convulsive seizures. The characteristic increase in blood sugar may be due to an increased output of adrenalin. Serum sodium chloride was also slightly increased. The blood sedimentation rate was retarded after convulsions, and is interpreted as reflecting an increase of serum albumin. This change is discussed in its relation to the theories of protein metabolism changes in epilepsy (Frisch [15]).

Central nervous system stimulants such as carbon dioxide and ephedrine decreased the minimal convulsive dose of metrazol. Adrenalin and pituitrin were less effective in this regard. Strychnine, caffeine and theophylline had no such effect. Hypnotic and narcotic drugs—phenobarbital, sodium barbital and rutonal—exhibited a marked antagonistic action to metrazol while this effect was less with paraldehyde and chloral hydrate. Sodium bromide also has no antagonistic action. All of these observations tend to

corroborate the Pick-Molitor theory of the action of hypnotics. The dose of metrazol required to produce a convulsion during insulin hypoglycemic shock is much smaller which tends to show that unconsciousness in insulin shock is pharmacologically and neurologically different from the coma induced by hypnotics and narcotics.

In the human subject the results obtained were similar to those found in animal experiments. Previous administration of thyroxin to rats caused a decrease in the minimal convulsive dose of metrazol.

Metrazol convulsions in man are produced by an intense stimulation of the vegetative and motor centers of the brain stem.

BIBLIOGRAPHY.

1. Georgi, F.: The problem of convulsions and insulin therapy. Am. J. Psychiat., 94: 67 (May), 1938.
2. Sorger, E., and Hoffmann, E.: Beobachtungen und Ergebnisse bei der Cardiazol-Krampfbehandlung der Schizophrenie. Psych.-neurol. Wschr., 39: 462-65 (Oct. 9), 1937; 39: 473-78 (Oct. 16), 1937.
3. von Meduna, L.: Die Konvulsionstherapie der Schizophrenie. Carl Marhold, Verlagsbuchhandlung, Halle a. S., 1937.
4. Hildebrandt, F.: Pentamethylentetrazol (Cardiazol). Handbuch der Experimentellen Pharmakologie, 5: 151-183, 1937, edited by A. Heffter. Publ.: Julius Springer, Berlin.
5. Schoen, O.: Die antagonistische Beeinflussung der Narkose. Arch. f. exp. Path. u. Pharm., 113: 275, 1926.
6. Tartler, O. P.: Ueber den Antagonismus und Synergismus zwischen einigen Analepticis und Medinal. Arch. f. exper. Path. u. Pharm., 143: 160, 1929.
7. Mehl, W.: Addition der Wirkung von einigen Analepticis zu Medinal. Arch. f. exp. Path. u. Pharm., 151: 41, 1930.
8. Blume, W.: Ueber die Wirkung des Kampfers und einiger Kampferersatz-praparate auf das Rueckenmark der dekapitierten Katze. Arch. f. exp. Path. u. Pharm., 116: 234, 1926.
9. Kohn, R., and Jacobi, M.: Untersuchungen ueber qualitative und quantitative Beziehungen zwischen Schlafmitteln und Analepticis. Arch. exper. Path. u. Pharm., 179: 449.
10. a-Pick and Molitor: Hirnrinden- und Hirnstammschlafmittel. Arch. f. exp. Path., 115: 318, 1926.
    b-Pick: Ueber den Schlaf. Kli. Wochenschrift, 43: 1481, 1937.
11. Lampel and Feitelberg: Arch. f. exp. Path. (in print).
12. Hildebrandt, F.: Pharmakologie der Kreislaufanaleptika. Arch. f. exper. Path. u. Pharm., 181: 89, 1936.

13. Loevenhart, A. S.; Lorenz, W. F.; and Waters, R. M.: Cerebral stimulation. J. A. M. A., 92: 880, 1929.
14. Raab and Redlich: Quantitative Untersuchungen ueber Erregbarkeit des Vasomotorenzentrems. Zeitschrift f. Klin. Med., 129: 455, 1936.
15. Fried and Frisch: Serumeiweissbild als fuehrendes Prinzip therapeutischer Versuche bei Epilepsie. Zeitschrift f. exp. Med., 256: 756, 1927.
16. von Angyal, L.: Theoretical aspects of insulin schock therapy and metrazol convulsive therapy of schizophrenia. Monatschr. f. Psychiat. u. Neurol., 97: 280 (Dec.), 1937.
17. Hall, G. E.: Physiological studies in experimental insulin and metrazol shock. Am. J. Psychiat., 95: 553 (Nov.), 1938.

NEW YORK WIPES OUT LUNACY COMMISSIONS.

By STATE SENATOR THOMAS C. DESMOND,
Chairman, New York State Senate Committee on Affairs of Cities.

A decisive victory this year ended the long struggle in New York State to abolish lunacy commissions and to transfer their functions to psychiatrists in public hospitals.

A new law, which I sponsored at the 1939 legislative session (1) eliminates lunacy commissions; (2) substitutes examinations by psychiatrists in public hospitals; (3) restricts psychiatrists' examination to determination of defendant's sanity at time of trial; and (4) restates completely and makes flexible the provisions regarding sanity examination procedure in minor courts.

Abolishes Lunacy Commissions.

The most significant contribution of this new statute, which becomes effective September 1, 1939, to the determination of sanity is that it abolishes lunacy commissions.

Shocking scandals, principally in New York City, have been associated with lunacy commissions. Psychiatrists are well aware of the notorious political abuses, the unnecessary waste of taxpayers' money, and the unreliable sanity examinations which occurred under the lunacy commission system.

Under this system, the courts were authorized to appoint a three-member advisory committee consisting of at least one qualified psychiatrist and one lawyer. The third appointee was usually a layman.

Defying public opinion, judges appointed their relatives, political district leaders and followers to lucrative positions on these commissions. Appointments to lunacy commissions were party patronage.

Extravagance was another evil connected with lunacy commissions. Judges went out of their way to order examinations. From 1930 to 1938, inclusive, New York City spent $1,734,899 on 7031 lunacy commission appointments, as shown by Table I.

No appointments were made in Queens during this period because judges there sensibly sent prisoners to the psychiatric division of Bellevue Hospital.

New York City spent approximately $200,000 a year for lunacy commissions. Under the new Desmond law the cost will be about $50,000, a saving of $150,000 a year.

Psychiatrists on these lunacy commissions labored under difficult conditions, being out-numbered by non-medical men, by laymen ignorant of fundamental scientific facts regarding insanity, and by lawyers whose legalistic jargon frequently confused rather than clarified. As a result, the findings of lunacy commissions were in

TABLE I.

LUNACY COMMISSION EXPENDITURES AND APPOINTMENTS IN NEW YORK CITY FROM 1930-1938 INCLUSIVE.

County.	Expenditures.	Members.
Kings	$1,093,900	4264
New York	456,339	2038
Bronx	182,560	721
Richmond	2,100	8
Queens	0	0

many cases repudiated by the courts that appointed them. The presence of laymen on lunacy commissions was a clear example of the tendency of legislation to lag behind scientific developments.

SUBSTITUTES EXAMINATIONS BY PSYCHIATRISTS.

To replace the lunacy commission system, the new Desmond law establishes a scientifically sound procedure whereby the medical question of the sanity of a defendant at time of trial is determined solely by qualified psychiatrists.

The new statute provides that when it appears to the court that there is reasonable ground for believing a person indicted for felony or misdemeanor is in such state of idiocy, imbecility or insanity that he is incapable of understanding the proceedings or of making his defense, or if the defendant pleads insanity, the court may order the defendant to be examined to determine his sanity.

In New York City, upon request of the court, the director of the division of psychiatry in the City Department of Hospitals

is required to cause an examination to be made by two qualified psychiatrists, designated from the staff of the division. The director may be one of these psychiatrists.

Outside of New York City, because staff psychiatrists are not available in many parts of the state, the procedure is slightly different. Upstate, the superintendent of a hospital supported by the state or a political subdivision thereof, having a psychiatric service certified by the state commissioner of mental hygiene as having adequate facilities, is required to have the examination made. The superintendent must select from the hospital staff two qualified psychiatrists, of whom he may be one, to make the examination. If such qualified staff psychiatrists are not available, the superintendent is authorized to designate any qualified psychiatrists in the state.

Examinations may be made either in the jail or hospital. Psychiatrists are given the power of subpœna and are authorized to examine witnesses and receive such other information as may aid them in reaching a determination. Psychiatrists designated are required to take the oath prescribed for referees.

As a compromise with lawyers both within and without the legislature, the new law provides that in New York City the psychiatrists "shall be aided by an assistant corporation assigned for that purpose by the corporation counsel of such city." The assistant corporation counsel will not pass on the sanity of a defendant. He will provide such legal advice to the psychiatrists as they may require. This will entail merely an extension of the work now performed by the New York City corporation counsel with regard to questions of sanity in non-criminal cases.

Upon completing the examination, the hospital superintendent, or, in New York City, the director of the division of psychiatry, must give the court a full and complete report including the findings of the qualified psychiatrists who have conducted the examination to the effect that the defendant is or is not, at the time examination is made, in such a mental state as to be incapable of understanding the proceedings or of making his defense. The report must include a recommendation as to the appropriate institution to which the defendant should be sent, if committed.

If the psychiatrists find the defendant able to understand the proceedings, and the court concurs, the action against the defendant

is resumed as if no examination had been ordered. On the other hand, if the court does not concur with the findings of the psychiatrists, or if the two psychiatrists do not agree in their findings, either the action against the defendant may be resumed or the court may request appointment of a third psychiatrist.

A helpful provision of the Desmond law requires that a duplicate copy of the psychiatrists' report must be transmitted by the court clerk to the superintendent of the institution to which the defendant is committed. This should prove of value in treatment of the defendant.

The report of the psychiatrists must not be received in evidence at the defendant's trial but must be filed by the court in the office of the court clerk where it will be subject to inspection only on order of the judge.

Should the psychiatrists certify that the defendant is unable to understand the proceedings, and the court concurs in the finding, it must suspend trial until the defendant becomes sane. Opportunity, however, must first be given to the District Attorney and the defendant's counsel to be heard, before the court makes its decision. Following suspension of the trial, the court must commit the defendant to a state hospital for the insane under the jurisdiction either of the state department of correction or of the department of mental hygiene.

The new law permits transfers of the defendant during the period of his commitment to any state hospital for the insane whether under control of the department of correction or department of mental hygiene, upon approval of the respective department heads.

As in the old law, a defendant thus committed must remain in the institution to which he is sent (or transferred) until the superintendent certifies to the court that he has recovered and is able to understand the proceedings and make his defense.

In New York City, psychiatrists will receive no fees, for the work will be performed by the regular staff of the city hospital department. Outside New York City, psychiatrists are to be paid reasonable traveling expenses and a fee of not more than $50 in any case. These costs are made a county charge.

Provides for Determination of Sanity Only at Time of Trial.

The Desmond law purposely deals only with the medical question as to whether or not the defendant is able to stand trial. It does not require the psychiatrist to determine the mental condition of a defendant at time of commission of the crime.

No attempt is made in this new law to restate or redefine the legal concept of the mental responsibility of a defendant at the time the crime was committed, nor is any attempt made in this law to reconcile conflicting views of the legal and medical professions as to what constitutes "insanity."

The new law carries out my belief that the question of a defendant's ability to stand trial is a medical question which can properly be decided by psychiatrists, but that the question of a defendant's responsibility for the crime which he committed is a legal question which must be decided by the jury on the basis of testimony and cross-examination of witnesses.

The famous McNaughten rule, laid down in England in 1843 by leading judicial authorities, correctly set forth the considerations involved in this question when it stated that "a medical man under the circumstances supposed cannot in strictness be asked his opinion on this point because each of those questions involves the determination of the truth of the facts deposed to, which is for the jury to decide, and the questions are not questions upon a mere matter of science, in which case such evidence is admissible."

Psychiatrists should welcome the fact that they will be restricted in their determination only to the question of the ability of a defendant to understand the proceedings and make his defense. This will not only reduce and simplify their work, but also tend to give their findings greater acceptance by the general public.

Restates and Makes Flexible Procedure in Minor Courts.

The new statute completely restates and makes flexible provisions regarding sanity examinations in the minor courts.

If a defendant is charged with an offense which is not a crime (generally some form of disorderly conduct) and the qualified psychiatrists who have examined him report that though he is in such a state of idiocy, imbecility or insanity as to be incapable

of understanding the charge against him or of making his defense, they deem his discharge "not dangerous to the public peace and safety," the court may in its discretion suspend proceedings and release the defendant on bail or parole him on such terms as in either case, will provide for periodic receipt of information by the court as to the mental condition of the defendant.

When it appears to the court that the defendant is no longer in such mental state as to be incapable of understanding the charge against him or of making his defense, it must have the defendant brought into custody and proceedings against him must be resumed.

Should the psychiatrists, in cases of offenses which are not crimes, report that the defendant is incapable of understanding the charge against him or of making his defense and that they believe his discharge dangerous to public peace and safety, the court, instead of committing him to a state hospital for the insane, may direct that proceeding be taken to have him committed to a state hospital or a state school under jurisdiction of the department of mental hygiene where he shall remain until he can understand the proceedings.

Conclusion.

Of tremendous assistance in the difficult campaign of serving adoption of this reform measure were the New York Psychiatric Society; New York Neurological Society; New York State mental hygiene commissioner, Dr. William J. Tiffany; and his predecessor in that office, Dr. Frederic W. Parsons; Mr. Lawrence Veiller, president of the Citizens' Crime Commission of New York State; Miss Elsie M. Bond, assistant secretary of the State Charities' Aid Association, and newspapers throughout the state.

The social aim of this type of legislation is to assure a scientifically accurate examination of sanity in the most expeditious manner at the least cost to the taxpayer. The new statute attempts to meet this standard in every respect.

Although it does not solve numerous complex problems relating to psychiatry and criminal law, the new Desmond law does mark a tremendous step forward. To the public, the new statute is important because it smashes the lunacy commission "racket," removing a huge source of political patronage, and cutting the cost

of sanity examinations. To the accused, the new law assures a scientific procedure for it turns over to psychiatrists the medical problem of determining sanity at time of trial. To the psychiatrist, it means an opportunity to determine the sanity of defendants unhampered by political appointees, lawyers and laymen—an opportunity to serve the public more efficiently and more effectively.

PSYCHIATRIC NURSING EDUCATION.

Recommended Standards and Curricula for Undergraduate General Nursing Courses in Psychiatric Hospitals, Affiliate and Post-Graduate Courses in Psychiatric Nursing.

The Committee on Psychiatric Nursing has prepared outlines of standards and curricula for courses in psychiatric nursing, which have been approved by Council.

The standards and curriculum for the undergraduate general nursing course represent the first major revision since standards were set by The American Psychiatric Association in 1923. The National League of Nursing Education has co-operated in the preparation of these outlines as well as for the affiliate and post-graduate outlines.

Council has authorized the issuance of certificates of approval to courses which equal or exceed the standards recommended in these outlines. Applications for certificates of approval should be sent to the office of The American Psychiatric Association, 2 East 103rd Street, New York City, accompanied by a detailed description of the courses offered or proposed to be offered, including a list of personnel and facilities. Printed copies of these outlines are available on request.

THE FOLLOWING IS A SUGGESTED CURRICULUM FOR UNDERGRADUATE NURSES AFFILIATING WITH A MENTAL HOSPITAL FOR SPECIAL TRAINING IN PSYCHIATRY AND PSYCHIATRIC NURSING.

It is assumed that the student is enrolled in a registered school of nursing and that the time of affiliation shall be not sooner than the end of the student's first year and preferably some time during the third year, as experience has shown that a familiarity with medical terminology and technique is a necessary preliminary. It is also assumed that the student has some slight familiarity with psychological terms and psychological concepts.

In the curriculum as outlined below, it should be understood that the suggestions made allow for a certain degree of elasticity and modification. There are certain topics which should be regarded in the nature of essentials

and they will be so nominated. Others are to be regarded as highly desirable if it is possible to meet them in the setting in which the course is given.

1. *Affiliate Course in Psychiatric Nursing (Basic Training).*
   This course is offered as a part of the general nursing curriculum for undergraduate students. It aims to familiarize the student with the causes, symptoms and nursing care of the mentally ill, as well as giving an understanding of the psychological factors in other types of illness and an appreciation of the close relationships between mental and physical.
2. *Time Allotment*—12 to 16 weeks.
   The various assignments are based on the 16 weeks' affiliation. If the affiliation period is less than this, the assignments should be pro-rated downward.
3. *Teaching Personnel.*
   The various subjects should be taught by medical, nursing and other personnel who are well qualified in their fields and have an adequate background in the subjects which they are teaching.
4. *Education Committee.*
   To be responsible for content and conduct of course. To consist of the superintendent or a senior member of the medical staff designated by him, the director of nursing service, the director of education or instructor, and heads of other departments which contribute directly to the course.
5. *Teaching Facilities.*
   The course should be given in a setting which offers nursing experience with as great a variety of psychiatric cases as is possible, such as a mental hospital or the psychiatric division of a general hospital. It is expected that teaching would be done by didactic lectures, clinical demonstrations, formal and informal discussions on the wards, and attendance at medical staff conferences.
6. *Library.*
   A well-stocked library is essential with a varied list of psychiatric and nursing journals.
7. *Student Health.*
   A certificate from the home school that the student has had the usual physical examinations and protective inoculations should be satisfactory.
8. *Living Quarters.*
   A section of nurses' home if available. If satisfactory quarters are not available, students should find their accommodation outside the hospital.
9. *Record System.*
   Complete records as customary in other nursing schools, including health records.
10. *Certificate or Diploma.*
    On satisfactory completion of course, a certificate or diploma to be issued by the hospital or school giving the course.

11. *Approval of Courses by American Psychiatric Association.*

The American Psychiatric Association, through its Committee on Psychiatric Nursing, is prepared to issue certificates of approval for affiliate courses which comply with or exceed the suggested minimum standards here outlined. Any school of nursing desiring such certificate should prepare an outline of courses offered and send it to the Executive Assistant, American Psychiatric Association, 2 East 103rd Street, New York City.

CONTENT OF COURSE.

PSYCHIATRY—Essential.

(a) Didactic lectures—20 hours.
(b) Clinical demonstrations—Essential—12 to 16.
(c) Medical staff conferences—Essential—6.
(d) Child psychiatry—Highly desirable—4 hours.
(e) Psychology and mental hygiene—15 to 20 hours (if not previously taught in home school).
(f) Anatomy of the nervous system—Essential—10 hours.

PSYCHIATRIC NURSING—Essential—20 hours.

The course to be developed along the lines of the following general headings:

(1) The functions of a mental hospital.
(2) General considerations.
(3) The care of special types of patients.
(4) Nursing care in special therapies; *e. g.*, insulin, metrazol, prolonged narcosis and tryparsamide.
(5) Psychotherapy—3 to 6 hours—Essential.
(6) Special facilities of the hospital.

The following are considered highly desirable experiences for affiliate nurses to have, but are not all absolutely essential:

(a) Mental hygiene clinics and community relations. Where these exist in connection with the hospital service, they should be utilized for training purposes.
(b) Occupational, industrial and recreational therapy. When possible the student should spend two weeks in these departments, actually working with patients under the guidance of the departmental staff. Lectures—4-6 hours.
(c) Hydrotherapy—6 hours.
Classroom talks about the use of water and practical demonstrations of continuous baths, packs, local applications, sprays and colonic irrigation.
(d) Physiotherapy—4 hours.
Ultraviolet and infra-red rays, massages, sinusoidal current, diathermy, hyperthermic cabinets, etc.

(e) Social service—1 hour.
The aims, objectives and uses of social service in the mental hospital, the after care and follow-up of patients; treatment of relatives (you can often do a great deal for the relatives when you cannot do much for the patient).

(f) Nursing indications when special techniques are employed, *e. g.*, psychometric examinations, electro-encephalography, pneumo-encephalography, personality inventory, spinal punctures and tube feedings.

(7) Ward assignments (male wards should be used for part of the course).
Admission wards—3 weeks.
Refractory or disturbed wards—3 weeks.
Physically sick and infirm—2 weeks.
Convalescent and privileged—2 weeks.
Continued treatment or chronic—2 weeks.
Special treatment wards—such as insulin, metrazol, hyperpyrexia, etc.—2 weeks.
Occupational, industrial and recreational—2 weeks.

(8) Case studies.
The student, during her affiliation, should be expected to make one or two complete case studies and prepare symptom records, mental status notes and behavior study records. These studies should incorporate significant features in the patient's history, the student's interpretation of these, and the development of the patient's disorder; a detailed analysis of the patient's behavior for one week, the treatment employed, the results accomplished; a general formulation by the student in her own words.

SUGGESTED STANDARDS FOR SCHOOLS OF NURSING OFFERING POST-GRADUATE COURSES IN PSYCHIATRIC NURSING.

1. *Post-Graduate Courses in Psychiatric Nursing.*
These courses require to be diversified as to content and length, depending upon the particular objective. In all of them a part of the objective will necessarily be a broader understanding of the psychological aspects of all forms of illness and the gaining of new techniques for the better nursing of sick people, especially mentally sick people. In addition to these general objectives, there may also be special objectives, with modification of the courses to suit special requirements. It is understood that these outlines represent in a general way the essential minimum teaching and practical experience for various objectives. They may be modified or added to as may be deemed advisable.

2. *Student Qualifications.*
Students to be graduates of an accredited school of nursing and registered in the state from which they graduated.

3. *Education Committee.*
   To be responsible for content and conduct of course. To consist of the superintendent or a senior member of the medical staff designated by him, the director of nursing service, the director of education or instructor, and heads of other departments which contribute directly to the course.
4. *School.*
   May be located in a public or private psychiatric hospital, a general hospital with a special psychiatric division, or a college or university affiliated with a hospital which provides adequate psychiatric facilities.
5. *Faculty.*
   Instructors to be adequately trained and specialists in their respective fields. All nursing instructors to be registered nurses and well qualified for teaching or administration.
6. *Clinical Facilities.*
   Experience to be available in all departments, special therapies and wards carefully selected for their teaching facilities. All such wards to be in charge of registered nurses with special teaching ability.
7. *Library.*
   A well-stocked library is essential with a varied list of psychiatric and nursing journals.
8. *Student Health.*
   Physical examination, including chest X-ray, at beginning and ending of course. Usual protective inoculations where indicated. Psychological examination as part of general health examination. Staff physician to be designated for care of student health. Hours of duty should be such that the health needs of the nurse have proper consideration.
9. *Living Quarters.*
   A section of nurses' home if available. If satisfactory quarters are not available, students should find their accommodation outside the hospital.
10. *Record System.*
    Complete records as customary in other nursing schools, including health records.
11. *Certificate or Diploma.*
    On satisfactory completion of course, a certificate or diploma to be to be issued by the hospital or school giving the course.

Course No. 1—Deferred Affiliate Course.

(Essentially the same as the basic psychiatric training in the undergraduate course as outlined for the psychiatric affiliation.)

This is designed for graduate registered nurses whose basic training did not include theoretical and practical experience in psychiatric nursing. It is therefore offered to supply minimum training in this field and as a com-

pletion of basic undergraduate training. The nurse taking this course should be qualified to undertake the nursing care of a mentally sick person or to fill a general duty position on the wards of a psychiatric hospital.

1. Time Allotment—12 to 16 weeks.
2. Content of Course:

Psychiatry	15 to 20 hours
Mental hygiene and psychology	10 to 15 hours
Clinical demonstrations	12 to 16 hours
Medical staff conferences	4 to 6 hours
Child psychiatry	4 hours
Neurology	5 to 10 hours
Psychiatric nursing	20 to 30 hours
Special therapies (hydro, occupational, physio, psycho, etc.)	15 to 20 hours

Case studies—2 or 3.

3. Division of Services:

Admission wards	2 to 3 weeks
Acute wards	2 to 3 weeks
Infirmary wards	2 to 3 weeks
Convalescent ward	2 to 3 weeks
Continued-treatment wards	2 to 3 weeks
Special therapies	2 to 3 weeks

Course No. 2—Standard Post-Graduate Course.

This course to be not less than six months' duration. It will include all the curriculum in Course No. 1 (Deferred Affiliate Course) and in addition will offer instruction in certain advanced phases of psychiatry and related subjects to qualify the nurse for senior positions in psychiatric hospital practice or for greater efficiency in private duty.

Length of Course—6 months or longer (but less than 12 months).

5 to 10 hours per week—lectures, classes, conferences.
6 to 8 hours per week—study and preparation.
30 to 40 hours per week—clinical experiences (including incidental teaching and study).

Teaching Methods:

The group should be small enough to permit much of the teaching on an individual basis. Emphasis should be placed on student participation and conferences. Project and case study methods should be used as much as possible. The correlation of the clinical experience with all other forms of instruction cannot be too strongly emphasized. Practice assignments should be planned throughout, primarily with reference to the student's educational needs.

Division of Services.

Content:

Reception service	4 to 6 weeks
Acute service	4 to 6 weeks
Infirmary service	4 to 6 weeks
Convalescent service	4 to 6 weeks
Mental health clinic	4 to 6 weeks
Visits to other hospitals and social agencies	1 to 2 weeks
Occupational therapy department, including physiotherapy	4 to 6 weeks

Outline of Subjects.

Special anatomy and physiology (nervous system, endocrines, etc.)	15 to 20 hours
Psychology (personality structure)	15 to 20 hours
Psychiatry and neurology	30 to 40 hours
Psychiatric nursing and hydrotherapy	30 to 40 hours
Occupational therapy	4 to 8 hours
Mental hygiene (including parent education and child guidance)	15 to 20 hours
Psychometrics	6 to 8 hours
Public health nursing (psychiatric)	8 to 12 hours
Seminars	6 to 10 hours
Staff conferences	12 to 15 hours
Ward clinics	6 to 12 hours

COURSE NO. 3—ADVANCED POST-GRADUATE COURSE.

This course to be not less than twelve months' duration, and may be longer. The first six months should cover the curriculum of the Standard Post-Graduate Course (Course No. 2). The remainder of the period will offer experience and instruction in the psychiatric aspects of public health nursing, administration, teaching and higher executive duties, and neuro-psychiatric nursing of children. It should be thought of as supplemental to special courses in these subjects and for the special training of the nurse who plans a definite career in the psychiatric field.

Length of Course: Not less than 12 months. The first 6 months should cover the work of Course No. 2 (Standard Post-Graduate Course). The second 6 months to be still further advanced work as follows:

Division of Services.

Three months:

Reception service	4 weeks
Acute service	4 weeks
Convalescent or infirmary service	4 weeks

Outline of Subjects.

Advanced psychiatry and neurology	10 hours
Advanced psychiatric nursing	10 hours
Seminars	10 hours
Staff conferences	20 hours
Lectures by visiting speakers	6 hours
	56 hours

Final Three Months: (Three options are suggested. If desired, more than one may be taken, thus further lengthening the course by an additional three or six months).

(a) *Public Health.*

During this time, the student should practise mental hygiene in public health field under supervision. It is impossible to outline definitely this part of the program, as it will be conditioned by the resources available in the particular district.

(b) *Institutional.*

The student should be on one or two of the hospital services; first, helping younger students to plan programs of nursing care for their patients, and later, acting as assistant to the supervisor and learning the direction of the nursing personnel and general administration of the ward. There should also be included, a course, of from ten to fifteen classes in the principles of supervision and methods of ward administration. (These courses are not to be understood as being sufficient in themselves to provide adequate preparation for public or institutional supervisory positions. For nurses in group (a), the regular public health course should come first. Similarly, (b) is not in any way a substitute for the full post-graduate course in teaching or administration.)

(c) *Neuropsychiatric Nursing of Children.*

Special training in the psychiatry of childhood and practical experience in a center specializing in this field.

MINIMUM REQUIREMENTS FOR UNDERGRADUATE SCHOOLS OF NURSING IN MENTAL HOSPITALS.

A school of nursing in a mental hospital to be accredited by The American Psychiatric Association should give a sound basic preparation in nursing and meet the requirements set up by the Board of Nurse Examiners in its own state.

It should be well organized, equipped and financed to function in accordance with the standards set up. The director of the school should have the advice of a qualified board or committee in administrative and educational policies pertaining to the school.

Students, for acceptance, should be qualified by suitable personality and intelligence and by having successfully completed four years of high school work, during which emphasis should have been placed on physics, chemistry and biology.

The faculty should be well equipped, having adequate preparation in teaching and the subjects taught. Teaching methods employed should include lectures, laboratory work, clinical demonstrations, projects, recitations, formal and informal ward discussions, case presentations, attendance at staff conferences.

The school should provide suitable physical and clinical facilities, library and other educational resources for classwork, study and nursing experience.

Living conditions, recreational and social activities and an adequate health program should be provided in consideration of the welfare and happiness of students as well as their educational advancement.

The hours of duty should be so organized as to promote the educational program and preserve the health of the students.

The course should be not less than three years in length. Clinical experience should be correlated with all other forms of instruction and assignments planned, primarily with reference to the students' educational needs.

Experience must be provided in general medical and surgical nursing, including operating room technique, obstetric and pediatric nursing (preferably in a general hospital). The period of affiliation should be preferably the second year and should be approximately twelve months in length. For men students a course in nursing of genito-urinary diseases should be substituted for obstetric and pediatric nursing. One month should be given in a dispensary service and one month of experience in a diet kitchen, under the supervision of a qualified dietitian, in the computation and preparation of special diets.

Affiliation should meet the approval of the State Board of Nurse Examiners and The American Psychiatric Association. If experience in these services is given in the home hospital, the services should be segregated and adequate and meet the approval of the State Board of Nurse Examiners and The American Psychiatric Association. Affiliations of not less than two months are recommended in public health nursing and nursing in communicable diseases. Where an affiliation is impossible to arrange, lectures in communicable diseases, public health nursing and disease of eye, ear, nose and throat should be given.

SUMMARY OF COURSE OF STUDY.

Biological and Physical Sciences	180 hours
Anatomy and physiology	90 hours
Microbiology	45 hours
Chemistry	45 hours
Social Sciences	90 hours
Professional adjustments I (Ethics)	15 hours
History of nursing	15 hours

Psychology and mental hygiene	15 hours
Sociology (including psychiatric social service)	15 hours
Medical-social community problems (Sanitation)	15 hours
Professional adjustments II	15 hours
Medical Science	30 hours
Pharmacology and therapeutics	30 hours
Nursing and Allied Arts	620 hours
Introduction to nursing arts	120 hours
Nutrition and cookery	30 hours
Diet therapy	15 hours
Medical and surgical nursing	180 hours
Obstetric nursing	45 hours
Nursing of children	45 hours
Psychiatric nursing (including clinical psychiatry)	80 hours
First aid and emergency nursing	15 hours
Occupational therapy	15 hours
Hydrotherapy	15 hours
Physiotherapy	15 hours
Recreational therapy	15 hours
Advanced nursing	30 hours

G. H. STEVENSON, M. D., *Chairman,*
Committee on Psychiatric Nursing.

Comment.

DOCTOR HUTCHINGS RETIRES.

It is a matter of gratification and lawful pride to himself and to his friends that a man who has toiled long and faithfully in his chosen field is able to attain the major goals he has set for himself and from the vantage point of his years of labor can survey a record of solid accomplishment.

On July first, after completing his term as president of The American Psychiatric Association, Dr. Richard H. Hutchings retired from the superintendency of The Utica State Hospital over which he had presided for 20 years, following a 15-year period as superintendent of the St. Lawrence State Hospital at Ogdensburg. Dr. Hutchings' career in psychiatry, beginning in 1892, is approaching the half century mark, and during that time have come about most of the movements and methods which characterise the science of today. Dr. Hutchings was not one to await developments and then be carried along with them; by his foresight and initiative he has played a leading part in the reform and extension of hospital work, in bringing institutional practice in line with contemporary medical thought, disturbing the atmosphere of isolation and demonstrating the service functions of the mental hospital in its own community quite apart from the treatment of the patients within its walls. The practical and scientific contributions of Dr. Hutchings have been outlined in the excellent biographical sketch prepared by Dr. Cheney and published in this issue of the JOURNAL. They serve to establish the position of the retiring president as in a very real sense one of the pioneers and builders of modern American psychiatry.

A zeal for progress and innovation, evinced at the beginning of his career, has marked his professional work throughout the years; his youthful spirit has never deserted him. In his presidential address he appraises the mind sickness of the world of today, and draws lessons from the past to indicate what the psychiatry of the future should be.

The presidential year was one of trials and triumphs and withal of arduous toil. His work as an educator was fittingly commemorated last October in the dedication of Hutchings Hall at the Utica State Hospital to house the expanding activities which were a part of his program. That institution and the New York State service owe much to his devoted interest and constructive policies.

Dr. Hutchings has handed over to his successor the president's gavel and has relinquished the responsibilities of hospital administrator, but we look confidently ahead to his continuing labors and influence in the science and in the Association he has served so long and so well.

LUNACY COMMISSIONS ABOLISHED IN NEW YORK.

The JOURNAL has commented previously on the matter of the so-called lunacy commissions of New York State. These commissions, made up of three persons, only one of whom need be a so-called qualified psychiatrist, have been appointed in criminal cases to report to the court the mental state of the defendant. Quite aside from the fact that the reports have often been apparently biased and of doubtful psychiatric value, the commissions have furnished a very fertile source of fees for persons to whom the judges felt themselves under political obligation, and have caused tremendous expense, particularly in Brooklyn. An attempt was made last year to abolish these lunacy commissions, but unfortunately the bill appeared to suffer from some technical defects. This year Senator Desmond introduced a bill which was passed by both houses, and on June 16 was signed by Governor Lehman with the comment:

> I believe it corrects existing abuses and proposes a workable procedure which is greatly preferable to the present law.

The Governor further said:

> The purpose of this bill is to establish a sound procedure whereby the medical question of the sanity of a defendant at the time of trial will be determined solely by qualified psychiatrists instead of by a lunacy commission of three members, only one of whom need be a psychiatrist.

In New York City the bill provides that examination shall be made by psychiatrists on the staff of the Bellevue Hospital, and outside of New York City by staff physicians of the state hospitals.

The McNaboe bill, which would have permitted judges at will to commit suspects as insane, was vetoed by the Governor who said:

> In my opinion, this bill, rather than being an improvement over the present procedure, represents a backward step.

With the enactment into law of the Desmond bill New York State is to be congratulated on making a very important advance in dealing with the difficult issue of the insanity plea.

It is fortunate and opportune that we are able to publish in this issue of the JOURNAL a statement from the pen of the sponsor of the new legislation, State Senator Thomas C. Desmond, who clearly sets forth the provisions of the law, the advantages it will serve and the shocking abuses it will correct.

News and Notes.

MEETING OF THE AMERICAN PSYCHIATRIC ASSOCIATION.—The ninety-fifth annual meeting of The American Psychiatric Association was held at the Palmer House Hotel, Chicago, Ill., May 8 to 12, 1939.

On Monday there were meetings of the sections on convulsive disorders and forensic psychiatry. The meetings of the section on convulsive disorders were joint sessions with the American Chapter of the International League against Epilepsy, including a well attended informal evening panel discussion at which Dr. David R. Wilson presided.

The general session opened Tuesday morning, Dr. H. Douglas Singer, chairman of the Committee on Arrangements introducing the local officials, Honorable A. L. Bowen, director, Department of Public Welfare and Honorable John J. Sonsteby, Chief Justice of the Municipal Court of Chicago, welcoming the Association, following the invocation by Herbert W. Prince, D. D., rector, Episcopal Church, Lake Forest, Ill. Dr. William C. Sandy, President-Elect, responded to the addresses of welcome and then presided, while the President, Richard H. Hutchings, M. D., of Utica, New York, delivered a very able annual address.

The Association paid tribute to the memory of 28 deceased members.

Three section meetings on Tuesday afternoon included papers on the symposium on the integration of state hospital programs, clinical studies and studies from the laboratory.

On Wednesday there was a joint session with the section on psychoanalysis and the American Psychoanalytic Association; also, sections on clinical studies, organic brain pathology and social problems.

On Wednesday evening the annual dinner occurred at which the members and guests had the privilege and pleasure of hearing Dixon Ryan Fox, LL. D., Litt. D., President, Union College, Schenectady, N. Y., present the annual address entitled, "The

Attractive Aspects of National Poverty." This was one of the most stimulating and thought-provoking addresses ever presented before this Association. Following the president's reception, there was an enjoyable evening of dancing until 1.30 a. m. with music furnished by the Lew Diamond Orchestra.

On Thursday morning and afternoon there were well attended sections dealing with studies related to pharmacological shock therapy, extramural studies and special tests. On Thursday afternoon there was a joint session with the section on mental deficiency.

On Thursday evening there were 18 round table dinner discussions, the subjects being advisory psychiatric service in community social agencies, child psychiatry, college mental hygiene, forensic psychiatry: "After Prison, What?," former patient associations, language and thought in schizophrenia, occupational therapy, pharmacological shock therapy, psychiatric education, psychiatric nursing, psychiatric social service, psychiatry in industry, Veterans Administration, research organization, the inter-relation of schizophrenia and manic-depressive psychoses: the significance of their co-existence, psychosomatic relationships, the rôle of the psychoanalyst as an advisor in sexual and marital adjustment and unsuccessful sex adjustment in marriage.

On Friday there were two sessions, the subjects being clinical and therapeutic studies and a section on mental deficiency.

Interesting scientific exhibits under the special direction of Dr. Walter L. Bruetsch and many interesting moving picture demonstrations, under the supervision of Dr. J. D. Reichard, were given and attracted a very large attendance. The commercial exhibits included books, apparatus and other displays of interest to mental hospital administrators.

The Committee on Arrangements with Dr. H. Douglas Singer as chairman carried out the plans for the entertainment of the Association and its guests in an exceedingly interesting and entertaining manner. On Tuesday morning May 9, occurred the laying of the cornerstone of the new Illinois Neuropsychiatric Institute. Addresses were made by the president of the Association, Dr. Richard H. Hutchings, by Dr. H. Douglas Singer and Dr. Adolf Meyer. Transportation was furnished for members and many

attended this historic occasion for the development of psychiatric work.

A special program for ladies, under the direction of Mrs. Leon J. Saul, consisted of sight-seeing trips, tea at the Chicago Women's Club, and a luncheon, fashion show and bridge at Marshall Field's Department Store.

In accordance with the report of the Board of Examiners and upon recommendation of the Council, the Association elected 64 to associate membership; 150 to membership; 33 were transferred from associate membership to membership; 14 transferred from membership to fellowship and five reinstated.

The following were elected officers for 1939-40:

William C. Sandy, M. D., President, Harrisburg, Pa.
George H. Stevenson, M. D., President-Elect, London, Ont., Canada.
Arthur H. Ruggles, M. D., Secretary-Treasurer, Providence, R. I.
Council members elected for three years: Richard H. Hutchings, M. D., Utica, N. Y.; Lawrence Kolb, M. D., Washington, D. C.; William J. Tiffany, M. D., New York, N. Y.; and E. Armitage Baber, M. D., Cincinnati, Ohio.
Charles W. Castner, M. D., Austin, Texas, was elected an auditor for three years.

The following were elected the officers of the sections for 1939-40:

Section on Convulsive Disorders: David C. Wilson, M. D., chairman, University, Va., and Albert W. Pigott, M. D., secretary, Skillman, N. J.
Section on Forensic Psychiatry: James L. McCartney, M. D., chairman, Philadelphia, Pa., and Lowell S. Selling, M. D., secretary, Detroit, Mich.
Section on Mental Deficiency: E. P. Lewis, M. D., chairman, Toronto, Canada, and Oscar J. Raeder, M. D., secretary, Boston, Mass.
Section on Psychoanalysis: Clarence P. Oberndorf, M. D., chairman, New York, N. Y., and Flanders Dunbar, M. D., secretary, New York, N. Y.

It was announced that the ninety-sixth annual meeting will be held at Chateau Frontenac, Quebec, Canada, during the week of June 3 to 7, 1940.

The total registration was 1402, of which number 574 were members. This is the largest number of members ever to attend a meeting in the long history of the Association.

The unusually large attendance at this meeting is a tribute to the exceedingly well-balanced program that was prepared under the direction of Dr. Roscoe W. Hall, chairman of the Program Committee, and the especially well-organized publicity obtained

through the Committee on Public Education of which Dr. C. C. Burlingame is chairman, as well as the convenient geographical location of Chicago.

The excellent facilities of the Palmer House and their most cooperative convention department, which was represented by Mr. M. P. Mathewson, added greatly to the comfort of the members and the organization of the sections. The efficient manner in which the registration was handled was due to the Chicago Association of Commerce, through their representative, Mr. Maguire, in furnishing registration girls for the desk and both girls and typewriters for the publicity. It is worthy of much more than passing notice that for the first time in the history of the Association no part of the funds of the Association was used to finance this meeting, and the Association did not have to solicit financing from the Committee on Arrangements; in fact, under the able direction of Mr. Austin M. Davies, executive assistant, there was a very slight balance accruing to the Association.

ARTHUR H. RUGGLES, M. D., *Secretary.*

SYMPOSIUM ON VISUAL FATIGUE.—On May 20-21, 1939, in Washington, D. C., under the auspices of the Committee on Scientific Aids to Learning of the National Research Council, was held a two day symposium on Visual Fatigue. Dr. Walter R. Miles, Chairman, presided. Approximately forty were in attendance representing different research interests and institutional connections.

The first day was devoted to fourteen contributions by leading representatives of the following universities and research laboratories: Yale University 2, Harvard University 3, Brown University 1, Columbia University 2, University of Rochester 1, Dartmouth Medical School 1, University of Minnesota 1, Tulane University 1, General Electric Co., Cleveland 1, and Eastman Kodak Co. 1. A Round Table Conference was held the second day, at which the speakers of the previous day endeavoured to formulate research plans in the general field of visual fatigue studies. It is probable that research will be undertaken in the general field under the auspices of the Committee on Scientific Aids to Learning of the National Research Council.

BURDEN NEUROLOGICAL INSTITUTE.—Professor F. Golla supplies the following information concerning the new institution for psychiatric research at Bristol, England.

The Institute, founded by the generosity of Mrs. R. G. Burden, has been built in Stoke Park, a large estate in the vicinity of Bristol, in which is a group of well-known hospitals for mental deficiency, which were founded by Mrs. Burden's late husband.

The present institution is however autonomous and under the direction of Professor Golla, assisted by a staff of physiologists, biochemists and endocrinologists. The laboratories are well equipped and two small observation wards, each containing eight beds, are attached to the institution.

It is proposed to conduct intensive research work on selected cases both of psychosis and of epilepsy, in conditions which free the workers from the cares and exigencies of a large hospital.

In addition to the extensive laboratory accommodation there is a neuro-surgical unit, and it is proposed to utilize this for the surgical treatment of suitable epileptic cases, and incidentally to learn much about the physiology of the human cerebral cortex. The endocrinological side has also been fully developed, and under Professor Reiss of Prague it is proposed to investigate the relationship of psychosis to disturbances of the regulation of internal secretions.

MICHAEL REESE HOSPITAL PSYCHIATRIC SERVICE.—Dr. Roy R. Grinker, chairman of the department of neuropsychiatry, Michael Reese Hospital, Chicago, announces the appointment of Dr. Maxwell Gitelson, formerly chief of staff at the Institute for Juvenile Research, as attending psychiatrist in a full-time capacity beginning July 1, 1939. Coincident with this appointment the departments of psychiatry and neuropsychiatry have been combined into one department of neuropsychiatry, the personnel of which will be both full-time and part-time clinicians. Dr. Gitelson, as full-time psychiatrist, will act as executive director of the psychiatric services. There is yet another opening for a full-time psychiatrist on the children's service and several openings for part-time practicing psychiatrists. The new department encompasses all the psychiatric services in the hospital and clinic, neurology and neurosurgery. Working complementary and auxiliary to this department

is the psychological laboratory, directed by Dr. Samuel Beck, in which routine and research psychological testing is carried out.

The hospital has just authorized the opening of an inpatient closed psychiatric unit in which both private and service psychiatric cases will be hospitalized. It is hoped that this unit will be open for occupancy within a very short time and will assist in furthering the progress within this country of the care of those psychiatric problems which are part of the responsibility of a general hospital and its supporting community.

PROFESSOR RÜDIN RECEIVES THE GOETHE MEDAL.—On the occasion of his sixty-fifth birthday in recognition of his services in the field of race hygiene in Germany, Professor E. Rüdin was awarded the Goethe Medal for Art and Science.

The Reichsminister of the Interior, Dr. Frick, transmitted to Prof. Rüdin the following congratulatory message: "To the tireless pioneer in race hygiene whose meritorious services prepared the way for the race hygiene ordinances of the Third Reich I send, on his sixty-fifth birthday, my heartiest good wishes. May you long be permitted to continue your studies for the welfare of mankind."

BIOLOGICAL PHOTOGRAPHIC ASSOCIATION.—The ninth annual convention of the Biological Photographic Association will be held September 14-16, 1939, at the Mellon Institute for Industrial Research, Pittsburgh, Pa. The program will be of interest to scientific photographers, scientists who use photography as an aid in their work, teachers in the biological fields, technical experts and serious amateurs. It will include discussions of motion picture and still photography, photomicrography, color and monochrome films, processing, etc., all in the field of scientific illustrating. Up-to-date equipment will be shown in the technical exhibit; and the Print Salon will display the work of many of the leading biological photographers here and abroad.

The Biological Photographic Association was founded nine years ago because of the growing need for expert illustrative material for scientific research and teaching. Many workers were solving their problems in their own way; but obviously they were

wasting time and effort in individually repeating experiments that had been worked out elsewhere. The B. P. A. was formed to act as a clearing house for new ideas, to pool experiences, record standard procedures and disseminate information. Its aims were scientific and all services have been volunteered by officers and members on a non-profit basis.

The B. P. A. Journal is published quarterly, constituting a Volume of about 250 pages, which is furnished free to members. Membership privileges include an authoritative question and answer service; also the right to borrow loan albums and exhibits of scientific prints for study and display.

Further information about the Association and the convention may be obtained from the secretary of the Biological Photographic Association, University Office, Magee Hospital, Pittsburgh, Pennsylvania.

HEMATOLOGY SYMPOSIUM, UNIVERSITY OF WISCONSIN.—The University of Wisconsin Medical School is to conduct an Institute for the Consideration of the Blood and Blood-Forming Organs, September 4-6, 1939. The program is to include papers and round-table discussions by European and American workers in the field of hematology. In addition to the discussions, the following formal papers are to be presented:

Dr. L. J. Witts, Oxford, England, Anemias due to Iron Deficiency.

Dr. Cecil J. Watson, Minneapolis, The Porphyrins and Diseases of the Blood.

Dr. Cornelius P. Rhoads, New York, Aplastic Anemia.

Dr. E. Meulengracht, Copenhagen, Denmark, Some Etiological Factors in Pernicious Anemia and Related Macrocytic Anemias.

Dr. Harry Eagle, Baltimore, The Coagulation of Blood.

Dr. George R. Minot, Boston, Anemias of Nutritional Deficiency.

Dr. Russell L. Haden, Cleveland, The Nature of the Hemolytic Anemias.

Dr. Jacob Furth, New York, Experimental Leukemia.

Dr. Claude E. Forkner, New York, Monocytic Leukemia and Aleukocythemic Leukemia.

Dr. Edward B. Krumbhaar, Philadelphia, Hodgkin's Disease.

Dr. Louis K. Diamond, Boston, The Erythroblastic Anemias.

Dr. Edwin E. Osgood, Portland, Marrow Cultures.

Dr. Charles A. Doan, Columbus, The Reticulo-Endothelial System.

Prof. Hal Downey, Minneapolis, Infectious Mononucleosis.

Dr. Paul Reznikoff, New York, Polycythemia.

Physicians and others who are interested are cordially invited. A detailed program may be obtained by addressing Dr. Ovid O. Meyer, chairman of Program Committee, University of Wisconsin Medical School, Madison, Wisconsin.

THE SIXTH BIENNIAL CONGRESS OF THE INTERNATIONAL HOSPITAL ASSOCIATION.—This Congress to be held in Toronto, Canada, September 19 to 23, 1939, was previously announced in the March number of the JOURNAL.

The five-day Congress will consist of several general, or plenary, sessions, together with more individualized meetings of the 40 Study Committees, reporting their two years of work in their special fields, ranging from hospital architecture and management to various professional services. In all the meetings of the Congress, standardization of common functions and mutual interests will be emphasized. Mobilization of scientific resources and facilities will be reviewed with an aim toward increased understanding and efficiency.

The detailed program of the Congress may be seen in *Nosokomeion*, IX: 253-268.

Neurologists will be especially interested in the reports and discussions of Study Committee XXVIII. Dr. Lewy and his committee will present a program dealing with "The Management of Head Injuries in the General Hospital." The neurological, surgical, sociological and medico-legal aspects of the problem will be reviewed, and should awaken liberal discussion.

Psychiatrists will be interested in the presentation of a symposium on schizophrenia by Study Committee XXVII. This committee anticipates a liberal discussion of the problems of schizophrenia, and hopes that some of the master students of the problem will outline their suggestions for a unification and amalgamation of the gains already made, and will voice ways and means for analyses and studies still to be made.

All who are interested in the care of the sick will be interested in this Congress, and it is hoped that the stimulating help to be obtained from it will make an urgent appeal for attendance.

THOS. J. HELDT.

ADVISORY COUNCIL ON MEDICAL EDUCATION.—At a meeting in Chicago June 24, 1939, of eleven national organizations concerned with the training of physicians to meet the present day needs of medical care for the country, was created the Advisory Council on Medical Education. The need for some central representative agency has long been recognized as necessary to make medical training more effective than it has been in its service to the public.

Dr. Willard C. Rappleye, Dean of the Faculty of Medicine of Columbia University, who was elected president, stated that the purpose of the Advisory Council is to correlate the efforts of the universities, medical schools, hospitals, licensing bodies, public health associations and boards of specialists.

This organization brings together for the first time the various national bodies dealing with all phases of the training and licensing of physicians beginning with his preparatory college work and including medical education, hospital internship, residency, licensure and graduate training for specialization. Cooperation and coordination are to be substituted for present overlapping and competing functions of existing agencies.

The organizations represented on the Advisory Council on Medical Education are:

	Number of representatives.
Association of American Medical Colleges	3
American Hospital Association	3
Catholic Hospital Association	3
Federation of State Medical Boards of the U. S. A.	3
Advisory Board for Medical Specialties	3
National Board of Medical Examiners	1
American College of Physicians	2
American College of Surgeons	2
Association of American Universities	2
American Association for the Advancement of Science, Division of Medical Sciences	1
American Public Health Association	1

Two new organizations were elected to membership:

American Protestant Hospital Association	1
Association of American Colleges	2

Among the important problems considered by the Council at its meeting were those of proper educational standards of the hospital internship, adequate training for the specialist, sound programs

for the continued education of physicians in practice, modifications in college preparation for medical studies, the simplification of the procedure for licensure in the 48 separate states, and the status of training of graduates of foreign medical schools. Special committees of the Council were appointed to study these various questions.

The officers of the Advisory Council are:

Dr. Willard C. Rappleye, dean of the Faculty of Medicine of Columbia University ..... President.
Dr. Maurice H. Rees, dean of the School of Medicine of the University of Colorado ..... Vice President.
Dr. Robin C. Buerki, director of study of the Commission on Graduate Medical Education ..... Secretary-Treasurer.

The Executive Committee consists of the officers and the following:

Dr. Anton J. Carlson, professor of physiology of the University of Chicago.
Dr. Harold Rypins, secretary of the New York State Board of Medical Examiners.
Dr. Hugh J. Morgan, professor of medicine of Vanderbilt University.
Dr. Arthur W. Allen, surgeon, Boston.

DEATH OF DR. O'MALLEY.—Dr. Mary O'Malley, for many years clinical director of Saint Elizabeths Hospital, Washington, D. C., died on January 30, 1939, in Buffalo, N. Y. Dr. O'Malley was born in Medina, N. Y., February 8, 1867, was graduated from the University of Buffalo Medical School in 1897 and for several years was a member of the staff of the Binghamton State Hospital. In 1903, Dr. William A. White, on leaving Binghamton to become superintendent of Saint Elizabeths Hospital, persuaded Dr. O'Malley to accept an appointment there. She rendered valuable service to Saint Elizabeths Hospital until her retirement on account of ill health in 1935. During her service she made many valuable contributions to psychiatric literature, notably on hydrotherapy and pellagra. In 1931 she was delegate to the Medical Women's Congress in Vienna, and in 1933-34 was president of the Medical Women's National Association. She was for many years a member of the American Medical Association, American

Psychiatric Association, and the Washington Psychopathological Society.

Dr. O'Malley was one of the most widely known women in the field of psychiatry, and her many friends mourn her passing.

DR. CARROLL PRESENTS HOSPITAL TO DUKE UNIVERSITY.—Dr. Robert S. Carroll of Asheville, N. C., has presented to Duke University, Highland Hospital, a private institution for nervous and mental patients, of which he was the founder and which he has served for many years as medical director.

The hospital, delightfully situated in 400 acres of mountain woodland, is to be used as a university clinic for teaching and treatment, in connection with the new unit now being built at Durham to permit the development of the department of psychiatry in the medical school of Duke University. Dr. Carroll will continue as medical director of the hospital for five years, during which a clinical staff under the department of psychiatry will be organized.

DR. DUNTON RETIRES FROM HOSPITAL WORK.—Dr. William Rush Dunton, Jr., a senior member of the editorial board of the JOURNAL, and who for many years has been the medical director of Harlem Lodge, a private sanitarium for nervous and mental patients at Catonsville, Md., retired from this position March 31, 1939.

Dr. Dunton took charge of Harlem Lodge, December 1, 1924, after having served for a considerable number of years as senior resident at the Sheppard and Enoch Pratt Hospital at Towson, Md., and as an instructor in psychiatry in the Johns Hopkins Medical School. He is affiliated with the Laurel Sanitarium and will continue private and consultation work and his activity as a member of the editorial board.

AMERICAN ASSOCIATION ON MENTAL DEFICIENCY.—The sixty-third annual meeting of the American Association on Mental Deficiency was held at the Palmer House Hotel, Chicago, Illinois, May 3-6, 1939.

The meeting was called to order on Wednesday, May 3, by President Neil A. Dayton of Boston, Massachusetts. A total of

58 papers made up the program which occupied four days with double sessions in both mornings and afternoons. Two round table meetings were held each day. The round table on "The Capitalization of Research" was conducted by Dr. Edgar A. Doll of Vineland, New Jersey, as moderator. At the second luncheon discussion on "What Should Be Included in a Study of Educational Practices for Mentally Defective Children" Dr. Elise Martens of Washington, D. C., presided as moderator. On Friday, section I of the round table luncheons had as its subject "Problems in Institution Administration" and Dr. Benjamin W. Baker, of Laconia, N. H., acted as moderator. At the second luncheon with "Psychological Service and Research in State Institutions" as the topic Dr. Elaine F. Kinder of Thiells, N. Y., presided. The section I luncheon round table on Saturday on "The Intelligence Quotient and Its Interpretation" had Dr. Grace Munson of Chicago, Ill., as moderator. At the section II luncheon "The Future for Extra Institutional Supervision" was discussed, with Miss Mabel Ann Matthews of Mansfield Depot, Conn., as moderator.

Following the president's dinner and address on Friday evening, a dance was held which was greatly enjoyed by the membership.

The papers presented covered the four fields of medicine, psychology, social work and education. Extremely interesting papers were read by Dr. J. A. Fraser Roberts, principal investigator, Burden Mental Research Trust, Stoke Park Colony, Bristol, England and by Dr. Lionel S. Penrose, research medical officer, The Royal Eastern Counties' Institution, Colchester, England.

The new officers for the coming year are: Dr. Fred Kuhlmann, director, Division of Examination and Classification, State Department of Public Institutions, St. Paul, Minnesota, president; and Dr. Meta Anderson, director of Binet Schools, Newark, N. J., vice-president.

Dr. E. Arthur Whitney, superintendent, Elwyn Training School, Elwyn, Pennsylvania was re-elected as secretary-treasurer.

Attendance at Chicago set a new high for this Association. A total of 456 persons registered for the various sessions.

The 1940 meeting will be held in Atlantic City, New Jersey, May 15-18, 1940.

PSYCHIATRY AT THE MEDICAL COLLEGE OF VIRGINIA.—There is under construction at Richmond an 18 story hospital building in which medical students of the Medical College of Virginia will receive instruction. One floor of this new structure providing for 38 beds will be devoted exclusively to psychiatry. It is expected that the hospital will be opened during the spring of 1940, and that regular clinical instruction can be offered with the beginning of the academic year 1940-41.

Dr. R. Finley Gayle, Jr., is professor of the department of neuropsychiatry, having succeeded Dr. Beverley R. Tucker one year ago.

THE POSITION OF PSYCHIATRIST, NEW YORK CITY PUBLIC SCHOOLS.—The examination for license as school psychiatrist for the New York City public schools will be announced in the near future. Those interested in this examination should write for particulars to Dr. Joseph K. Van Denburg, Board of Examiners, 500 Park Avenue, New York City.

Applicants for examination for this position must be between 30 and 45 years of age; graduates of a recognized grade A medical school and hold a license to practice in the state of New York. The candidate must have had, in graduate or undergraduate work, 12 semester hours in approved and appropriate courses in psychiatry and psychology and 6 semester hours in approved courses in education. The course in education may be completed, however, within three years after the license is issued. The candidate must have had five years of practice in psychiatry, including 800 hours of appropriate clinical experience in approved institutions for the treatment of mental diseases and mental defects and 1200 hours of appropriate experience in approved clinics for the study and treatment of personality and behavior disorders of children.

SMITH COLLEGE STUDIES FOR SOCIAL WORK.—This JOURNAL is the official organ of the Smith College School for Social Work, Northampton, Mass. It made its first appearance in September 1930, and is issued quarterly under the editorship of Helen Leland Witmer, Ph.D., Supervisor of Research, and Professor Everett

Kimball, Director of the Smith College School for Social Work. The JOURNAL publishes theses submitted by students of the School as required for the degree of Master of Science. These studies cover a wide range of current developments in the fields of social work, social psychiatry, sociology and psychology. During the year 1938-39 44 agencies in the states of Connecticut, Delaware, Illinois, Maryland, Massachusetts, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, Wisconsin and District of Columbia cooperated with the School in the field work of its students.

The Smith College School for Social Work opened as a graduate school for psychiatric social workers July 8, 1918. The National Committee for Mental Hygiene anticipating the great need of trained psychiatric social workers to aid in the treatment and reeducation of soldiers suffering from war neuroses and psychoses, brought the matter to the attention of Dr. William A. Neilson, president of Smith College, who was already considering several types of war work for a possible summer school.

The founding of the Smith College School for Social Work represented a milestone in the history of social welfare inasmuch as it was the first to recognize the importance of psychiatry and mental hygiene, and to provide an adequate training course for psychiatric social workers. Under the auspices of The National Committee for Mental Hygiene, in conjunction with the Boston Psychopathic Hospital and a sub-committee composed of Dr. E. E. Southard, chairman, Drs. William L. Russell, L. Pierce Clark, Walter E. Fernald and President Neilson, a course of eight weeks didactic work was provided at Smith College followed by six months practical work at hospitals and organizations in four cities, Baltimore, Philadelphia, New York and Boston. The course dealt with pertinent topics from the fields of psychology, sociology and psychiatry. Miss Mary C. Jarrett, who had been associated with Dr. Southard at the Boston Psychopathic Hospital, had general supervision of the course and of the practical work. Some 70 women, ranging from girls just graduated from college to experienced social workers were selected from all parts of the country, 20 states and 20 colleges being represented. During the course 26 distinguished psychiatrists from the Pacific to the Atlantic came to Smith College to give lectures on special topics; there were also two from Canada who contributed of their experience with the war neuroses.

Smith College continued the School after the war as a permanent institution; and the 21st winter session opened with an enrollment of 112 students in the field. The School now presents an educational programme of 5 sessions covering two years and two months, that is, three summers of academic work at the School and two winters of supervised field training in social work in agencies selected by the School. This course leads to the degree of Master of Science.

WILLIAM ALANSON WHITE MEMORIAL LECTURES.—The superintendent and staff of Saint Elizabeth's Hospital and the board of trustees of The William Alanson White Psychiatric Foundation announce the first William Alanson White Memorial Lectures on Modern Psychiatric Conceptions by Harry Stack Sullivan, M. D.

Lectures will be given on Friday evenings at 8.30 p. m. at the Auditorium, Interior Department, Washington, D. C.

SUBJECTS.

Conceptions of Modern Psychiatry	27 October, 1939
Organism and Environment	3 November, 1939
Diagnostic and Prognostic Syndromes	10 November, 1939
Explanatory and Therapeutic Conceptions	17 November, 1939
Prospective Developments and Research	24 November, 1939

Book Reviews.

THE STARTLE PATTERN. By *C. Landis, Ph.D.*, and *W. A. Hunt, Ph.D.* (New York: Farrar & Rinehart, Inc., 1939.)

The interest of Landis and Hunt in this theme has for some years been familiar to those who follow current psychological publications. Those who desire details of method and evidence must go to the several papers they and colleagues have published, which form part of a considerable list of citations. Possibly the work would have gained by a little closer coordination of these references to the body of the text; at least to the reader who gets his introduction to the topic through this volume. As a digest of the more technical contributions it is very ably done.

The topic is, of course, the pattern of relatively unadaptive movement that takes place on sudden intense stimulus, preferably loud noise. It has some history, comparable to the pre-Rorschach study of ink-blots; a similar function has been applied to the study of fitness for certain hazardous occupations. The normal pattern is a general flexion (*cf. zusammenschrecken, zusammenfahren,* "struck all of a heap"). The reaction times of these responses have some theoretical interest in relation to emotion (*cf.* p. 153), and the present ones are probably the most accurate measures we have. Eye-blinks run on the order of 40 σ; arm movements about those of voluntary reaction to sound. Habituation generally diminishes the response, with much individual difference; save that the eye-blink seems always retained.

The startle effect may be a function of the intensity or unexpectedness of the stimulus, in any relation. Other types of stimulus than loud noise were tried, in general less effective than the gun-shot, though a jet of ice-water was comparably so. Some degree of conditioning could be established, again with marked individual difference. One would expect to find differences in personality organization reflected in the work, and it is so stated, but not enlarged upon. In the primate mammals, and invertebrates generally, the flexion pattern of the startle response is preserved, but it is not found in reptiles or amphibia.*

Relation to the Moro reflex, a generally extensor pattern, is discussed; the authors prefer the view that the Moro is the original startle response,

* The reviewer takes occasion to mention at this point an analogous phenomenon sometimes reported for orb-weaving spiders, *i.e.*, dropping at the approach of a vibrating tuning-fork. It is not characteristic save of small species or young individuals, and then only in the daytime. With nightfall there is gradual transition to a pattern more aggressive in character; the reverse phenomenon at sunrise.

gradually supplanted by the conventional startle pattern during the age period six weeks to four months. From then on the pattern appears constant throughout life. Work with twins throws out the tentative but very interesting suggestion that "confused" hemispherical dominance may increase the startle response through diminished cortical control.

Little effect on the pattern is observed in the common neuropathological pictures. Discussion of its nervous mechanism centers about the separation of the eye-blink from the more postural elements. These are looked on as displaying a brief overactivity of the red nucleus; the eye-blink having a separate and less established mechanism. Among psychoses, the catatonic classification was distinguished by exaggerated response. Little difference appeared between the phases of manic-depressive psychosis, and the cases of general paralysis observed were quite comparable to the normal pattern.

Among the feeble-minded group it is some matter for surprise that clinical varieties are not more distinguished by the authors. Qualitatively the pattern is similar to the normal, greater in amount. This is not given the interpretation that might be expected, lack of cortical inhibition, but understood on more intellectualized grounds. The response is diminished under hypnosis, in the absence of specific instructions. With instructions to inhibit, it may be practically abolished, independently of whether the subject is "not to jump" or "not to hear the gun." The very limited ability to inhibit the response during the waking state makes this observation especially striking.

Among the more distinctive observations are those on epilepsy, where a marked disturbance of the pattern is shown. The normally constant lid reflex is particularly disturbed. "In our experience missing or incomplete lid reflexes seem to be specific for epilepsy" (p. 99) . . . . "Wherever this disorganized startle pattern is found we can safely say that epilepsy exists" (p. 151).

Taking a lay attitude towards the background of the work, one is overtempted to a brief excursion in casuistry. Psychiatry, and certain branches of physiology and psychology are distinctive in that their research involves the organism as a whole, and a consequent treatment of the individual as "means". Where human beings are involved, the ethics of such practices depend on (1) the degree of momentary or lasting hurt to which the individual may be exposed, (2) the intelligent willingness of the individuals to assume discomforts and/or risks involved. The progress of science may be modified by oversensitivity to these issues (1) on the part of investigators, (2) on the part of the lay public, through pressure of opinion and legislation (*cf.* animal experimentation). Actually it is doubtful if the former of these factors has been or will be of serious import. The latter will bear more watching. It is a certain irony that the totalitarian polity, much inveighed against as a suppressor of scientific spirit, furnishes conditions in some ways advantageous to inquiries of this kind, through its very alienation of "individual rights." An equalitarian culture on the other hand, may well be more jealous of "rights" concerned, and relatively critical of motives in the more elementary forms of investigative instrumentalizing. Whatever

inviolability rests in the spirit of research as such, it is no esoteric wisdom that the motives by which a particular individual is impelled to serve or criticize it may be functions of his constitution, psychogenesis, endocrines, or what have you, (*Der Weg zum Ruhm geht durch den Schlafzimmer des.. . . .*) Various deception tests, hypnotic procedures, "experimental panics" and other studies of emotional life are subject to scrutiny from these standpoints; and if such matters are indiscreetly managed, their disciplines may one day face a Researchers' Oath as well as a Teachers'. An ominous picture, some cousin of Almus Pickerbaugh getting up in a legislative hearing at the State Capital of Winnemac, "The Startle Pattern" open at p. 62 in his hand, and Luke, Ch. XVII, v. 2, on his lips.

The relative modesty and simplicity with which the material of this book is presented must not make the reader overlook the great amount of work with high technical ingenuity and skill of which it is a product. The economic sponsors are to be congratulated on this contribution to the understanding of fundamental behavior mechanisms, and the advancement of research technique, which they have made possible.

F. L. Wells, Ph. D.,
Harvard University.

Martin Luther's Umwelt, Charakter und Psychose sowie die Bedeutung dieser Faktoren fur seine Entwicklung und Lehre. I. Die Umwelt. By *Dr. med. Paul J. Reiter.* (Copenhagen: Levin & Munksgaard, 1937.)

This book is an attempt at a psychiatric diagnosis across the centuries. To make this possible the physician turns historian. Thus the place of consultation or interview is taken by a careful scrutiny of the environment and the biographical detail. *Umwelt* as conceived by the author comprises the common consciousness of the time, the condition and problems of the Church in the century preceding Luther's activity, the political, economic, cultural and social aspects of the period, the local circumstances and the home. This is followed by a brief analysis of the milieu and its effect upon the personality of Luther after 1517. The bulk of the book deals with the spiritual and professional milieu, the Church (pp. 37-276); the political and social factors are compressed into 31 pages, the local factors into 30 pages. The discussion of the home together with its evaluation for the later personality of the reformer occupies a mere 16 pages; the origin of his neurosis is linked with the abnormal severity, gloominess and superstitiousness of the home tyrannized by the father. The last chapter of the book (6 pp.) reviews and partly also anticipates the following volumes, a procedure for which there seems to be little motivation.

The pattern of Luther's life as it here emerges, is simple. There are two periods divided, as by a watershed, by the great protests in the years 1517-20. The earlier part is dominated by his neurosis accentuated by the constraints of the monastic life, especially the celibacy, and the second part

is the release and abreaction. The bull of excommunication and marriage are the chief events in this process of recovery and the vehement polemic aids and accompanies it. Married life, conviviality in his home, and public activities all have a share in the removal of the neurosis and the essentially sthenic and syntonic personality becomes extraverted.

The importance of the subject of this biography is suggested by the author's opinion that Luther is perhaps the greatest German of all times, an evaluation which is too vague and too general to carry much meaning.

The historical chapters are a restatement of commonly known facts, newly arranged however for the special purpose of the author, *i. e.*, to catch the tone of everyday life of the period, to suggest its values and provide a clue to the elements of Luther's consciousness. What the author does with these premises can only be judged from the later volumes.

The pictorial material is comparatively slight and can add nothing that is new.

The book is printed in good type and on substantial paper but the typographical errors rather exceed the usual quota—the book having been printed in Denmark. In the table of contents greater clarity could have been achieved by distinguishing headings and subdivisions by different type.

R. K. Arnold, Ph. D.,
University of Toronto.

Why Grow Old? A Guide-Book for the Man Who Seeks to Remain Physically and Mentally Young. By *Frank S. Caprio, M. D.*, and *Owsley Grant, M. D.* (Indianapolis: Maxwell Droke, 1938.)

Mankind still plays with magic. Whether we consider the alchemists of another day who would transmute lead into gold, or Ponce de Leon seeking the elixir of life, they have their modern counterparts. Only the technique changes. Now lead is bombarded with neutrons and we perform a vasectomy on the fountain of youth.

The present authors present a sort of geriatric psychotherapy. They frankly tell the reader they have no remedy for disintegration, but point out how modern medical science is prolonging life and ameliorating the degenerative diseases. They explain that "old age" in its ordinary connotations is a state of mind only. Their readers—persons of 40 and upwards—are advised not to seek youth in strenuous exercise or medicine, but in serenity of soul, in widening the interest, and in the "natural" exercises, walking, swimming, dancing.

For the elderly person who worries over his health, there are a number of reassuring chapters on heart disease, rheumatism, cancer, tuberculosis, etc. There are also several chapters on mental hygiene of a rather Pollyannish nature. One chapter is given to the sex life, with some useful aphorisms, the authors themselves admitting they cannot discuss it adequately in the allotted space.

On the whole, a book, which, while not especially profound, can safely be placed in the hands of the person who is growing old and doesn't like it. To many such a one it will undoubtedly be beneficial.

JOHN E. LIND, M. D.,
St. Elizabeths Hospital,
Washington, D. C.

ESSENTIALS OF PSYCHIATRY. By *George W. Henry, M.D.* Third Edition. (Baltimore: Williams & Wilkins Co., 1938.)

The publication of the third edition is ample evidence that Henry's *Essentials of Psychiatry* has found a place for itself in the field of psychiatric textbooks. The new edition, while enlarged and amplified, follows the same general pattern of presentation as did the previous ones and therefore has somewhat similar values and defects.

The presentation of the principles of psychiatry in the form of a textbook is a matter of extreme difficulty. Every teacher of psychiatry has felt that it is possible to present the matter in a better way than has yet been done, but has been inhibited by the difficulty of the problem when he put his mind to its presentation.

The kernel of psychiatric presentation is in the description of the major psychoses. This differs little from one book to another, and in each instance is essentially a longer or shorter abstract of the Kraepelinian classification of mental disorder on the descriptive level. This, in itself, is a simple text without much chance for inspired reading. The author of the book under consideration devotes something under 200 pages of a total of 447 pages to this particular portion of psychiatric presentation. In doing so, he has tried, and probably very successfully, to offer the material in more or less classical style, utilizing and combining the Kraepelinian and Meyerian nomenclature, as well as some psychoanalytic verbiage. Illustrative case material is interspersed with the descriptions. Here again, one finds, as is characteristic of all textbooks, that the case presentations lack the interest that a living case affords. Only the inspired dramatist would be able to present psychiatric cases in an entrancing and gripping fashion.

But, as already suggested, Henry has attempted much more than a synopsis of the clinical syndromes. Due to his experience of having written a text on psychopathology, he is well-equipped to present a variety of discussions and concepts about personality in the normal and in the abnormal. Unfortunately, with space limitations, nothing more than the essence of the subject can be presented.

The author has tried to give this material in a somewhat judicious fashion, apparently not wishing to represent one school of thought. One recognizes a leaning toward psychoanalytic formulations and an effort not to be dogmatic. Here again, one meets the difficulty of this type of presentation in that it is neither "fish nor fowl." As a result, one can readily make plenty of criticisms, partly explained by the brevity of the

presentation that does not allow ample room for the development of the ideas, and partly explained by the attempt to give a broad presentation. For example, in his opening paragraph, he states that "A clue to the understanding of these disorders may be obtained from the general principle that whenever an individual is unable to adjust to stress, he tends to revert to an earlier and less-complicated form of adaptation." Perhaps one agrees with this, or perhaps one has doubts as to its validity. He attempts to express his point of view as to the difference between psychopathy, the psychoneuroses, and the psychoses as follows: "The psychopaths cling to juvenile modes of behavior and the psychoneurotic evades adult responsibilities through the development of symptoms, but the psychotic reverts to youthful and even infantile sources of pleasure." Such a statement would appear to be an over-simplification. He does add correctives, to be sure, to his simplified explanation. For example, he says, "However plausible these explanations of personality disorder may be, the specific causes in individual cases remain obscure."

One could quote at some length from this type of simplification of the problem, and one could form his own opinion as to whether the statements are proven facts or mere speculations. However, one must grant that if read carefully, there is much value in the challenges that are offered.

Thirty-three pages are devoted to a consideration of the principles of treatment. Without doubt, the average physician not trained in psychiatry, and the medical student find their chief criticism of psychiatry and psychiatric texts in the fact that so little definite information concerning methods of treatment are offered. Certainly, 33 pages does not allow sufficient opportunity for any fundamental development of treatment methods. Henry has given brief descriptions here of the Freudian method, concerning which he concludes: "As a method of therapy it is still in the experimental stage"; of the Jungian psychoanalysis; of the Adlerian viewpoint. He discusses briefly suggestion, hypnotism, persuasion, reeducation, surgery, intellectual diversion, occupational therapy, hydrotherapy, electro-therapy, massage, seclusion and restraint, as well as prevention of suicide. From this list of the subjects discussed, it is self-evident that not very great enlightenment can be given as to the various techniques. While the reviewer believes that this chapter is very inadequate, he would offer the defense that nowhere has a much more satisfactory discussion of treatment of psychiatric conditions been offered.

A chapter of equal length follows on psychiatric nursing, and while this chapter contains a variety of interesting advice, rules and methods of procedure, again, it is probably insufficient in itself and takes up space that could be well devoted to other subjects.

Chapters on the psychopathology of the normal, mental hygiene, on the disorders of childhood, psychiatric social service, and the medico-legal aspects of psychiatry follow in this order. Again, the criticism seems valid that the attempt to cover so much ground in such a short space detracts from the general value.

One of the most interesting chapters in the book, and probably one that serves an important place in psychiatric propaganda, is the chapter entitled "Psychiatry in General Hospital Practice." This chapter is, in the reviewer's opinion, really very well done. Brief thumb-nail sketches of cases occurring in a general hospital, with psychiatric components or basic psychiatric problems are presented and call attention to the frequency with which such conditions occur without being readily recognized in ordinary general hospital practice.

The title of the book *Essentials of Psychiatry* indicates what the author has attempted to do, namely, to give some of the essence of this wide and diversified art. In any attempt to cover the wide field, certain of the defects mentioned are almost unavoidable. One regrets that no mention is made of insulin and metrazol therapy, or of electro-encephalography, or other pharmacological approaches such as continuous sleep methods. It is also unfortunate that there is not more discussion of the various investigations that are being undertaken on a physiological level, because whether these turn up any important material or not they nevertheless take up a large block of psychiatric thinking.

With all these criticisms, one must end the review by again stating that the book has great merit; that it presents a variety of angles of psychiatric work in clear, concise English; that it rather skillfully avoids taking too strong a stand on any one point of view; that it is easy reading, and offers a relatively comprehensive picture of psychiatry.

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THE INTELLIGENCE OF PRE-SCHOOL CHILDREN AS MEASURED BY THE MERRILL-PALMER SCALE OF PERFORMANCE TESTS. By *Beth L. Wellman, Ph. D.* (Iowa City: University of Iowa Studies in Child Welfare; Vol. XV, No. 3, 1938.)

As Dr. George Stoddard, editor of the series, states in his foreword, this monograph represents "the most intensive study of the Merrill-Palmer scale since Dr. Stutzman's original presentation." The data on which it is based include the results of 510 testings of 281 children of pre-school age. Practice effects commonly noted when the Merrill-Palmer scale is given repeatedly to the same subjects are effectively differentiated from changes in scores indicative of something else—which Dr. Wellman identifies with "increased brightness." The latter phenomenon is associated, by the author, with attendance in the pre-schools of the Iowa Child Welfare Research Station, and is shown to be unrelated to occupational or educational status of the parents. Certainly, the experimental data justify the conclusion that "pre-school attendance materially affected ability on the Merrill-Palmer test"; and that "the changes in Merrill-Palmer ability were less clean-cut than in Binet ability," (p.77).

Comment upon the significance of this monograph to psychological theory is undoubtedly out of place, unless a careful review were undertaken of the

large body of work for which Dr. Wellman is justly famous. The monograph fits perfectly into the scheme of research which Dr. Wellman sponsors; and the reviewer understands that her point of view—which may be symbolized by the phrase "the nurture of intelligence"—will shortly be examined and appraised in a forthcoming year-book. At the risk of anticipating a matter that will doubtless be discussed at that time, the reviewer would like to suggest caution in interpreting experimental data of the kind reported in the present monograph. It would be distinctly unwise if the stimulating, painstaking and discerning work of Dr. Wellman were to be regarded as representing a return to earlier conceptions of the I. Q., with the simple revision that the constancy of the I. Q. is now being called into question. Educational psychology of a former era undoubtedly over-stressed the interpretation of "intelligence" in terms of a substantial entity, possessed by an individual to a degree specified by a supposedly fixed quantity. But later developmental psychology turned its attention to the dynamics of growing up, of maturing, of educational procedures, of personality organization. To be informed by experimental data that "the I. Q." can be raised, might entrench a type of research in which the understanding of such terms as "stimulating environment," "developmental needs," "educational values" would tend to be secondary to the mere artifacts of I. Q. measurement. This, as Dr. Wellman's whole body of research shows, would be as unwise as it is contrary to the very point of view which directs her whole work.

The monograph is one of the most stimulating and significant productions of modern psychological research. Adequate interpretation, however, must await a careful research programme initiated for that purpose.

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Behaviorism at Twenty-Five. By *A. A. Roback* (Cambridge, Mass.: Sci-Art, 1937.)

As the title of this volume implies, the author has seized the occasion of the twenty-fifth anniversary of Watson's most famous statement to write about Behaviorism. In a preface, somewhat lacking in form, we are led to understand that the time is ripe for some form of accounting in regard to the sweeping promises made by Watson 25 years ago, the political analogy being invoked as justification for such a contention. At the same time, the author makes it clear that he is a member of the opposition, at least to the extent that he confesses thus: "If I have become a convert to a doctrine that seemed at first far-fetched and fantastic, it is to the fundamental tenets of psychoanalysis, which, if it does not adhere to the rigid canons of logical thinking, operates in the sphere of the unconscious (neurosis, dreams, lapses), where reason is conspicuously absent (*e. g.*, duality, conflict, ambivalence)." He adds that "unlike psychoanalysis, behaviorism makes pretensions to dealing with observable and tangible facts open to experimental analysis and productive of tangible results." "In that domain," he contends, "behaviorism has been wanting."

The book might, therefore, be entitled "Behaviorism found wanting"—save for the fact that such a title would indeed place an obligation on the author that he should be unwilling to assume at the present time. Nevertheless, there can be no question about the author's opinion, which is, and always will be, that behaviorism never had (or could have had) a chance! It was wrong at the start, and therefore always will be.

An objective appraisal of what behaviorism has achieved to date would have been interesting. A thoroughly systematic critique of the tenets of behaviorism would have been valuable. Either or both of these could legitimately be expected from the title of the book, and from knowing who the author is. The reader will find neither in the book under review.

Part I shows that Watson was not original; takes issue with the behavioristic denial of instinct; ridicules the identification of thinking with speech-habits; questions the effectiveness of behaviorism in directing the education of young children; and while giving some credit to the objective point of view for assisting psychological progress in recent decades, denies that behaviorism had much to do with this.

Part II revives the Hunter-Roback controversial correspondence, and draws on letters from Titchener, Thorndike, Angell, Judd, Woodworth and Warren to show that these leaders were not as forthright in their espousal of behaviorism as was presumably supposed by Hunter.

Part III discusses some aspects of behavioristic experimentation, lists some of the outstanding investigators who are to varying degrees related to the objective movement, and playfully examines a few features of Watson's autobiography.

A bibliography of some of the main contributions to behavioristic literature since 1922 (a continuation of that contained in the author's *Behaviorism and Psychology*) completes the book.

Positive features of this book are hard to discern. If the reader is a rabid anti-behaviorist, he will derive some satisfaction from the constant belittling of Watson's contribution. If he is imbued with the faith in objectivism as a means of attacking psychological problems, he will be irritated or disdainful.

The reviewer must confess to being disappointed in the treatment given. The postulates of behaviorism may be (as the reviewer believes) frankly unsuited to the scientific investigation of many significant psychological problems by which statement one implies an incompatibility between those postulates and what one considers significant. But that could be made clear only by careful exposition of the bases of systematic enquiry. Similarly, an evaluation of the achievements of behaviorism to date would involve an understanding and (for purposes of the evaluation) an acceptance of the behavioristic postulates; and thence a survey of the results of such an acceptance on the part of purposeful investigators. Neither of these major issues can be clarified by mere polemic, even if good-humoured, such as characterizes Roback's treatise.

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EMOTIONAL HYGIENE—THE ART OF UNDERSTANDING. By *Camilla M. Anderson, M. D.* (Phila.: J. B. Lippincott Co., 1937.)

The book is attractively written, with an introduction by Dr. Ross McC. Chapman and cartoon illustrations by Dorothy G. Stevenson. It is divided into three parts. The first part is devoted to the biologic and social bases of behavior. Here, the author presents in a simple and direct fashion the biological and psychological stages of human development. The intrauterine existence, the phases of infancy and childhood, together with the psychosexual levels of development are discussed. The possibilities of fixations and the natural tendencies toward growth in the right direction are illustrated by brief and well chosen clinical communications. The explanation of psychoanalytic concepts is too condensed. A great deal of information is contained in these first chapters.

The second and third parts are directed to members of the nursing profession. The second unit on personality and adjustments contains pertinent advice for any young person who is beginning life away from home. Acquiring the psychiatric point of view in the care of all patients is encouraged by the use of apt illustrations. The advice to those who are having difficulties in adjusting to their associates and superiors is sound.

The third part, dealing with the emotions in relation to special fields, begins with a short history of psychiatric nursing. An optimistic note is sounded when our attention is called to the many and varied possibilities of professional advancement in the field of nursing.

This is a stimulating and worthwhile book. It should be read by all nurses in training, and by all persons who wish to attain the level of "understanding rather than judging."

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BASIC PROBLEMS OF CRIMINOLOGY. By *Professor Olof Kinberg, M. D.* (Copenhagen: Levin & Munksgaard, 1935.)

The current writings on criminology are so prolific and variegated that it is necessary when looking at a new publication, to ascertain first the viewpoint of the writer. Is he writing about crime and criminals as a lawyer, interested chiefly in defining crimes and criminal behavior; or is he writing as an administrator or a penologist; or as a laboratory expert, qualified in police science; or as a sociologist, or an economist; or as an anthropologist, defining the physical characteristics of the criminal; or as a psychologist or psychiatrist, interested in the causes or correction of abnormal behavior?

Professor Kinberg is a psychiatrist with many years of experience in the mental examination of criminals. His *Basic Problems of Criminology* belongs in that category of criminological literature which deals with the application of psychiatric methods to the field of crime, but its scope is some-

what wider. The author states (page 11), "I therefore decided to widen my scope, to try to give at the same time an account of all the more important present day aspects of the criminality problem."

At the outset (chapter 2) the author points out the inadequacy of contemporary legal tests for criminal responsibility. These tests tend to be tests of intellectual capacity. Their weakness lies in their failure to measure emotional deviations which are so often the most striking feature of the mental illness.

Several chapters are devoted to "imputability," or legal responsibility for crime. The author does not shrink from a thorough exposition of fallacies of the doctrine of free will. He is clearly of the opinion that a scientific treatment of criminals must assume a psychological determinism. A great deal of space is occupied by this kind of philosophic speculation. Without detracting from the importance of a matter so fundamental, it is thought that some readers may be deterred by the abstruseness of these early chapters from reaching the many valuable observations based on the author's own experience contained in the latter part of the book.

Professor Kinberg advocates the adaptation of punishment to the individual nature of each criminal (chapter 5). In most jurisdictions the penalty is based upon the nature of the crime. The idea of adapting the penalty to the nature of the criminal, particularly to his dangerousness, is a revolutionary departure from tradition. In many jurisdictions the safeguards in the criminal law represent a long struggle against an authoritarian regime and are properly regarded as a valued safeguard of the individual against oppression or tyranny. The surrender of such safeguards is one of the chief objections to the plan propounded by the author. In *Crime and Justice*, Professor Sheldon Glueck has advocated this same plan and has considered in detail many of the difficulties which would arise in working out such an innovation.

In the part of the book dealing with causes of crime (chapter 8) there is an interesting summary of the available methods of determining the causes. The author sees a difference between the European and American attitudes towards criminological research, (page 167),—"One might perhaps venture the assertion that the tendency of European criminology is rather towards the individual, endogenous theory of criminality, while Northern American criminologists are more inclined to see its chief causes in social factors." In the pages following this quotation, is an explanation of the tendency of European criminologists to look for the causes of criminality chiefly in individual traits of criminals and the inclination of Amercan criminologists to see them chiefly in factors of environments. Consideration is give to the relative importance of inherent and environmental factors in the production of criminal behavior. The author has this to say of the "born criminal" (page 217), "Every practical criminologist knows that there are individuals in whom the natural tendency to asocial or anti-social acts is so strong that—whatever the environment in which they find themselves—they will always commit crime. This type of criminal is by Lombroso called *rei nati*, "born criminals."

Evidence is furnished that many insane criminals are tried and sentenced without any recognition of the mental disorder. The data gathered by the author at the Forensic Psychiatric Clinic of the Central Prison in Stockholm offers substantial support for his contention. It is estimated that of 238 prisoners who had received the full sentence, at least 39.5 per cent should have been declared exempt or had their sentence reduced. More striking, however, than the statistics are the cases cited. It is truly remarkable to read some of these case histories and to note the marked degree of insanity which could exist without attracting any interest or attention in a criminal court. Imposing a term of imprisonment in the case of an insane prisoner is not only an injustice to the individual, but what is even more important when the sentence is served he is liberated without regard to his future dangerousness to society.

What can be done to remedy this situation? The author states (page 374), "The proposal for the obligatory examination of certain accused persons, even as comprehensive as that suggested in 1908, is now out of date. It is now perfectly clear that the demand for a mental examination of every accused person can no longer be dismissed." The reader will find in chapter 11 an account of what has been done in this field in some European countries and in the United States.

In this connection it is of interest to read that (page 394), "Fortunately, a Central Criminological Archive was established at the Stockholm Forensic Psychiatric Clinic in 1934 by private initiative and with financial assistance from the Rockefeller Foundation for the collection of the reports of all cases in the country where mental examination of the prosecuted has been ordered. This material will be supplemented and prepared for statistical use in order to serve scientific and practical purposes."

Basic Problems of Criminology is an authoritative and engaging volume, filled with careful criticism of existing procedure and suggestion for reform. The views expressed are as international as the problem of crime. The language is somewhat stilted, perhaps because of a too literal translation, and the addition of an index would be useful. These are superficial matters; the substance is an admirable justification of the title.

K. G. Gray, M. D.,
University of Toronto.

The American Journal of Sociology. (Special issue). (Chicago: University of Chicago Press, 1939).

This journal, which has just completed its 44th year of publication, devotes the final issue of the present volume to a symposium upon "The Relation between the Individual and the Group." There are presented 12 contributions by leaders in the fields of psychiatry, psychology, sociology, cultural anthropology, and the symposium is international in character.

The problem posed is basic to all the social sciences and the editors express the hope that by bringing together in this way the viewpoints

of representatives of the separate disciplines something may be accomplished toward reconciling prevailing diversity of opinion.

Contributors to the symposium are: Florian Znaniecki of the University of Posznan, who discusses "Social Groups as Products of Participating Individuals." He points out that the objects of study are not individuals or social groups as such, but rather the data of human experience which, expressive of the group, are cultural products with definite historical patterns. Maurice Halbwachs, professor of sociology of the University of Paris: "Individual Consciousness and the Collective Mind," exemplifying the relations between the individual and collective consciousness on the basis of Durkheim's teaching. William E. Blatz of the University of Toronto: "The Individual and the Group" describes researches in social behavior by means of observation of the Dionne quintuplets. John E. Anderson of the University of Minnesota: "The Development of Social Behavior." In this contribution studies in child development reported in the literature are summarized under the headings (1) Social organization, (2) Social attachments, (3) Motivation within the group, (4) Differentiation of functions within the group. J. F. Brown of the University of Kansas: "Individual Group and Social Field." The author presents the theory of the "social field" as a corrective of the misleading antithesis between the individual and the group. Kurt Lewin of the University of Iowa: "Field Therapy and Experiment in Social Psychology." The field theory is further elaborated to demonstrate the interdependence of physiological, psychological and sociological data in determining social behavior. Floyd H. Allport of Syracuse University: "Rule and Custom as Individual Variations of Behavior Distributed upon a Continuum of Conformity." The author attempts to replace such vague concepts as folkways by more precise methods of estimating the degrees of conformity of individuals in interpreting social behavior. Thomas M. French of the Chicago Institute for Psychoanalysis: "Social Conflict and Psychic Conflict." An analogy is drawn between individual psychic conflict and social conflict as reflected in mass delusion and phobia. Harry Stack Sullivan of the William Alanson White Psychiatric Foundation: "A Note on formulating the Relationship of Individual and the Group." The author points out that since human life is expressed in such large part in interpersonal processes, these rather than the person as an isolated individual are fundamental to the study of human behavior. Bronislaw Malinowski of the London School of Economics: "The Group and the Individual in Functional Analysis." In evaluating the impingement of culture upon the individual the former must be analyzed in terms of its institutions, such as the economic, educational and political aspects of the social pattern all of which contribute to the conditioning of the individual. Lewis Wirth of the University of Chicago: "Social Interaction: The Problem of the Individual and the Group." In this closing contribution the Chicago sociologist reviews the papers constituting the symposium, and indicates the necessity of a closer rapprochement of the several disciplines dealing, each in its own way, and by its own techniques with the problem of human behavior in its group relationship.

Toward such a rapprochement The American Journal of Sociology makes its contribution by presenting this noteworthy symposium. This special issue is obtainable separately.

C. B. F.

AMERICAN FOUNDATIONS AND THEIR FIELDS, IV. Compiled by *Geneva Seybold*. (New York: Raymond Rich Associates, 1939.)

This report is the fourth of a series, the first three for the years 1930, 1931 and 1934 having been compiled and published by the Twentieth Century Fund. This Foundation turned over its records to the Raymond Rich Associates who have published the present issue.

The book is an amazing record of philanthropies in various fields of human activity made possible by the establishment of self-perpetuating foundations by wealthy individuals and agencies. The total capital of foundations represented in this study exceeds $955,000,000; and this huge sum does not constitute the total amount from which public benefaction can be derived, since many of the smaller funds issue no public reports and data concerning them are not available.

In this record will be found information concerning 243 foundations located in 30 states and the District of Columbia. The headquarters of 116 of the foundations are in New York State, and of these 106 are located in the city of New York. The wealthiest of these bodies is the Rockefeller Foundation with a ledger value capital (1937) of $184,435,515.93. The largest total grant for the same year was paid by The General Education Board which disbursed $9,716,277.15, or approximately 25 per cent of total grants reported by the foundations for 1937.

There are interesting tables showing the number of foundations in each field and the amounts of grants paid in the several fields. The largest number of foundations (63) is in the field of education; medicine and public health come next with 62 foundations; then social welfare with 56; child welfare 32; aesthetics 26; social sciences 20; religion 17; physical and biological sciences 15; with 12 other fields represented by foundations ranging in number from 9 to 1.

The fields are arranged in a somewhat different order with respect to the total amounts paid in grants. Approximately 35 per cent of total disbursements for 1937 went into the field of medicine and public health; education followed with approximately 24 per cent; social welfare 12 per cent; physical and biological sciences 6 per cent; and the remaining fields trailing down.

The book contains a directory of American foundations alphabetically arranged with all pertinent data concerning each, together with financial statements and details of grants in the several fields.

Those who rail indiscriminately at the capitalist system might profit from acquaintance with the substance of this report.

C. B. F.

CORTICAL SPREAD OF EPILEPTIC DISCHARGE AND THE CONDITIONING EFFECT OF HABITUAL SEIZURES.*

By WILDER PENFIELD and EDWIN BOLDREY, Montreal.

A. Review of cortical localization of simple somatic motor and sensory representation.
B. Atypical responses in selected cases.
C. Localization of initial phenomenon in epileptic discharge.
D. Discussion.
E. Conclusions.

The cerebral cortex of the epileptic patient ordinarily gives elaborate response to electrical stimulation but there occur at times marked fluctuations of stimulability varying from a completely refractory state to marked activation and hyperstimulability. At times there may be responses at a great distance from the accepted cortical representation. This paper contains the most atypical cases of stimulation of the epileptic cortex set aside over a period of years to await analysis. We believe that these bizarre results may now cast light upon the mechanism of spread during an epileptic seizure and perhaps upon the nature of cortical conditioning.

The present study includes the analysis of 200 craniotomies in 165 of which the cortical stimulation was satisfactory. The patients were operated upon under local anesthesia and during the operation a report of the nature of each positive response was dictated. Small numbered or lettered tickets were dropped upon the surface of the brain to mark the site of the positive responses. Photographs were made at operation to give accurate detail. Furthermore, before the tickets were removed the operator copied their position on a standard chart such as is shown in Fig. 2.

In most of these cases the diagnosis was focal epilepsy. The clinical results of such operations up to the year 1935 have already been published completely (Penfield 1936) and will not be discussed here.

* Read before the ninety-fourth annual meeting of The American Psychiatric Association, San Francisco, Cal., June 6-10, 1938.

From the Montreal Neurological Institute, and Department of Neurology and Neurosurgery, McGill University, Montreal.

A. Review of Cortical Localization of Simple Somatic Motor and Sensory Representation.

Somatic motor and sensory representation in the cerebral cortex has a practically invariable order (Fig. 1) from above downward in the Rolandic (pre- and postcentral) convolutions (Penfield and Boldrey, 1937). In this representation the distance of any one

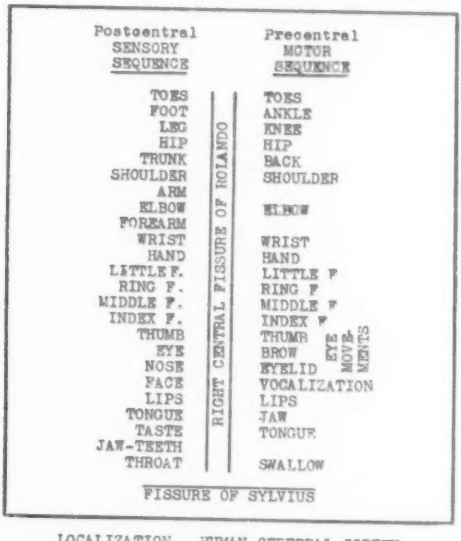


Fig. 1.—Sequence of motor and sensory phenomena on the human cerebral cortex, right side.

element from the fissure of Sylvius may vary greatly from case to case as shown by the scattering of the dots in Figs. 2 and 3. The distance of responsive points from the central fissure of Rolando may vary considerably also but the fact that the order of responses from above down remains constant as listed in Fig. 1 indicates that there are upon each lip of the fissure separate foci for motor and for sensory representation arranged in series. It seems probable that the neurone connections of each of these foci pass out from the fissure in a parallel arrangement over the pre- and postcentral convolutions.

B. Atypical Responses in Selected Cases.

From this series of craniotomies certain unusual cases have been selected and will be described below. Each of these unusual cases has been rechecked and when doubt has arisen as to accuracy of record the case has been eliminated.

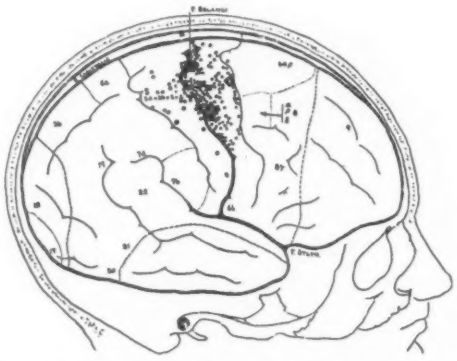


Fig. 2.—Movement of hand, arm and shoulder (from Penfield and Boldrey, 1937).

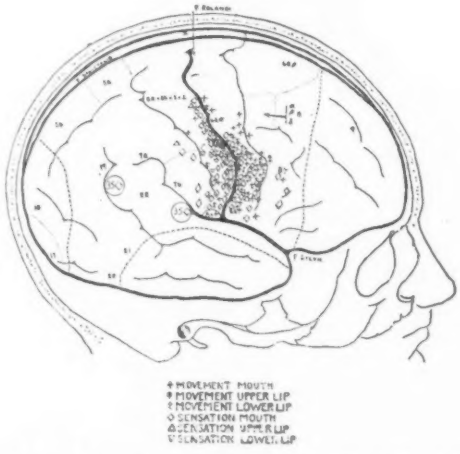


Fig. 3.—Movement and sensation of the mouth (from Penfield and Boldrey, 1937).

Case 1. *Large Cerebral Lesion, Displacement of Aura by Facilitation.*—J. H. A young man aged 29 had had hemiparesis of arm and leg from a birth ischemia (Fig. 4). A large cyst had replaced cerebral tissue along the upper side of the fissure of Sylvius. Evidently a small portion of the lower end of

the Rolandic cortex had survived and lay below the cyst (A K and I Fig. 4). Motor and sensory responses for arm and leg were found about where they might be expected. Face movements and speech were apparently subserved by the opposite hemisphere alone, as a wide excision produced no interference with these functions.

The patient suffered from two types of seizures neither of which developed into the other. The first type, which he called "minor," was characterized by a sudden sensation up the back, back of his neck and head. This aura was reproduced quite accurately by stimulation at points A, K, I and J in Fig. 4 (see also Fig. 5). The second type of seizure he called "major." This was ushered in by blinking of the lids and conjugate jerking of the eyes and was

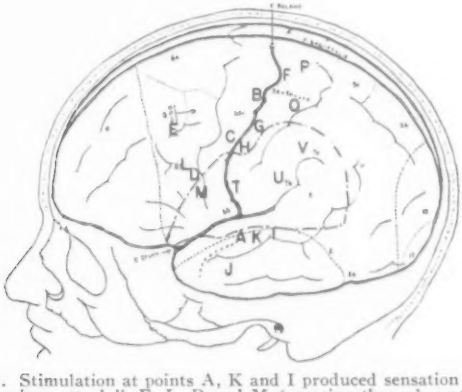


FIG. 5.—Case 1. Stimulation at points A, K and I produced sensation in head, neck and back "very like a minor attack." E, L, D and M (pressing through cyst to cerebral wall) produced fluttering of eyelids and movements of eyes "like what comes before a major attack." E—tingling of right hand. G—sensation in upper right arm. C—extension of hand. B—flexion of wrist and elbow. F—flexion of right leg at knee. O—sensation in right leg. P—sensation in right leg nearer foot.

characterized by clonic convulsive movements beginning in the right extremities. The prelude to a "major" was reproduced repeatedly at E, L, D and M. E seemed to be situated in the area of cortex where conjugate eye movements are ordinarily represented and probably is a true localization response. L, D and M on the other hand are examples of displacement of the initial motor phenomenon of a fit. This displacement may be called response from a distance.

Facilitation.—The minor aura which could be produced at A, K and I could not be produced initially on that same gyrus elsewhere. But by means of repeated advancing stimuli which started at A and were repeated once every second, the response of the aura occurred at every stimulus until the electrode had been carried along the previously silent gyrus to a distance of 4 cm. The dots in Fig. 5 indicate the course of the advancing stimuli over what seemed to be temporal lobe. When this was repeated and a fissure was crossed the responses stopped at the fissure. This is an example of extension by facilitation of a sensory response that had become a specialized epileptic aura.

CASE 2. *Motor Paralysis, Sensory Distant Response.*—A. M. This patient suffered from left-sided Jacksonian seizures. During the period of investigation an unusually severe seizure left her paralysed in the left arm and leg. After this paralysis had continued for eleven days, osteoplastic craniotomy and exploration were carried out. No objective lesion of the brain was found. Her course in the years that have followed bears out the diagnosis of idiopathic epilepsy.

As is stated in the legend that accompanies Fig. 6, stimulation produced no motor response except in the face at point 1. Sensory response did occur but only when a strong stimulation of from 9-10 milliamperes galvanic current

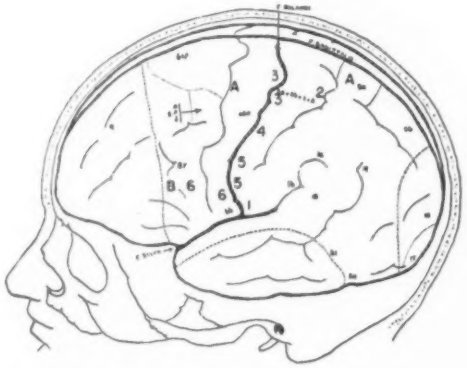


FIG. 6.—Case 2. 1—Twitching of right side of mouth 10 m.a. (m.a.=milliamperes). 2—Tingling of R. hand 10 m.a. 3—Same as 2. 4—Tingling of R. hand and tongue 10 m.a. 5—Same as 4, but side of tongue. 6—"Pins and needles" felt in right side of body 9 m.a. A—Tingling in right thigh. Faradic stimulus. B—"Pins and needles" in right shoulder, arm and hand. Faradic stimulation.

Note that point 1 is the only motor response in this patient.

was used. There was some tendency to distant response of cortical sensation; for example stimulation at 6 (Fig. 6) produced sensation in the right side of the body when a galvanic 9 m. a. current was used and stimulation at A and B also produced sensory response but no motor phenomena.

To summarize, this is an example of true motor paralysis, not a state refractory to electrical stimulation. Moreover this sensory response from a distance may be due to spread of the current which was comparatively strong and not to a vagary of neuronal conduction as in the subsequent cases.

CASE 3. *Motor Refractory State, Sensory Activation, Sensory Distant Response.*—W. O. This patient had an arteriovenous aneurysm within the substance of the left frontal lobe (Fig. 7), and an underlying cyst also within that lobe. He complained only of epileptiform seizures which consisted of sudden inability to speak followed by turning of head and eyes to the right and convulsive movements of the right arm and leg. There were no abnormal neurological findings and the two arms were equal in strength.

The operative stimulation record corresponding with the tickets on the brain in Figs. 7 and 8 is given in detail in the legend for Fig. 8. The numbers in parentheses show the strength of thyratron stimulation—higher numbers indicating a weaker current and lower numbers a stronger current.

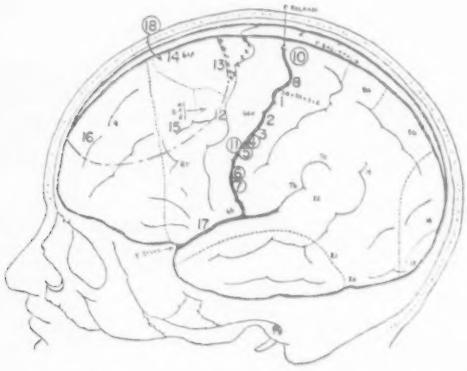


FIG. 8.—Case 3. Dotted line indicates abnormal area removed at this operation and line of crosses the area of removal at second operation. Stimulations carried out in order of 1 to 18. All stimulations produced some sensation in right arm or hand except 10, 5, 11 and 18.

(28) 1—"Electric shock" felt in right arm.
(32) 1—Repeated at strength 32 with no response.
(28) 2—Electric shock "went down into my hand. The other one did not go into my hand." Slight extension of fingers. "That is not the same as the feeling I have at the time of my attack."
(28) 3—Feeling in arm and hand. "You see, when I am going to have an attack the first thing is that it stops my speech."
(28) 4—Sensation in index finger and thumb, also arm; no movement.
(28) 5—Sensation in right shoulder and side of face.
(28) 6—"Same place—right side of my face, right shoulder and partly down my arm."
(30) One negative stimulation.
(30) 7—Slight sensation in right side "a little in my arm also."
(30) Three negative stimulations.
(28) One negative stimulation.
(28) 8—"Electric sensation in my right arm."
(28) 10—Patient hesitated—a little later said "I feel as though I am going to have an attack. I feel just a little dizzy."
(28) 11—Marked twitching of right side of face, mouth drawn downward and slight vocalization.
Two negative stimulations.
(28) 12—"Slight electric shock down my right arm."
(28) 13—Slight electric shock in right arm.
(28) 14—Slight electric shock in right arm and hand.
(24) 15—Shock in right arm, hand and shoulder.
(24) 16—"Another shock in my right arm and hand."
(24) 16—Repeated without warning "Shock in my right arm and hand."
(20) 17—Right arm and hand. Repeated without warning. "In an attack my hand begins to shake without this electric feeling."
One negative stimulation.
(20) 18—Shock in right leg from knee down. Continues to complain of numbness in right leg following this.

The precentral gyrus was stimulated repeatedly but reference to Fig. 8 shows that there were no motor responses except at 11 which gave twitching of face and slight vocalization. On the other hand the postcentral gyrus

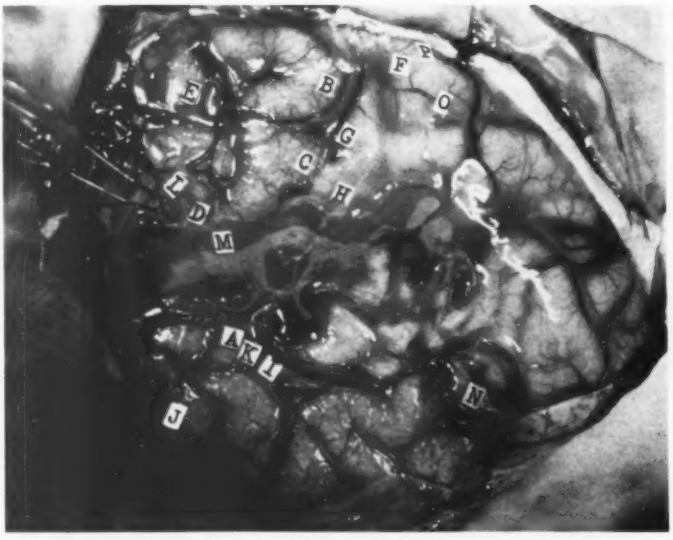


FIG. 4.—Case 1. Operative photograph. Focal destruction of brain by occlusion of middle cerebral artery either natal or prenatal. Letter tickets placed on brain after each positive stimulation correspond with those in Fig. 5 and are explained in that legend.

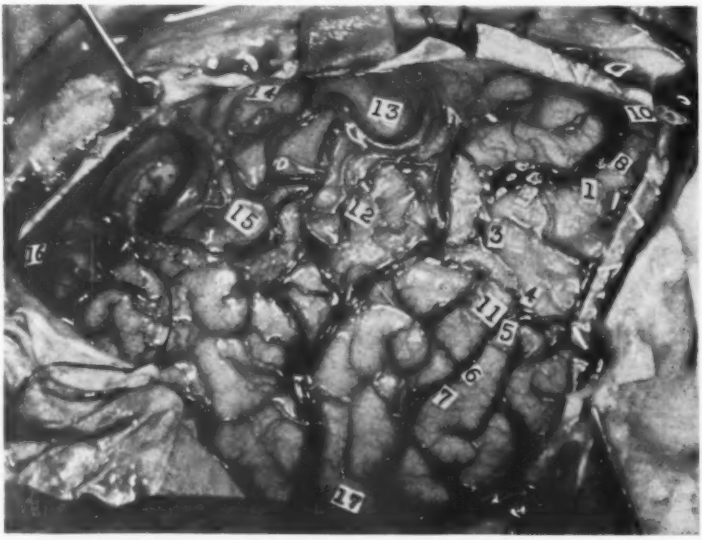


FIG. 7.—Case 3. The left frontal lobe at operation showing arteriovenous aneurysm. The tickets of stimulation correspond with those in Fig. 8.

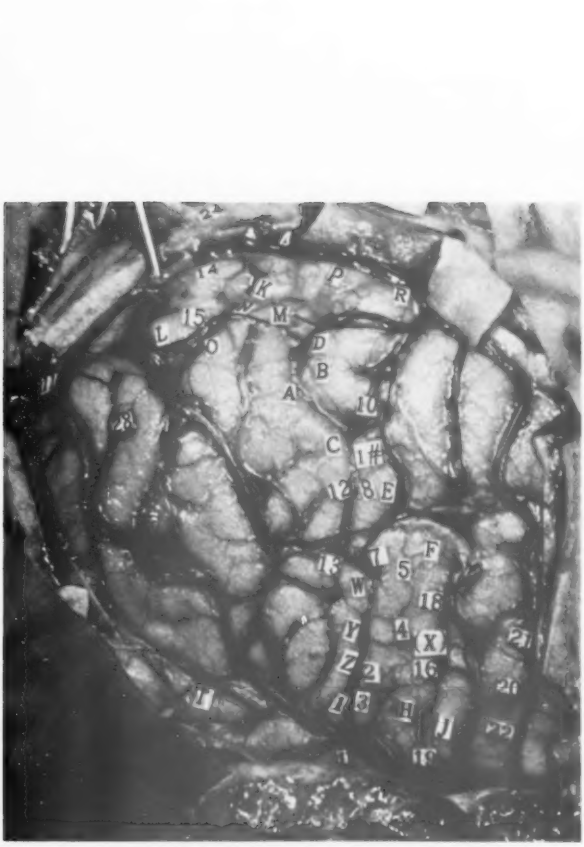


FIG. 12.—Case 7. Epileptic patient in whom no objective lesion was found. The tickets were placed when response followed stimulation, letters being used first and then numbers in sequence. Some of these responses are reproduced in a diagram in Fig. 13.

was invariably responsive to a weak stimulation* and there were extraordinary distant sensory responses. Every response in Fig. 8 except those numbers in circles consisted in "an electric shock" in the right arm or right arm and hand. At 4, 6 and 7 he reported other sensations but he added that there was also sensation in the right arm.

No attack was produced and no effort was made to produce one. Stimulation at 10, however, caused arrest of speech as in a typical attack and after hesitation the patient said he felt as though he were going to have an attack. The arm sensation, however, was not considered by him to be a part of his seizures. This was evidently therefore a true sensory response produced by stimulation at a great distance. Care was taken to check the truth of his reports. Points 16 and 17 were restimulated silently and with no warning but he reported the same sensation as soon as the electrode was applied.

After a series of stimulations (12, 13, 14) which resulted in arm sensation in each of which he was warned at the time of stimulation by the word "now," he was told that he would be stimulated again but no stmiulus was applied. At the word "now" he said "I did not feel hardly anything, just a small electric shock down my right arm." This response is of psychological interest whether true or false. If he really felt it, this was an example of rapid conditioning to an auditory stimulus.

Stimulation well down in the longitudinal fissure at 18 produced the sensation of shock in the right leg although the location of stimulation was well precentral.

This distant stimulation presumably had its effect upon the postcentral gyrus by neuronal conduction along connecting nervous pathways. The only alternative to this explanation would seem to be spread of current across the brain to that gyrus. This seems unlikely for two reasons: First that the current was so near the threshold value that decreasing it from 28 to 32 had abolished the response even when the postcentral gyrus was stimulated directly; and secondly, stimulation of the precentral gyrus immediately anterior to the sensory arm area produced no response, either motor or sensory.

The postcentral gyrus was hypersensitive. Both arm and leg sensations were produced by distant stimulation. These are the parts of the postcentral gyrus nearest the aneurysmal lesion. There seemed to be no hypersensitivity of the face area below but stimulation here produced arm sensation as well as the anticipated face sensation. The precentral gyrus seemed to be less responsive than normal to electrical stimulation and yet the patient had what was believed to be normal control of his extremities and normal deep reflexes. He had had no recent seizure.

Although the arteriovenous aneurysm was removed successfully "en bloc," as shown by the broken line in Fig. 8, the seizures continued. Consequently a second operation was carried out upon this patient eleven months later to remove a small nubbin of scarred tissue which had been left adjacent to

* 28 on this thyratron stimulator, an instrument which was described by Schmitt and Schmitt, Science, New Series, 76, 328, 1932 is quite weak, often failing to give any response in the normal human cortex.

the precentral gyrus (the area surrounded by a line of crosses in Fig. 8). At the time of the second procedure the precentral gyrus was quite as responsive to electrical stimulation as the postcentral gyrus. There was no evidence of spread of any sort. Both operations were carried out under local anesthesia, and the second has been followed by freedom from attacks.

In this case the initial incident in each fit was movement of the hand, not sensation. Nevertheless at the time of the first operation the precentral gyrus was refractory so that even the initial movement of his fits could not be reproduced and yet sensation of that same hand seemed to have a lowered threshold and to give distant response. Eleven months later, at the time of the second operation, the condition was changed and the precentral gyrus easily stimulable. This suggested that refractory state and distant response were transient phases only.

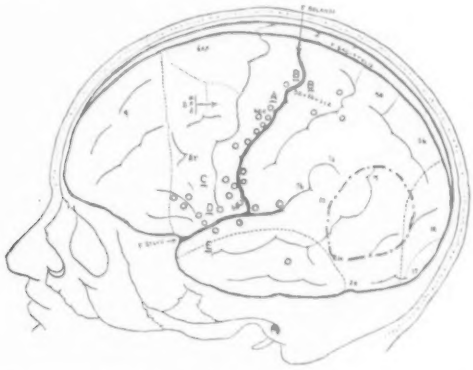


FIG. 9.—Case 4. Dotted line indicates site where deep infiltrating tumor approached surface. Small circles indicate points where stimulation gave no response.
Galvanic: A—Upward movement of right eyebrow.
Faradic: B—Closure of both eyes, repeated three times without warning but with the same result. C—Tonic closure of both eyes. D—Blinking of eyelids with wide opening of eyes. E—Blinking.

CASE 4. *Sensorimotor Refractory State, Isolated Activation and Distant Response.*—R. E. A woman of 34 years complained of recent headaches and seizures of 7 years' duration characterized by transient loss of memory and speech but with no twitchings or convulsive movements. She had no paralysis and had had no seizure for some days. She had been taking phenobarbital for several weeks but had none on the day of operation.

At operation a glioma was found deep in the posterior temporal region (indicated by broken line in Fig. 9). Exploration was carried out under local anesthesia but the neoplasm was not removed because of its situation near the speech area. Although the pre- and postcentral gyri were stimulated from the fissure of Sylvius up to within a centimeter or two of the longitudinal sinus no response was obtained except eye movements. These were obtained on the hemisphere (C, D, E, Fig. 9) and also high up where hand movements were anticipated (A and B).

In this case the motor and sensory centers were completely refractory but

the neoplasm did not produce any paralysis either before or after operation. At the same time a strange displacement of eyelid response occurred from the lower precentral position where it belonged up and down the Rolandic fissure. At one point brows were raised; at another lids were closed; at another there was blinking, and these results were rechecked with no warning.

Reference to Fig. 1 shows how many detailed responses one might have expected. For point "A" galvanic current was used and for the others faradic current. Perhaps the refractory state of all but eyelid and eyebrow movement caused the operator to use stronger currents than usual which spread across the refractory areas to reach the one responsive element.

Any effect that phenobarbital may have had would be expected to influence the eyelid movement as much as other elements. Also nearly every patient in the series of 200 was an habitual user of phenobarbital with no consistent

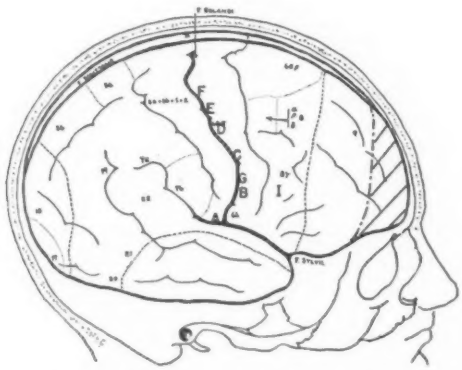


FIG. 10.—Case 5. Meningocerebral cicatrix anterior to dotted line. Thyratron at 24: A—Swallowing. B—Swallowing and lips to left. C—Closure of mouth and pulling of mouth to left. D—Closure of left eye (right not seen) and turning of eye to right. E—Clonic movement of mouth to left and down, also swallowing. F—Flexion of thumb and wrist. G—Mouth closed and drawn to left. Thyratron at 14: 1—Mouth movement.

depression of stimulability. There seems to be no adequate cause for the refractory state of the motor and sensory cortex unless the epileptic motor cortex does pass through a stage in which it is resistant to electrical stimulation but responds normally to volitional impulse.

CASE 5. *Sensorimotor Refractory State, Isolated Activation.*—S. E. This young man, aged 34 years, had been kicked in the head by a horse at the age of twelve. He was unconscious for from 12-18 hours. The only immediate sequelæ observed were dimness of vision in the right eye and a poor memory. At the age of 14 epileptiform attacks made their appearance.

Upon admission to our service the attacks were habitually ushered in by a "queer feeling." The patient's eyes then turned to the right and the mouth was drawn to the left. The whole body followed to the left, the feet remaining fixed. A generalized convulsion of a few minutes' duration followed.

An osteoplastic craniotomy was carried out under local anesthesia and a scarred area of right frontal lobe was removed. While the bone flap was

being turned there were two spontaneous small seizures characterized by turning of the head to the opposite side and swallowing movements. After the brain was exposed no seizure could be produced by stimulation in the vicinity of the lesion. (Seizures have never been produced by stimulation of the frontal pole.) As shown in Fig. 10 stimulation at E, C, G, B, A and I resulted in mouth movement or swallowing or both. Only at F were there thumb movements (at a point farther from the fissure of Sylvius than usual) and at D eyelid and eye movements. The rest of the frontal lobe and the lower two-thirds of the postcentral gyrus were stimulated without response.

The unusual feature of this case is that over an extent of at least one-half of the precentral gyrus only mouth and swallowing responses were obtained.

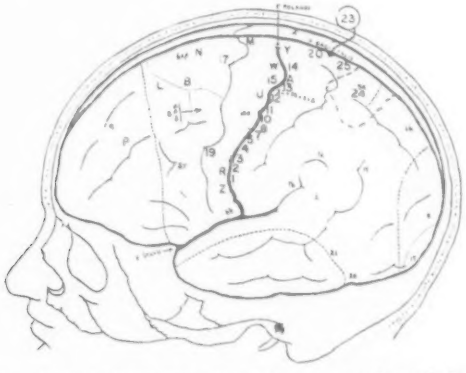


FIG. 11.—Case 6. Atrophic lesion indicated by dots. Aura of attack for this patient was sensation in right arm. A—Sensation in ring and little finger. 1-13—Sensation in right side of tongue, lips, face, fingers and hand in proper sequence. 14—Sensation in "my arm, it moved like when I take a fit only it did'nt go up." Y—Sensation in right arm "the way I get sick, and it started to move a little bit." Z—Sensation in throat. R—Mouth pulled to right. 19—Vocalization—repeated twice. U—Marked closure of hand—repeated. "It moved like when I have a spell." 15—Flexion of wrist and elbow. "Arm moved like in attack." W—Flexion of wrist and elbow, closure of hand. Sensation and movement "like when I am sick." M—Movement of hip. Repeated after interval. 17—Arm moved. N—Sensation in right arm like aura. B and L—Same as N repeated twice for each, once after long interval. P—"Yes I had the sensation, only my arm did not go up." Repeated after long interval without response. 20—Sensation in arm and a little in foot. 23 (in median fissure)—Sensation in thigh. 24—Sensation in arm like aura.

The current used was weak, *i. e.*, 24 on the thyratron. When strength was increased to 14 the mouth movement was obtained at I. There was obviously a lowered threshold for mouth stimulation and a raised threshold for other sensory and motor responses.

CASE 6. *Distant Response of Sensory Aura.*—N. I. This was the case of a boy of 16 who complained of Jacksonian seizures. His attacks began habitually with a sensory aura felt in the right arm followed by a jerking up of the arm. He had received an injury at birth which left an area of focal atrophy with narrow gyri in that part of the left hemisphere indicated by the broken line in Fig. 11. Ordinary responses could be elicited by stimulation of the pre- and postcentral gyri as indicated by the numbers and letters in the figure.

The surprising finding in this case was that with the same stimulation strength (thyratron 16) the arm sensory aura could be obtained well anterior to the Rolandic convolutions. Both W and 17 (see Fig. 11) gave arm movement. But anterior to this at N, B, L and even P arm sensation was produced without movement. All of these points except P were verifiable by restimulation after an interval of time.

In the area of focal atrophy about point 24 the arm aura could be produced at a number of points but not more strongly than when the anterior points upon the frontal lobe were stimulated. Because the rest of the brain appeared normal and because of its location the atrophic area was considered to be the epileptogenic focus and was radically removed. This has resulted in freedom from attacks during the months that have followed operation.

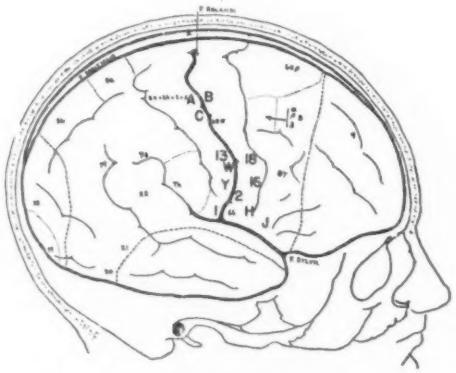


FIG. 13.—Case 7. Same as Fig. 12. A—Sensation in third and fourth fingers left side. B—Closure of hand, no sensation. C—Patient said "third and fourth fingers jumped." 13—"Numbness" in left face. W—Same. Y—Sensation in tongue. 1—Sensation in tongue and left cheek. 2—Facial grimace and grunt. H—Numbness in second and third fingers left side. Repeated once—same result. Repeated again with no warning—same result. After a lapse of time repeated again with no warning—verified again. J—Sensation of "tightening in stomach." Repeated after an interval—no response. Repeated after a further interval and patient exclaimed "Something funny in my stomach." 16—"Peculiar feeling in pit of stomach." Repeated twice—same result and quivering seen in abdominal wall. Patient said it was not like an attack. 18—Abdominal contraction more marked on left.

This is an example of aura response from a distance in the presence of a normally responsive sensorimotor cortex.

CASE 7.—*Distant Response of Sensory Aura.*—E. C. A woman of 43 complained of convulsive seizures thought to be due to a severe head injury at 7 years. The patient suffered from focal seizures characterized by a sensory aura in the epigastrium which might spread to tingling in left hand. Occasionally a generalized seizure resulted. In the operative exploration no lesion was found.

Many detailed responses were recorded as indicated by the tickets in Fig. 12 but the majority are omitted in Fig. 13 to avoid confusion. The unusual feature of this record was that stimulation at H (lower frontal lobe, Figs. 12 and 13) produced numbness in the fingers of the opposite hand just as stimu-

lation at A had done. A probably represented the cortical sensory hand area inasmuch as B gave rise to hand movement. J gave an unexpected response of epigastric sensation. We have since learned that representation of this sensory aura in some cases at any rate lies at the bottom of the central fissure. It may be possible that stimulation at J produced a response of the epigastric sensory point at the bottom of the central fissure near A.

Stimulation at H was repeated three times with the same result. These repetitions were after lapses of time during which many different procedures and stimulations were carried out and no warning was given the patient. J was also verified by repetition.

This sensory response from a distance can hardly be due to escape of current across the surface of the cortex inasmuch as many positive stimulation points lie between. There hardly could be escape of current to the internal capsule beneath because of the distance, the weakness of the current and the fact that none of the other cortical stimuli did so.

One must conclude then that at these points connecting neurones were stimulated which activated the cortical sensory areas for hand and for epigastrium. It is significant that these sensory phenomena made up her habitual epileptic aura. This patient had informed us that for the past 8 or 9 years any sudden sound was apt to give her a shaking sensation in the left side of the abdomen. Also she had likened her abdominal aura to a "sudden crash on the piano." That H and J represent points on a distant association line between auditory and sensory regions might be suggested quite tentatively.

CASE 8. *Incomplete Sensorimotor Refractory State, Distant Response.*—M. S. A girl of 20 years, had received a head injury at 4 years of age. Epileptiform seizures appeared at 10 and were ushered in by a sensation of tingling in the left arm, body and leg. Occasionally there was an aura of light before the eyes. Operation showed a triangular area of yellowish excavated brain (within broken line in Fig. 14) firmly adherent to the dura at one point. This was evidently the end-result of a contusion and subdural hematoma.

The small circles in Fig. 14 indicates areas stimulated without response. Stimulation of 4, 5 and 8 gave sensation in the left hand while 2, 3 and 6 gave rise to the same sensation but was preceded by a feeling that she might be going to have an attack. Obviously the threshold of stimulation of the postcentral hand area was lower than that of any other pre- or postcentral area, for the circles show where these gyri were stimulated without result. Furthermore, the zone of brain about the lesion enclosed in the broken line contained several points which were so connected with this postcentral hand area as to be able to activate the same area by conduction from a distance.

This may be called sensory hand response from a distance but there was an important difference in the result of stimulating 2, 3 and 6. At each of these stimulations she thought she was going to have an attack and added that there was a sensation in the hand. On stimulation of 4, 5 and 8, however, she referred only to the hand sensation. Therefore, stimulation at a distance must have produced primary sensation which was not put into words by her but which signified a true attack. One may surmise that during her habitual

spontaneous seizures a more distant discharge of nervous tissue occurred which was followed by, or associated with, neuronal conduction to the postcentral hand area.

CASE 9. *Incomplete Sensorimotor Refractory State, Distant Response, Epileptic Spread.*—N. B. A boy of 18 suffered from convulsive attacks ushered in by an epigastric aura. What he described as a feeling of disgust occurred beneath the xiphoid and rose slowly to the midsternum after which clonic generalized convulsion might occur beginning with movements in the right hand.

The dotted line in Fig. 15 indicates an area of atrophied convolutions from which both aura and convulsive seizure could be produced (see legend of Fig. 15). This area lay posterior and adjacent to the postcentral convolution

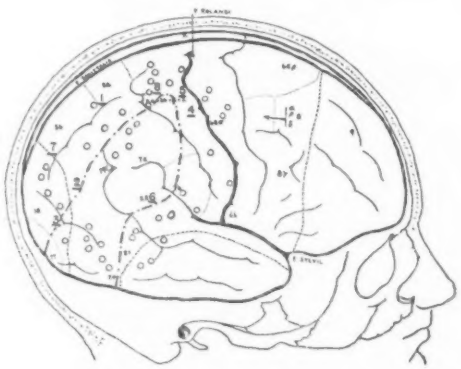


FIG. 14.—Case 8. Dotted line surrounds area of abnormal brain.

Galvanic stimulation: 1—Red lights before both eyes 6 m.a. 2—Sensation in left hand "like when I have an attack" 5.5 m.a. 3—"Felt as though I was taking a turn" 6 m.a. This was followed by recurring sensations in her hand like an attack. 4—Left hand went to sleep 6.5 m.a. 5—"Shaky feeling in left hand and arm" 7.5 m.a.
Faradic stimulation: 6—Felt as though she were going to have an attack—sensation in left hand. 7—"A whole lot of colors"—said to be seen in left eye. 8—Shook left hand voluntarily and said hand and arm had gone to sleep.

and yet when it was stimulated it communicated exclusively with one very small sensory area—that for the right side of the chest. This sensory area lay in the postcentral gyrus probably deep in the fissure between the large area for sensation of the upper extremity below and that for the lower extremity above (see trunk in the sensory sequence in Fig. 1). The direction of discharge would seem to be that indicated by the line of arrows in Fig. 15.

Galvanic stimulation was used pre- and postcentrally but no response at all occurred at the lower end of the Rolandic region. Jaw and mouth movements were produced unexpectedly high and hand movements as well. It is obvious that the postcentral gyrus on the morning of operation had a higher threshold for direct stimulation than the precentral gyrus except for the sensory chest area. This was in contrast to the findings in case 3 where the precentral gyrus seemed depressed and the postcentral more sensitive, the lesion being frontal.

In this boy's spontaneous seizures the aura of epigastric sensation was apt to last for from fifteen seconds to a minute and then either to stop altogether or to advance into motor phenomena beginning with hand movement. The direction of conduction in this case from the abnormal area was obviously the direction of conduction during each of his habitual seizures. Because of the selective character of the transmission it was presumably over a neurone pathway. It seems likely that repeated discharge from this focus had conditioned that particular neurone pathway so that it alone carried outgoing impulses instead of conduction occurring along the many other possible pathways.

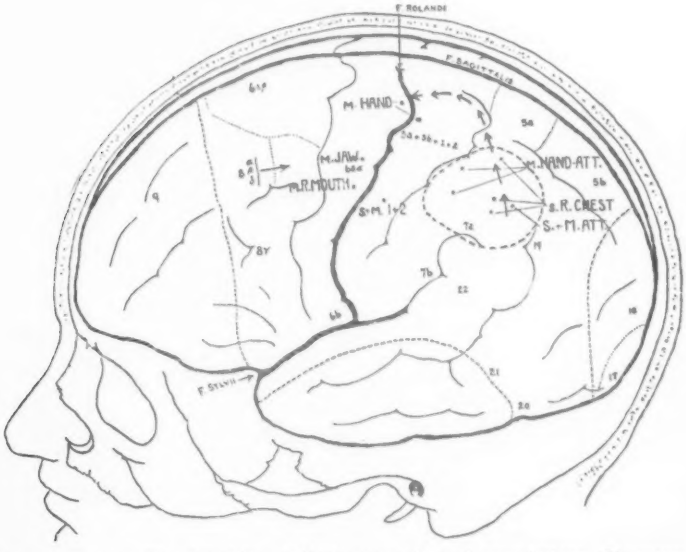


FIG. 15.—Case 9. The dotted line indicates area of small convolutions. M=movement. S=sensation. 1=thumb. 2=index, etc. SR=sensation in right side of chest said to resemble the habitual aura, one of the responses seemed to give sensation through the whole body but resembled aura. M Hand ATT=convulsive seizure involving right hand. S and M ATT = attack ushered in by typical chest aura and followed by convulsive movements of right hand.

To summarize in this section the observations on atypical responses to stimulation of the epileptic's cerebral cortex: There is recorded *distant response, refractory state, activation* of otherwise *silent areas* and marked *fluctuation of stimulability*. These phenomena will be treated under the general discussion in section D. But before doing this the cortical localization of initial epileptic manifestations must be taken up.

C. Localization of Initial Phenomenon in Epileptic Discharge.

Does the existence of distant response in the epileptic cortex mean that seizures involving one area may be produced by distant electrical stimulation? To answer this question we have placed together those seizures which began with movement of an upper extremity in one group, movement of the face in another, and so on. We have then selected certain of the groups containing the largest number of examples for study. For each group the stimulation points were placed upon one brain chart.

The analysis of each attack pattern is not always easy. For example, hand movement may be the first objective evidence of an attack. But if the movement was preceded by evidence of a dream or of loss of consciousness then this attack does not belong with the group in which hand movement was the first phenomenon. To illustrate this a few cases are described below in which the nature of initial manifestation was open to discussion.

These induced seizures are not all identical with the habitual attack from which the individual suffered. Some are what one might call physiological fits in which case they most often occur from stimulation of the Rolandic convolutions.

1. *Beginning with Upper Extremity Movement.*—In Fig. 16 the induced attacks which began with movement of one upper extremity are grouped together, the site of stimulation in each case being recorded.

Point 117 (Fig. 16) should not be included here as loss of consciousness seems to have preceded the movement. But it may be examined as an example of the analysis required to establish each point. The first objective evidence of seizure was inward rotation of contralateral hand followed or associated with turning of head to the ipsilateral side and a bewildered expression of the face. This was followed by convulsive movement of the contralateral arm, of the eyelids and by puckering of the mouth. The patient responded to questions only 15 seconds after cessation of movements and then he knew nothing of the attack. Because of his not remembering any movement it seems possible that the initial response was unconsciousness and not hand movement and the seizure is therefore eliminated.

This supposition is further borne out by the fact that previous stimulation at the same point, with a little less intensity of thyratron current, caused the patient to say, "I have some kind of feeling in my head." He then did not respond but he moved his head. After being spoken to twice he said "I had a sort of dream feeling." This point of stimulation was adjacent to an area of focal atrophy considered to be epileptogenic.

If point 117 be eliminated then all of the points in this chart (Fig. 16) are either located upon the precentral gyrus or within 2 cm. of it. Therefore the localization of the stimulation points which produced initial convulsive movement in the upper ex-

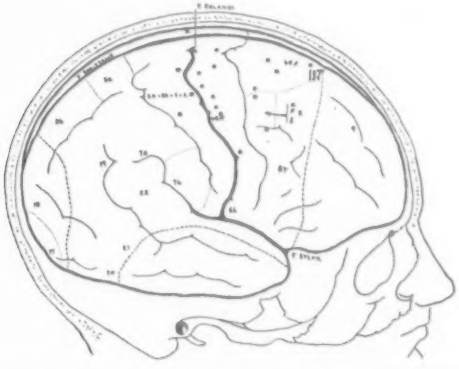


FIG. 16.—Stimulation points (dots) which produced seizures beginning with movement in one or other upper extremity. The number in this figure and the numbers in the subsequent figures refer to the case in question. Electric current usually from thyratron; less often faradic coil. For explanation of elimination of point 117 see text.

tremity corresponds fairly closely with the localization of simple motor movements in upper extremity as shown in Fig. 2. The convulsive response points do extend anterior to the motor gyrus which the simple responses seldom did. But the important observation to be made is that there is no convulsive response produced from a great distance. The distant responses cited in the previous section were simple responses not convulsive responses.

2. *Beginning with Upper Extremity Sensation.*—In Fig. 17 it is seen that the induced attacks beginning with sensation of an upper extremity are relatively few but all the points are in or near the expected area in the postcentral gyrus.

3. *Beginning with Mouth and Throat Movement.*—The induced

attacks beginning with mouth and throat movement are more frequent (Fig. 18). They fall, for the most part, in the expected region as may be seen when Fig. 18 is compared with Fig. 3 which lists the non-epileptic motor and sensory responses for the mouth.

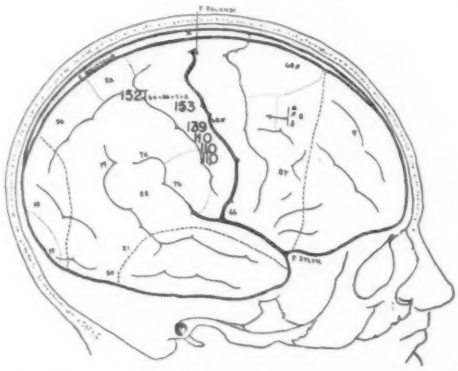


FIG. 17.—Stimulation points for seizures beginning with sensation in one upper extremity.

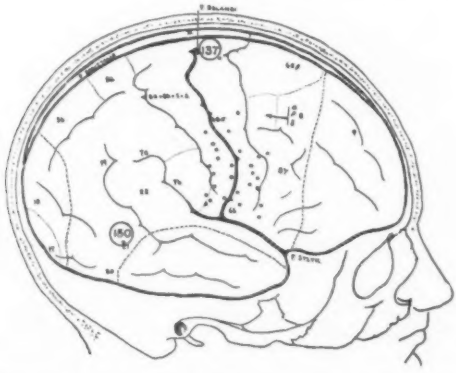


FIG. 18.—Stimulation points producing seizures which began with movements of mouth and throat. For explanation of elimination of points 150 and 137 see text.

There were only two examples of induced attacks beginning with face sensation alone and consequently no chart is made.

Point 150 from Fig. 18 must be excluded when the case is examined closely. It is from a case in which an habitual attack was started at this point next to an epileptogenic focus. The first movement was swallowing; the patient then said he felt nauseated; his pupils dilated, then contracted and his eyes turned in tonic manner

to the opposite side and he became unconscious. Obviously this swallowing was an expression of his feeling of nausea and not a true motor movement.

Point 137 likewise should be eliminated. When this point was stimulated the patient opened his mouth as though startled but there were no clonic movements. The face movement seemed more an evidence of mental change than a convulsive movement. The cerebral pulsation was changed in a manner which showed that a true seizure had occurred.

4. *Beginning with Vocalization.*—Fig. 19 indicates the induced attacks in which vocalization was the initial phenomenon. In case

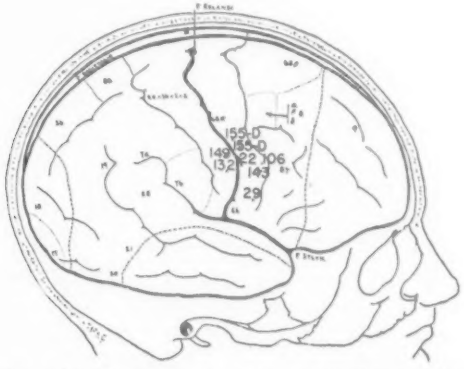


FIG. 19.—Stimulation points for seizures in which vocalization was the first phenomenon except 155-D in which difficulty of speech was the first manifestation of an attack.

22 (point 22 in Fig. 19) it was not quite clear whether convulsive face movement preceded or followed vocalization.

The points 155-D in Fig. 19 represent difficulty of speech. These two attacks began in this way without any forced vocalization. Inability to speak has often been a non-convulsive response to stimulation occurring in widely separated parts of the cerebral cortex in which cases it was not followed by seizure.

D. General Discussion

Much of the explanation which follows must be considered hypothetical but the material presented has been factual and he who reads may also reason.

1. *Point of Origin of Induced Seizures.*—When all examples are carefully analysed as in section C above it is seen that electrically

produced fits which begin with motor or sensory manifestation have their stimulation points in or near the corresponding motor or sensory zones of the cortex.

The distant-motor or distant-sensory responses which have been described above do not usher in epileptic seizures. These distant-responses are evidently produced by marked lowering of the stimulation threshold for certain elements in the Rolandic cortex associated with connecting neurone links ready to conduct toward that area.

2. *Activation.** In the cortex of the epileptic it is usually found that great detail may be elicited by stimulation of pre- and post-central gyrus particularly if it has been habitually involved in epileptic discharge. Furthermore areas of brain, such as the temporal lobe, ordinarily silent on stimulation may give responses if these areas have been the seat of an habitual epileptic discharge.

On the other hand the response of the epileptic cortex may be more complicated and may reveal elements of stored experience, as for example the little girl (Penfield 1938) in whom a harrowing memory of a man who frightened her could be produced at will by stimulation in the supramarginal region. That part of her cortex had been conditioned by the original experience associated with fright. That is to say the experience was recorded in a pattern of neuronal connections. The conditioning process was then reinforced by subsequent nightmares in which the same man appeared to frighten her. Later on, epileptiform seizures made their appearance because of an early subdural hematoma in the region where the above neurone pattern had been established. The epileptic discharge followed that pre-established pattern of neurone connections. The result was that in her aura the man appeared again behind her and she was terrified. If the seizure went further one-sided convulsive movements appeared and spread to both sides, followed at times by a transient hemiplegia.

Activation and conditioning of the cortex in one sense would seem to be identical processes. Thus activation may render accessible to the electrode the evidence of innate function present in all normal brains or it may reveal the acquired experience peculiar to one individual.

* Much of the evidence on activation may be found in Penfield (1938) and Penfield and Boldrey (1937).

3. *Refractory State.*—Even the most easily elicitable responses fail in the epileptic cortex at times. This may be the immediate effect of a convulsion without paralysis. It may be associated with post-convulsive paralysis or it may be quite independent of obvious previous seizure.

The case of a boy may be cited who while he was being operated upon had a spontaneous Jacksonian seizure in an early stage of the operation (reported in detail by Penfield, 1937). When the dura was opened he was conscious but drowsy. He could move all his extremities but the cortex gave no response whatever to stimulation even though a comparatively strong current was used. He was allowed to sleep on the operating table for 40 minutes after which time he was alert and then the Rolandic gyri gave good response to stimulation. This is an example of post-convulsive refractory state without clinical evidence of paralysis. Case 2 described above in section B was an example of refractory state associated with post-convulsive hemiplegia.

But the Rolandic gyri may be in a refractory state when there has been no evidence of recent seizure and no weakness of voluntary movement, as in case 4 where it (the refractory state) was associated with astonishing displacement of one responsive element far across the cortex. In cases 3, 5 and 8 there was an incomplete refractory state, also without evidence of recent seizure.

4. *Distant Response, Simple Somatic.*—Distant response may apply to one or more elements in the sensory or motor somatic sequence which do not figure in that patient's epileptic march so far as can be determined. Or it may apply exclusively to the initial phenomenon in his epileptic seizure whether it be motor or sensory. In either case distant response much more often involves a sensory than it does a motor element, and if motor the face is involved and not an extremity.

In case 5, in the presence of a frontal pole lesion, the postcentral gyrus was refractory and there was a slight tendency to distant response of a motor element. In case 4 in the presence of a profound refractory state of motor and sensory elements movements of eyelids occurred in two areas, one at least of which was far from the expected area for such movements.

Distant response is often associated with a refractory state of other elements. For example in case 3 hand movement which came

early in his habitual seizures could not be elicited, but hand sensation was hyperstimulable and it gave distant response far over the frontal lobe. Associated with this there was a marked refractory state of all motor elements. Even in the postcentral gyrus hand sensation responses spread over a wide extent of the gyrus where other sensory responses would normally be expected.

Such distant response might in some cases be considered as a result of the refractory state of other elements inasmuch as a stronger stimulating current might be used. This was true in case 2 where motor hemiplegia existed. It was also true in case 4, but usually the distant responses were elicited with weak stimuli.

5. *Distant Response, Aura.*—This may also be associated with a refractory state of other elements as in case 8, or it may exist in the presence of other well activated responses as in cases 6 and 7. Distant response of an epileptic aura may occur widely with little reference to the epileptogenic focus as in case 6, but it is found more frequently in the neighborhood of the lesion, and between it and the proper representation in the Rolandic cortex, as in cases 8 and 9. Occasionally distant displacement of an aura occurs with no intervening responses and no obvious explanation, as in case 7.

A sensory aura may give responses much more distantly than a motor aura (if an initial motor response may be called aura). But there are some few examples of the latter at moderate distances, for example the eyelid movements produced at a distance at points L, D and M in case 1 (Fig. 5).

6. *Fluctuation of Stimulability.*—We are dealing in this paper with the exceptional cases. As a rule the epileptic cortex is easily stimulable and the responses occur where expected. We have assumed that the irregularities of responsiveness, the refractory state, the activation, the hyperstimulability and the reciprocal arrangements of these phases are peculiar to the epileptic's cerebral cortex and are a result, or a part, of the epileptic process.

We make this assumption not on the basis of an equal number of explorations of normal individuals, (such a thing is obviously impossible) but on the basis of the regularity of response to stimulation of the subhuman mammalian cortex. Furthermore we have occasionally stimulated a non-epileptic human cortex to determine how wide neoplastic removal may be made without damaging the Rolandic convolutions, and these have shown none of the irregular

phases. But it must be admitted that the number of such non-epileptic cases is too small to be of any statistical importance.

Grünbaum and Sherrington (1901 and 1903) pointed out the "instability" of a motor point upon the cortex and the fact that it is influenced by previous neighborhood stimulations but nothing similar to these examples of spread are to be found in their experimental reports. Of course experimental work applies only to motor manifestations as no evidence of cortical sensation is to be obtained from animals. We have just pointed out above that distant response is usually sensory or if motor then it is facial.

Graham Brown and Sherrington 1912 showed that the response of one point might be qualitatively changed from flexion to extension, a phenomenon they called reversal. With Sherrington (and also alone) Graham Brown (1915a; 1915b) showed that by repeated subliminal stimulations a spot could be sensitized, and furthermore that a motor response could be displaced by repeated advancing stimulation, and this was called facilitation.

We have pointed out in man (Penfield and Boldrey 1937) that a motor or a sensory response obtained at one point can, by regularly repeated stimulations, be moved across a convolution into a position which formerly was not stimulable. Furthermore in case 1 above we have pointed out that a sensory aura can be similarly displaced as far as 4 cm. into previously unresponsive territory if a fissure is not crossed.

It seems likely that in a few epileptics, or perphaps on rare occasions in all epileptics, spread inhibition and unexpected activation would be present if the cortex could be examined at the appropriate time. It seems to us probable, except for certain transient periods when inhibition affects them, that those areas of cortex which are habitually subjected to epileptic discharge have a lowered threshold to electrical stimulation and no doubt a lowered threshold to epileptic stimulation.

It seems probable that ordinarily any phenomenon that initiates an epileptic seizure has some tendency to spread. That is, the phenomenon can be produced at one point which may be called primary and at a little distance from that point as well. But true distant response of an isolated epileptic phenomenon is largely that of a sensory aura.

7. *Electrical Stimulation and Epileptic Seizures.*—If one stimulates briefly an area of precentral convolution a simple motor response may occur. If one stimulates the same spot with the same current for a longer period there may result a seizure which is to say that the convulsive movement continues and may spread after the withdrawal of the electrode. Therefore, whether or not a continuing local discharge occurs depends upon the strength of stimulation of that local collection of ganglion cells and upon the duration of stimulation.

There is in the brain of many, perhaps all, epileptics a focus where proper electrical stimulation would reproduce a seizure which is identical with the seizures from which that unfortunate individual suffers. The spontaneous and the induced seizures are identical objectively and, what is more significant, identical in the patient's own experience, provided of course that there is no initial loss of consciousness. The point for such stimulation cannot always be found for it may lie beyond the reach of the exploring surgeon or the point in question may be temporarily refractory. Furthermore the proper type of stimulating current has not yet been devised for certain areas such as the frontal pole.

Electrical stimulation of the Rolandic gyri, short of producing a seizure, reproduces a movement or sensation which is identical with the convulsive movement or sensation of that part. That is, such movements are never adroit or purposeful and such sensations are never similar in quality to ordinary sensations.* Electrical stimulation, however strong, either produces no obvious effect at all or it seems to result in the undiscriminating discharge of the ganglion cells stimulated. That discharge is doubtless for the moment complete.

It is fair to reason therefore that in an epileptic seizure, as Jackson surmised long ago, there is violent discharge of nerve cells and that they are the same nerve cells discharging in the same way as though the cause of discharge were an electrode instead of that enigmatic stimulating influence that exists in the vicinity of an epileptogenic focus.

* An exception must be made here. When cortical stimulation produces a sensation of movement in a part, the sensation does seem to be a normal experience.

So much for the initial epileptogenic discharge. The question now arises as to whether the initiating stimulus can operate from a distance as in the simple motor or sensory distant-responses.

It was with the intention of answering this question that Figs. 16, 17, 18 and 19 were prepared.

Comparison of Fig. 16 with Fig. 2 shows that the stimulation points of fits beginning with arm movement correspond with the location of motor points for arm movement except that the former extend about 2 cm. farther away anteriorly. The same is true in regard to mouth movement as shown by comparing Figs. 18 and 3. The points which produced fits beginning with mouth movements are only a little more widely spread. In the preparation of motor and sensory representation charts (2 and 3) the examples of distant response were excluded. In the preparation of the seizure charts (16, 17, 18 and 19) all of our relevant cases were included, no matter where the site of stimulation might be. There was a careful analysis to include only those seizures in which the motor or sensory phenomenon in question was actually the primary incident but no other selection was made.

Therefore it seems safe to conclude that electrical stimulation does not primarily initiate epileptic discharge at a distance from the electrode and it seems likely also that spontaneous fits are not produced by focal lesions which lie at a distance. Instead of that, the epileptic discharge begins in each case at a point in the brain adjacent to the electrode and, by analogy, probably adjacent to an epileptogenic lesion.

8. *Epileptic Spread.*—If, as suggested above, an epileptogenic discharge begins in a circumscribed area of grey matter near the originating stimulus how does this discharging state spread to other parts of the brain?

Penfield, Sántha and Cipriani (1939) have shown that in electrically produced seizures in animals a remarkable increase in blood flow is shown by the Gibbs thermocouple but the increase occurs only in the motor cortex and in the basal ganglia not elsewhere in the cerebral cortex no matter how severe the fit. It does cross to the opposite motor cortex when the convulsion becomes bilateral. In man they showed also that blood flow increase occurred at the site of discharge but that large areas of brain showed no obvious change and it was concluded that the epileptic discharging state does

not become generalized in the brain even when the body is completely convulsed.

It seems to be a general principle that the discharging state migrates from any particular point upon the cortex toward the Rolandic gyri. It seems in general to be "downhill" toward this area of the cortex and hence seizures of diverse character, if they progress at all, end with the same gross convulsive movements of body and extremities. Thus an attack which begins at a focus in the parietal lobe does not spread backward into the occipital lobe but forward toward the central fissure.

Granting therefore that the disturbance is most apt to move toward the central fissure it seems hardly possible that the disturbance moves across the brain as an all-discharging wave including all centers on the way because of the complicated and somewhat discriminating pattern of some seizures. Certain examples may be introduced into the argument at this point.

In case 8 there was marked activation of the postcentral representation of hand sensation and the rest of the Rolandic convolution that could be reached for stimulation, was refractory. Hand sensation was produced by stimulation at points 4, 5 and 8 on the postcentral gyrus (Fig. 14) and at a distance it was produced by stimulating in the vicinity of her lesion at 2, 3 and 6. But when 2, 3 and 6 were stimulated there was an additional phenomenon. She said she thought she was going to have an attack and then added that there was sensation in her hand. These latter stimulations produced distant response of hand sensation but evidently there was also a local primary process set up which produced the aura of her habitual seizure.

Therefore it seems likely that her spontaneous seizures began by a discharge in the vicinity of the lesion. The spread of this discharge then occurred along a neuronal pathway to the postcentral hand area. If this bombardment of hand area was sufficiently intense she had a spreading Jacksonian seizure. If it was less intense she had only an aura without sequel.

If such selective conduction occurs in the epileptic state the line of conduction must be established by a conditioning process which may be reinforced by each succeeding epileptic discharge. In complicated seizures the attack often evolves slowly and deliberately. There are not infrequently familiar scenes or people in

such seizures, as there are so often in dreams. One example of this was the case of the little girl already mentioned whose remembered experience had become a seizure pattern, a pattern that could be activated by stimulation.

In such cases it may well be that there is a complicated system of nerve cells and nerve fibers welded together by repeated conduction and the impulse which sets off this system originates in an epileptogenic focus. This neurone system, which must be similar to the neuronal basis of memory, comes to be isolated from other pathways by multiple synaptic inhibition which may constitute the essential factor in the establishment of such a fixed pattern.

E. General Conclusions.

The cerebral cortex of the epileptic passes through varying phases of stimulability. Simple response both motor and sensory may occur as the result of stimulation at a distance. Such distant response is due to focal lowering of threshold and to selective neuronal conduction. In general, cortical stimulation produces movements and sensations which are not like normal movements and sensations but qualitatively identical with those that may occur in epileptic seizures. The cortex of an epileptic may be found to be refractory to electrical stimulation and at such times may still be functionally available to the patient for volitional purposes.

An habitual seizure, by virtue of its frequent repetition, may eventually establish a complicated neurone pattern, so functionally organized that it may be stimulated electrically although no such complicated response could be obtained from stimulation of the normal brain. Induced seizures do not arise from distant stimulation. They begin by discharge in the neighborhood of the stimulating electrode. This is followed by spread, which is not diffuse, but which is along a definite neurone system which may be preformed or acquired. By "preformed" is meant a system of neuronal connections ordinarily recognized as physiologically functional in all normal brains. By "acquired" is meant a pattern of neuronal connections established by the conditioning influence of previous individual experience. In this sense habitual epileptic seizures should be considered true conditioned reflexes in the cortex of any patient.

In complicated seizures the advance of discharge along this neurone pattern may be so slow and episodic as to suggest that isolated

ganglionic collections are fired in an advancing series, each collection being set off only after neuronal bombardment of sufficient intensity and duration from other cell collections already discharging.

BIBLIOGRAPHY.

Brown, T. Graham: 1915A. Studies in the physiology of the nervous system. *22*: on the phenomenon of facilitation. 1: Its occurrence in reactions induced by stimulation of the "motor" cortex of the cerebrum in monkeys. Quart. Jour. Exp. Physiol., 9: 81.

Brown, T. Graham: 1915B. Studies in the physiology of the nervous system. *23*: on the phenomenon of facilitation. *2*: Its occurrence in response to subliminal cortical stimuli in monkeys. Quart. Jour. Exp. Physiol., 9: 101.

Brown, T. Graham, and Sherrington, C. S.: 1912. On the instability of a cortical point. Proc. Roy. Soc., 85B: 250.

Grünbaum, A. S. F., and Sherrington, C. S.: 1901. Observations on physiology of the cerebral cortex of some of the higher apes. Proc. Roy. Soc. of London. 69: 206.

Grünbaum, A. S. F., and Sherrington, C. S.: 1903. Observations on physiology of the cerebral cortex of anthropoid apes. Proc. Roy. Soc. of London, *72*: 152.

Penfield, W.: 1936. Epilepsy and surgical therapy. Arch. Neurol. and Psychiat., 36: 449.

Penfield, W.: 1937. Circulation of the epileptic brain. Assn. Res. Nerv. and Ment. Dis., Proc., Vol. 18.

Penfield, W., and Boldrey, E.: 1937. Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. Brain, 60: 389.

Penfield, W., Sántha, K. von, and Cipriani, A.: 1939. Cerebral blood flow during induced epileptiform seizures in animals and man. J. Neurophysiology. II, 257-267.

Penfield, W.: 1938. The cerebral cortex in man: 1. The cerebral cortex and consciousness. Arch. Neurol. and Psychiat., 40: 417.

A STUDY OF ACCIDENTS IN A MENTAL HOSPITAL.*

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Considerable progress has been made during the past few years in overcoming popular prejudice against mental hospitals. The publicity which follows any serious accidental injury to a patient must necessarily retard the development of a better understanding on the part of the public of the problems which the institutions are facing every day; and therefore a high accident rate may be largely responsible for the feeling of distrust which still prevails among the relatives of patients. Also some accidents, in addition to damaging the reputation of a hospital, involve considerable expenditure of time and money.

The need for a detailed study of this problem may be illustrated by reviewing briefly two accidents which caused our hospital unusual concern:

The first of these mishaps befell a woman diagnosed as a case of involutional melancholia, a patient of five months' residence and in a convalescent ward at the time of the accident. She fell on a polished floor and received a fracture of the left hip. Following this injury, her mental condition became much worse. Inasmuch as it was held to be the hospital's responsibility in this case, private quarters and special nursing care were provided for the patient during the ensuing year. There were also, at the expense of the hospital, medical and surgical consultations, one operation and numerous X-ray examinations. While convalescing from the fracture she developed malignant hypertension and had several slight cerebral assaults with transient paralyses. She was taken home by her relatives for the Christmas holidays, and died there a week later of hypertension and left ventricular heart failure. Despite the evidence to the contrary, her relatives held the fracture responsible for her death.

* A preliminary study covering a nine-month period was presented at the ninety-second annual meeting of The American Psychiatric Association, St. Louis, Mo., May 4-8, 1936.

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The second of the two accidents concerned a woman of 75 years, a case of psychosis with cerebral arteriosclerosis. She was brought to the hospital by her relatives as a last resort, when they found it impossible to care for her at home. A month after her admission she was pushed by another elderly patient, fell to the floor and fractured her left hip. She died a week later. This accident did not involve serious financial cost, but it was embarrassing to the medical and nursing staffs. The patient's relatives suspected negligence in supervision on the part of the employees.

Inasmuch as accidents are of every day occurrence in the community, it may be expected that hospitals will have their share. It is only the occasional serious mishap that commands such attention as to cause a general inquiry into all accidents. This study was undertaken to ascertain the common elements in the majority of casualties, with a view to recommending preventive measures. So little has been written on this subject that it seemed worth while to offer a new approach to the problem. Accordingly we have made a careful analysis of each accident, however trivial, for the two-year period from June, 1935, to June, 1937.

Method of Study.

For each accident which occurs in any state hospital of Massachusetts, as in many other states, a printed form is filled out including the report of the attendant on the ward and the results of all subsequent investigations. (Following the investigation, an explanatory letter is sent to the patient's relatives.) These individual reports served as the source of information for this study. Of course it is recognized that there are unavoidable errors when so many persons contribute to the data. It is quite likely also that some accidents which resulted in little or no visible injury were not reported, especially those not witnessed by an employee. The best we can do is to accept the reports as they stand.

Certain items found in these reports have been treated statistically, especially the following: age, the time and place of occurrence, causative factors, nature of injury, treatment required, and personal responsibility for the accident. More detailed analyses have been made of fractures, suicidal attempts, lacerations requiring sutures, and a few other serious accidents.

Results.

The total number of accidents reported during the two-year period was 4755, of which all except 413 were trivial in effects. The resident population of Danvers State Hospital is usually about 2350 patients, with approximately 100 more women than men. The monthly average of accidents was 76 for the male and 121 for the female patients. The number of accidents per patient per year was 1.08.

Ages of Patients Suffering Mishaps.—In order to ascertain the comparative frequency of accidents at different periods of life, the patients were divided arbitrarily into age groups by decades. (See Fig. 1.) The graph shows for each age-group the number of

TABLE I.

Average Number of Accidents per Patient in Each of Four Groups.

Type of patient.	No. of accidents.	Number of patients.	Average accident per patient.
Workers	406	104	3.90
Excited or deteriorated	2670	708	3.77
Newly admitted	931	598	1.55
Infirmary	748	779	0.96

patients suffering accidents, in proportion to the usual number of corresponding age in the resident population. (On the assumption that the age distribution of the patient is reasonably constant from month to month and from year to year, the number of hospital patients in each decade was estimated at the time when the tabulations were made.)

It will be noted that the accident rate was highest among the youngest and the oldest patients—6.9 accidents per patient under 20 years of age, and 3.8 accidents per patient among those upwards of 80 years. These findings at the extremes of age might reasonably be expected, because the psychosis in these groups is usually on an organic rather than a functional basis. Also, the younger patients are perhaps more liable to accident on account of over-activity; and the aged ones because of inability to take care of themselves.

Type of Patient.—(Table 1.) To determine the comparative

number of accidents among patients of different types, the resident population was divided roughly into four classes, this grouping being based largely upon the wards in which the patients lived: (1) workers; (2) excited or deteriorated patients; (3) newly admitted patients; (4) infirmary patients. The number of accidents among patients of a given group was divided by the number of resident patients belonging to that group, to obtain the average number of accidents per patient in that class. The highest accident

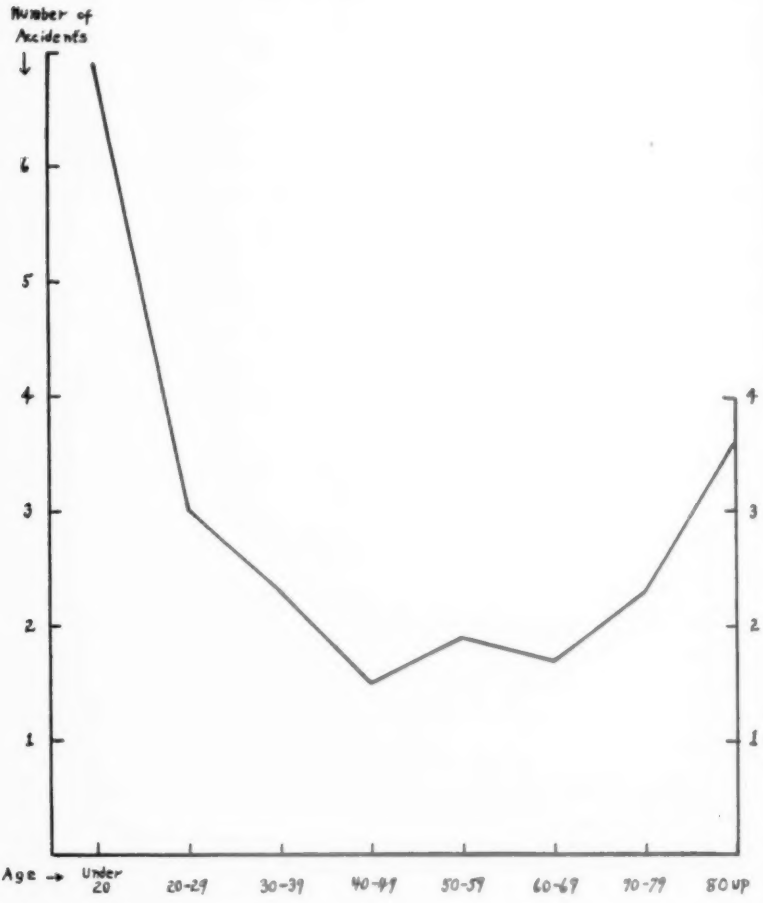


FIG. 1.—Average accident per patient in each age group during two-year period.

rate was among the workers, 3.9 per patient. The group of excited and deteriorated patients followed closely, with a rate of 3.77 per patient; this group was large in population and accounted for more than half the total number of accidents. The accident rate for the newly admitted patients was 1.55 per patient; and for the infirmary patients only 0.96 per patient.

Hour of Occurrence of Accidents.—(Fig. 2.) The distribution of accidents throughout the day was ascertained by dividing the day into intervals of varying length according to the patients'

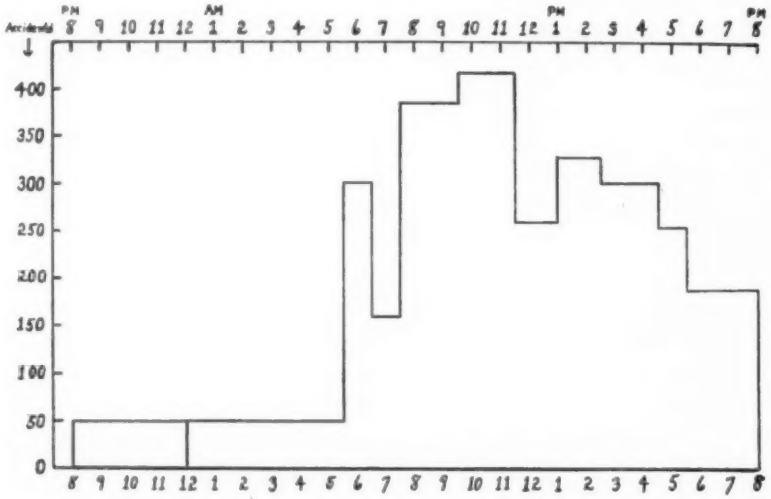


FIG. 2.—Average number of accidents per hour at different times of day.

routine of living. The number of accidents occurring during each interval was divided by the number of hours in the interval, to determine the number of accidents per hour for that particular time-period. The graph shows a low and fairly constant average from 8.00 p. m. to 5.30 a. m., during which period the majority of the patients were asleep. The rise in the curve from 5.30 to 11.30 is broken only by the drop during the breakfast hour. There is another drop at the dinner hour, followed by another rise which does not approach the high morning average. The highest point in the afternoon is from 1.00 to 2.30, following which there is a gradual decrease until bedtime. At each of the meal periods there is a notable decrease in accidents. This drop during the break-

fast hour is especially significant in that it represents the only break in a steady rise during a six-hour interval. More than half the patients are fed in a cafeteria which is at some distance from many of the wards. Going to and from meals they must necessarily travel up and down stairs, also through corridors and tunnels where they are crowded and where supervision is difficult. In view of these conditions, it is rather surprising that so few accidents have been observed at these times.

Place of Occurrence.—Eighty per cent of the accidents occurred in the hospital wards. Four per cent occurred in each of the following places: cafeteria, passageways, grounds. The remaining

TABLE II.

4755 ACCIDENTS CLASSIFIED ACCORDING TO CAUSES IN APPROXIMATE PERCENTAGES.

Cause.	Male.	Female.	Total.
Assault or altercation	37	41	39
Fall	19	21	20
Over-activity	12	9	10
Occupational	12	7	9
Breaking glass	6	5	5
Epileptic seizure	4	6	5
Suicidal attempt	1	1	1
Other causes	5	5	5
Unknown	4	5	5

eight per cent happened in small numbers in various other parts of the institution. There were more outdoor casualties among men than women, presumably because a larger proportion of male patients work on the grounds.

Causative Factors.—(Table II.) Forty per cent of the accidents were attributable to personal violence on the part of other patients—either altercations or unprovoked assaults. Falls accounted for 20 per cent. The other 40 per cent were distributed among the following causes: over-activity, breaking glass, epileptic seizures, suicidal attempts, accidents incidental to occupations and undetermined causes. A small group was made up of unusual or infrequent causes which could not be classified. For examples, a man was struck during the night by a piece of plaster which had fortuitously detached itself from the ceiling; a woman had the misfortune to sit on a tatting shuttle; and a man suffered a severe

nosebleed from being accidentally jostled against a wall which he was in the act of kissing.

For male patients the largest proportion of altercations was among those in their twenties; whereas for the female patients it was in the thirties and forties.

In nearly 300 of 1500 assaults by patients the injured patient was attacked with a weapon of some sort, most commonly a shoe held in the hand. It is noteworthy that the shoe was utilized by female patients in 129 instances, in contrast to only five among the male patients. Various other weapons were used in 114 instances by women and 45 by men. Thus the women held the preponderance not only of simple assaults, but also an overwhelming majority of the assaults with a weapon. Other articles used as weapons by the ladies were brooms, dustpans, aluminum cups, dishes, broken glass, silverware, mop handles, basins, flowerpots, canes, chairs and beds. The gentlemen favored such objects as sticks, stones and shovels. Two of the unusual weapons used by men were a doll and the stem of a pipe.

The majority of patients suffering from falls were over 50 years old—80 per cent for men and 67 per cent for women. Approximately 60 per cent of the total number of falls were the result of slipping on the floor in the wards or lavatories. In about 30 per cent of cases the patients fell out of bed.

There were 56 suicidal attempts, 40 by women and 16 by men. Only three resulted in death. There were six lacerations requiring sutures, and one operation for removal of foreign bodies from the stomach; but the other 46 attempts resulted in no appreciable injury. Ten patients made repeated attempts and were responsible for 27 of the total number. About half the men made their attempts at mealtime when the ward was not so well supervised, whereas the women did not take advantage of this situation. The methods used by women and men show a marked difference. The female patients attempted suicide by tying torn bedding or bandages around their necks or cutting their wrists with scissors or pieces of broken glass. A few succeeded in taking some drug. One woman swallowed 20 pieces of bedspring and innumerable smaller objects such as common pins, safety pins and hairpins. She hoped to die following the operation which would be necessary, but her recovery was uneventful. Most of the men battered their heads

against fixed objects such as walls or doors. One leaped headlong down a flight of stairs, one tied a sheet around his neck, and one tried to drown himself by swallowing water while being bathed. The one death among men resulted from suspension with a belt. Of the two fatalities among female patients, one suspended herself from a window guard with a strip torn from a mattress pad; and the other swallowed about three ounces of lysol while working in an employee's room. This woman, a case of manic-depressive psychosis, had so far improved as to be granted parole.

TABLE III.

4755 ACCIDENTS CLASSIFIED ACCORDING TO NATURE OF INJURY IN APPROXIMATE PERCENTAGES.

Nature of injury.	Male.	Female.	Total.
Contusions	23	32	28
Abrasions	36	24	28
Lacerations	27	22	25
No evidence of injury	9	14	12
Fracture	2	2	2
Other injury	3	6	5

Nature of Injury.—(Table III.) Eighty per cent of the injuries were contusions, abrasions or superficial lacerations. In 12 per cent of the accidents there was no visible injury to the patient. Of this group 395 were reported from the female service and 165 from the male service, a proportion of more than two to one. This difference is believed to indicate that the female employees—including a larger number of nurses and student-nurses—are more careful than the male attendants to report trifling accidents which result in no apparent injury.

Treatment.—In 33 per cent of the accidents no treatment or special attention was necessary. Only those accidents counted as serious—less than 9 per cent—required complicated treatment, such as reduction of fractures, sutures or special medication or attention. In the remaining cases simple treatment was required, such as antiseptics, dressings, compresses or ice.

Personal Responsibility for Accidents.—(Table IV.) Thirty-seven per cent of the accidents were classified as accidental, on the ground that neither the patient nor any definite circumstance

could be held directly responsible. This class includes falls, mishaps incidental to patients' occupation, epileptic seizures and similar unpredictable events. For another 37 per cent other patients were to blame, although of course not legally responsible. In 18 per cent of the cases, the injury was caused by the patient himself. Only 10 accidents (0.21 per cent of the 4755 cases) were known to have been caused by attendants. There were a few other cases, however, in which rough handling by an attendant was suspected although it could not be proven.

TABLE IV.

4755 Accidents Classified According to Personal Responsibility in Approximate Percentages.

Persons responsible.	Male.	Female.	Total.
Fortuitous	39	35	37
Self	15	19	18
Another patient	37	38	37
Unknown	8	7	7

Analysis of Serious Accidents.

The number of accidents considered serious was 413, 8.7 per cent of the total number. In this group there were 177 male cases and 236 female cases.

The number of fractures during the two years was 106, of which number 77 occurred in patients over 50 years of age. The principal causes of fractures were falls (51 cases); and personal violence on the part of other patients (34 cases).

There were 251 lacerations which required sutures or skin clips, and the principal causes of these injuries were as follows: personal violence, 103 cases; falls, 41 cases; epileptic seizures, 29 cases; and breaking glass, 24 cases.

The names of patients who were responsible for more than one serious injury to others have been listed, and it has been found that 13 male patients were responsible for two to six accidents each; and that 18 female patients were responsible for two to four accidents each.

Another list was prepared including all patients who suffered more than one serious injury. There were 13 men who experienced

from two to five different accidents, and 30 women who suffered from two to six accidents each. Fifty per cent of all the serious accidents involved these 43 patients.

Measures for Prevention of Accidents.

The most important environmental elements in the causation of accidental injuries are overcrowding, insufficient personnel, and inadequate facilities for the segregation and treatment of disturbed patients. As these conditions are improved so as to approach the ideal, it may be expected that accidents will be accordingly diminished.

In the meantime there are certain practical and immediately feasible measures which might reduce the number of accidents in mental hospitals. The following suggestions are offered as worthy of careful consideration:

1. There should be a training course for attendants in every mental hospital. In these lectures, stress should be placed upon the common causes of accidental injuries and upon the importance of reporting with utmost care and accuracy every accident observed. (Quite by chance it was discovered that some of our attendants feared they might forfeit their positions if they reported too many accidents.)

2. Constructive outlets for energy among excited and deteriorated patients should result in a decrease of the injuries caused by this group. In many hospitals the initiation of a daily sewing class for some of the most troublesome women has served well to keep these patients pleasantly occupied. Hemming sheets or towels by hand requires little skill, but it gives the patients an opportunity to take pride in the work of their hands. The mental stimulation thus gained is in marked contrast to the dullness of merely sitting or walking about the ward, and it leads the patients to develop a more cheerful attitude of mind. Male patients of this type may be set to work in the fields with pick and shovel, under careful supervision.

3. Inasmuch as noise has a tendency to make patients more excitable and irritable, anything which reduces the ward noises might be expected to lower the accident rate. Vocal noises cannot be entirely eliminated, but they might be appreciably reduced if

all unnecessary noises were brought under control. Agitated and whining involutional cases should be segregated.

4. When new buildings are constructed or when older ones are remodeled, floors of cork composition would be less hazardous than waxed floors of polished wood or slippery linoleum; and they would be a factor also in reducing ward noises.

5. In new buildings there should be a more liberal allowance of floor space, especially for lavatories and day quarters. Stairs should be eliminated as far as possible, preferably by constructing one-story buildings or at least by substituting ramps for stairs.

6. All accessible glass should be safety glass, or else should be protected by wire screening. Radiators and other sources of heat should be covered or concealed. There should be a sufficient number of crib-beds for elderly and epileptic patients.

7. Furnishing the patients with soft shoes having rubber soles and heels would reduce the efficacy of shoes as weapons, would diminish the danger of slipping on polished floors and would tend to reduce the ward noises. Simple rubber matting in the lavatories would be a factor in diminishing falls.

8. Cleaning fluids should be kept carefully locked up when not in actual use; as should also any ward equipment which might make a convenient weapon, such as brooms and mop handles. Frequent searches should be made, preferably at irregular intervals, for any objects which patients may have secreted for use later as instruments for suicide, assault or escape.

9. The exceptionally high accident rate per hour in the latter part of the forenoon indicated the need of closer supervision at this time. Inasmuch as the nurses and attendants are very busy with the routine housecleaning and the morning errands, it might be desirable to have an extra helper assigned to each ward for this limited period.

10. Irritability resulting from fatigue and hunger might possibly be relieved by permitting the patients to sleep an hour later in the morning, thus shortening the interval between breakfast and dinner. Milk or other light nourishment between meals might serve to keep some of the disturbed patients in a more peaceable frame of mind.

11. More careful attention to groups of patients who require

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11. More careful attention to groups of patients who require

special diet or medication might result in a lowered accident rate among patients suffering from epilepsy and other organic disorders.

12. It would be worth while to prepare a list of names, for the use of physicians and nurses, of those patients who are exceptionally aggressive or quarrelsome; and it would be well to have another list of patients who suffer repeated mishaps. These two groups obviously require a little more concentrated attention than is necessary for the average patient.

13. Care should be exercised in the separation of any two patients who are known to be mutually antagonistic.

14. A patient who is reported as having been restless at night should receive special supervision during the following day.

15. A graphic representation of the fluctuations in the accident rate from week to week would serve as a constant reminder of the watchfulness required for prevention of avoidable accidents. This chart should be posted in the medical office, and should be brought up to date each week.

16. The installation of anti-scald valves to prevent burns from hot water is desirable.

Summary.

Routine reports of all accidental injuries to patients of Danvers State Hospital were collected for a two-year period, including even those accidents which were so trifling as to result in no visible injury. These reports were analysed statistically with reference to such factors as age of patient, time and place of occurrence, causative factors, nature of injury, treatment required, and personal responsibility for the accident.

The number of accidents reported for the two-year period was 4755, an average of slightly over one accident per year for each patient of the resident population.

The accident rate for female patients was appreciably higher than for male patients.

The very young patients and the very aged ones suffered more accidents, in proportion to their numbers, than those of any intermediate age group.

More accidents occurred in the latter part of the forenoon than at any other time of day. The accident rate per hour was lower at meal times than at any other time of the waking day.

The most common cause of accidents was personal violence on the part of some other patient. The most common weapon used by patients, especially female patients, was a shoe held in the hand. With or without a weapon, there were many more assaults reported among the women than among the men.

The accidents include 56 suicidal attempts, three of which resulted in death.

The accidents counted as serious number 413, or 8.7 per cent of the total number. These included 106 fractures and 251 lacerations which required closure by sutures. A detailed analysis was made of these serious accidents, especially with reference to their causes.

This study offers definite indication that many accidents are directly traceable to over-crowding of wards, insufficiency of personnel, and other environmental factors which are beyond the immediate control of any individual state hospital. However, without waiting for desirable changes which should be brought about gradually, we can offer recommendations concerning a few practical measures which may be expected to lower the accident rate. For example, so simple an expedient as the use of rubber-soled shoes in the female wards might render some of the unavoidable accidents less serious.

We believe that similar studies conducted in other institutions, based essentially upon reports of routine investigations, might contribute importantly to our resources for prevention of accidental injuries in state hospitals.

NOTE.—The authors wish to acknowledge the assistance, in framing this report, of Neil A. Dayton, M. D., Leo Maletz, M. D., and Grace H. Kent, Ph. D.

A REVIEW OF THE RESULTS OF THE PHARMACOLOGICAL SHOCK THERAPY AND THE METRAZOL CONVULSIVE THERAPY IN NEW YORK STATE.*

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AND
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For the past two years we have submitted reports on the results of insulin shock treatment of schizophrenia in the New York state hospitals of the Department of Mental Hygiene. Last year we included a report on results obtained by the use of metrazol. We propose today to bring these results up to date and draw from them what conclusions we may.

In San Francisco, last year, my report was based on 1356 cases of dementia præcox treated with insulin.[1] Included were 1039 cases which formed the basis of a statistical study by Dr. Malzberg, senior statistician for the New York State Department of Mental Hygiene.[2] Since this latter number have recently been studied further as to their present mental status, we shall use these original 1039 cases instead of the 1356 mentioned above for purposes of comparison. Actually the results are approximately the same in the two groups. Of these 1039 patients, 134, or 12.9 per cent, were considered recovered after the completion of treatment; 282, or 27.1 per cent, were much improved, and 263, or 25.3 per cent, were improved. Those showing some degree of improvement thus totaled 679, or 65.4 per cent. These results were compared with the outcome of treatment among a group of 1039 first admissions to the New York Civil State Hospitals who did not receive any form of shock therapy. In selecting these latter cases we maintained the same total for each hospital, in the same sex proportion and with the same distribution of types of dementia præcox, in order to make the comparison with the treated group as valid as possible. In the control group 3.5 per cent were discharged as

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Ill., May 8-12, 1939.

recovered, 11.2 per cent as much improved and 7.4 per cent as improved. Some degree of improvement was thus shown in 22.1 per cent of the controls as compared with 65.4 per cent of the insulin-treated patients. We note especially that the latter group had a recovery rate of 12.9 per cent as compared to 3.5 per cent for the controls. Since the insulin group included a large proportion of chronic cases, whereas the controls consisted entirely of first admissions, the contrast in favor of the former group takes on a still greater significance.

An additional 718 patients have been treated since February, 1938, making a grand total of 1757 cases. Of these 195, or 11.1 per cent, were described as recovered at the termination of treatment; 466, or 26.5 per cent, were much improved, and 457, or 25.4 per cent, were improved. Those showing some degree of improvement totaled 1118, or 63 per cent. These figures are in very good agreement with the results of the preceding 1039 cases reported by Dr. Malzberg and the 1356 cases reported by me at San Francisco. They are significantly in excess of the percentage of improvement shown by the control group.

The treatment with metrazol, which I reported at San Francisco, was based on a total of 523 cases.[1] Twenty-three, or 4.4 per cent, were reported recovered after treatment; 51, or 9.8 per cent, were considered much improved; while 168, or 32.1 per cent, were improved. The rates of the recovered and the much improved cases were decidedly less than those reported due to insulin, but the rate of "improvement only" was somewhat higher. Another analysis on a larger scale was made by Dr. H. M. Pollock and was based on a series of 1140 dementia præcox patients receiving metrazol treatment.[3] This brings the report up to date. Of this group 18, or 1.6 per cent, were recovered; 113, or 9.9 per cent, were much improved and 279, or 24.5 per cent, were improved at the completion of treatment. A total of 36.0 per cent showed some degree of improvement.

Table 1 classifies the patients according to the hospital in which they were treated with insulin and also shows the condition of the patient at the conclusion of treatment. From this table it will be seen that not all the hospitals in New York State are in agreement as to the value of the hypoglycemic therapy. The Brooklyn State Hospital treated the largest number, while Middletown

TABLE 1.

TABLE 1.

OUTCOME OF INSULIN TREATMENT OF DEMENTIA PRÆCOX PATIENTS IN THE NEW YORK CIVIL STATE HOSPITALS.

State hospitals.	Total.			Recovered.			Much improved.			Improved.			Unimproved.			Died.		
	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.
Binghamton	28	40	68	5	10	15	9	14	23	6	5	11	8	11	19	..	..	..
Brooklyn	144	180	324	41	29	70	51	40	91	29	63	92	21	47	68	2	1	3
Buffalo	68	48	116	4	3	7	22	15	37	26	11	37	16	19	35	..	..	..
Central Islip	92	76	168	2	3	5	28	20	48	28	5	33	31	47	78	3	1	4
Creedmoor	51	25	76	6	4	10	14	8	22	14	3	17	16	10	26	1	..	1
Gowanda	16	19	35	6	7	13	5	6	11	3	3	6	1	2	3	1	1	2
Harlem Valley	74	53	127	9	9	18	24	17	41	35	9	44	6	18	24	..	..	..
Hudson River	50	50	100	1	1	2	5	1	6	10	14	24	32	33	65	2	1	3
Kings Park	41	82	123	...	3	3	14	20	34	6	21	27	21	37	58	..	1	1
Manhattan	27	18	45	...	...	...	4	4	8	12	3	15	10	9	19	1	2	3
Marcy	32	36	68	1	1	2	15	9	24	8	10	18	8	15	23	..	1	1
Middletown	7	18	25	...	1	1	2	4	6	...	4	4	5	9	14	..	..	..
Pilgrim	28	46	74	...	...	...	6	9	15	8	15	23	13	22	35	1	..	1
Psychiatric Institute	44	47	91	4	16	20	4	12	16	11	4	15	25	15	40	..	..	..
Rochester	40	45	85	...	...	...	17	10	27	15	18	33	8	17	25	..	..	..
Rockland	30	19	49	...	...	...	8	6	14	9	5	14	13	8	21	..	..	..
St. Lawrence	22	35	57	2	2	4	11	5	16	5	8	13	4	20	24	..	..	..
Utica	40	28	68	17	3	20	7	5	12	6	8	14	10	11	21	..	1	1
Willard	38	20	58	2	3	5	10	5	15	12	5	17	14	7	21	..	..	..
Total	872	885	1757	100	95	195	256	210	466	243	214	457	262	357	619	11	9	20

TABLE 2.

PER CENT DISTRIBUTION OF OUTCOME OF TREATMENT OF DEMENTIA PRÆCOX PATIENTS IN THE NEW YORK CIVIL STATE HOSPITALS.

State hospital.	Insulin-treated patients.					Control group.					
	Recovered.	Much improved.	Improved.	Unimproved.	Died.	Recovered.	Much improved.	Improved.	Unimproved.	Still in hospital.	Died.
Binghamton	22.1	33.8	16.2	27.9	...	2.0	18.4	6.1	8.2	61.2	4.1
Brooklyn	21.6	28.1	28.4	21.0	0.9	5.6	16.9	8.5	2.3	56.5	10.2
Buffalo	6.0	31.9	31.9	30.2	...	9.0	17.9	4.5	3.0	62.7	3.0
Central Islip	3.0	28.6	19.6	46.4	2.4	...	15.8	7.9	2.6	71.1	2.6
Creedmoor	13.2	28.9	22.4	34.2	1.3	2.6	5.3	2.6	...	84.2	5.3
Gowanda	37.1	31.4	17.1	8.6	5.7	12.5	3.1	...	12.5	68.8	3.1
Harlem Valley	14.2	32.3	34.6	18.9	...	4.3	5.3	3.2	5.3	79.8	2.1
Hudson River	2.0	6.0	24.0	65.0	3.0	1.3	8.0	8.0	10.7	64.0	8.0
Kings Park	2.4	27.6	22.0	47.2	0.8	1.8	12.3	7.0	7.0	71.9	...
Manhattan	...	17.8	33.3	42.2	6.7	...	...	20.8	...	76.0	4.0
Marcy	2.9	35.3	26.5	33.8	1.5	...	32.0	4.0	4.0	60.0	...
Middletown	4.0	24.0	16.0	56.0	...	...	11.1	11.1	...	66.7	11.1
Pilgrim	...	20.3	31.1	47.3	1.4	2.6	7.9	13.2	2.6	65.8	7.9
Psychiatric Institute	22.0	17.6	16.5	44.0	...	3.2	4.8	11.3	56.5	24.2	...
Rochester	...	31.8	38.8	29.4	...	...	10.4	8.3	2.1	77.1	2.1
Rockland	...	28.6	28.6	42.9	...	2.1	12.8	17.0	...	63.8	4.3
St. Lawrence	7.0	28.1	22.8	42.1	...	...	2.1	...	10.4	83.3	4.2
Utica	29.4	17.6	20.6	30.9	1.5	9.1	6.1	12.1	3.0	69.7	...
Willard	8.6	25.9	29.3	36.2	...	3.3	6.7	...	3.3	80.0	6.7
Total	11.1	26.5	26.0	35.2	1.1	3.5	11.2	7.4	7.5	65.8	4.6

and Gowanda State Hospitals treated very few. Some of the institutions discontinued insulin treatment and have limited themselves to the use of metrazol. One institution, however, after using metrazol for a time, has again returned to the use of insulin. Middletown and Gowanda are homeopathic hospitals and, as insulin therapy is hardly in accord with the homeopathic belief, this fact may account for so few cases being treated at these institutions. The shift to metrazol by other hospitals can only be explained by the fact that metrazol is so much easier to administer and requires little skill. It does not take the time necessary in the insulin treatment and larger numbers can be treated. If the results obtained with metrazol compared favorably with those obtained with insulin there would be no question as to which drug would be more desirable.

TABLE 3.

CONDITION OF PATIENTS WITH DEMENTIA PRÆCOX AT THE TERMINATION OF TREATMENT, IN PER CENT, ACCORDING TO TYPE OF TREATMENT.

	Total.	Recovered.	Much improved.	Improved.	Un-improved.	Died.
Insulin	100.0	11.1	26.5	26.0	35.2	1.1
Metrazol	100.0	1.6	9.9	24.5	63.5	0.5
Control group....	100.0	3.5	11.2	7.4	73.3	4.6

In the face of the figures which we are submitting today it seems that such a shift is hardly justified. Table 3 summarizes the results of insulin and metrazol treatment and compares both with the control group of patients. It will be observed that *the recovery rate for the hypoglycemic treatment exceeded that of the metrazol group by a ratio of 6.9 to 1; the rate of much improved by 2.7 to 1.* Of the insulin group 63 per cent showed some degree of improvement while in the metrazol group only 36 per cent were improved. *Clearly, the results following insulin treatment far exceeded those obtained with metrazol.* Furthermore, *it will be observed that the recovery rate following treatment with metrazol was less than half that obtained with the control group,* and the rate of much improved was also higher in the control group. Only with respect to the number showing *"only improvement" was the metrazol group in excess,* the percentage being 24.5

for those treated with metrazol as against 7.4 per cent in the control group. Even this, however, is a statistical delusion for the control group included a large proportion (65.8 per cent) who were still in the hospital one year after admission and whose condition had not been otherwise classified. Undoubtedly a certain proportion showed some degree of improvement, insufficient, however, to justify discharge or parole. Experience showed that an adjustment for this factor should bring the improvement rate in the control group to approximately 20 per cent, which compared favorably with the rate of the metrazol group.

It is well known that there have been many accidents such as dislocations and fractures as complications of the treatment with metrazol. There has been an occasional report of fracture of the spine. In our own experience at Harlem Valley State Hospital two such cases occurred and early led us to believe that metrazol alone was not only not particularly effective but that it was also a very dangerous drug and one not to be taken lightly or used indiscriminately. In selected cases in combination with insulin it has proved itself of value and in our opinion should be used only in such combinations. By correspondence with a number of institutions my attention has been called to the fact that fractures of the spine were not infrequent and the New York Psychiatric Institute has reported in the Journal of the American Medical Association fractures of the spine in 43 per cent of the patients treated with metrazol.[4] When one contrasts with this the fact that only seven deaths occurred during the course of insulin treatment during the past year, not all of which can be ascribed directly to the treatment, one is led to the conclusion that the improvement in, and additional experience with, the technique has largely eliminated the dangers of the hypoglycemic therapy. It would seem, therefore, that in selecting a treatment for cases of dementia præcox there should be no question as to the superiority of the hypoglycemic over the convulsive therapy.

Table 4 shows the outcome of insulin treatment according to the age of the patients at the beginning of treatment. Those aged 15 to 19 years had a recovery rate of 17.6 per cent. With one exception, the recovery rate declined as the age at beginning of treatment increased. If we combine all degrees of improvement, we find a

OUTCOME OF INSULIN TREATMENT ACCORDING TO AGE AT BEGINNING OF TREATMENT.

Age (years).	Total.			Recovered.			Much Improved.			Improved			Unimproved.			Died.		
	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.
NUMBER.																		
Under 10	2	...	2	...	...	...	...	...	...	...	...	...	2	...	2	...	...	...
10-14	4	6	10	...	...	...	2	...	2	...	2	2	2	4	6	...	...	...
15-19	92	78	170	16	14	30	28	18	46	22	19	41	26	27	53	...	...	...
20-24	225	180	405	27	23	50	67	56	123	67	40	107	61	61	122	3	...	3
25-29	225	220	445	25	18	43	62	55	117	67	62	129	67	83	150	4	2	6
30-34	177	209	386	19	25	44	54	42	96	43	52	95	60	88	148	1	2	3
35-39	99	113	212	8	11	19	28	25	53	34	24	58	26	50	76	3	3	6
40-44	34	57	91	4	4	8	11	12	23	8	10	18	11	29	40	...	2	2
45-49	11	19	30	1	...	1	3	2	5	2	4	6	5	13	18	...	...	...
50-54	2	3	5	...	...	...	...	...	...	...	1	1	2	2	4	...	...	...
55-59	1	...	1	...	...	...	1	...	1	...	...	...	...	...	...	...	...	...
Total	872	885	1757	100	95	195	256	210	466	243	214	457	262	357	619	11	9	20
PER CENT.																		
Under 10	100.0	100.0	100.0	...	...	...	...	...	...	...	...	...	100.0	...	100.0	...	...	...
10-14	100.0	100.0	100.0	...	...	...	50.0	...	20.0	...	33.3	20.0	50.0	66.7	60.0	...	...	...
15-19	100.0	100.0	100.0	17.4	17.9	17.6	30.4	23.1	27.1	23.9	24.4	24.1	28.3	34.6	31.2	...	...	...
20-24	100.0	100.0	100.0	12.0	12.8	12.3	29.8	31.1	30.4	29.8	22.2	26.4	27.1	33.9	30.1	1.3	...	0.7
25-29	100.0	100.0	100.0	11.1	8.2	9.7	27.6	25.0	26.3	29.8	28.2	29.0	29.8	37.7	33.7	1.8	0.9	1.3
30-34	100.0	100.0	100.0	10.7	12.0	11.4	30.5	20.1	24.9	24.3	24.9	24.6	33.9	42.1	38.3	0.6	1.0	0.8
35-39	100.0	100.0	100.0	8.1	9.7	9.0	28.3	22.1	25.0	34.3	21.2	27.4	26.3	44.2	35.8	3.0	2.7	2.8
40-44	100.0	100.0	100.0	11.8	7.0	8.8	32.4	21.1	25.3	23.5	17.5	19.8	32.4	50.9	44.0	...	3.5	2.2
45-49	100.0	100.0	100.0	9.1	...	3.3	27.3	10.5	16.7	18.2	21.1	20.0	45.5	68.4	60.0	...	...	...
50-54	100.0	100.0	100.0	...	...	...	...	...	...	...	33.3	20.0	100.0	66.7	80.0	...	...	...
55-59	100.0	100.0	100.0	...	...	...	100.0	...	100.0	...	...	...	...	...	...	...	...	...
Total	100.0	100.0	100.0	11.5	10.7	11.1	29.4	23.7	26.5	27.9	24.2	26.0	30.0	40.3	35.2	1.3	1.0	1.1

decline from approximately 70 per cent among those aged 15 to 19, and 20 to 24, to 40 per cent among those aged 45 to 49 years.

A more significant relation is found between the duration of the psychosis before treatment and the outcome of insulin treatment. The data are shown in Table 5. The recovery rate declined from a maximum of 26.1 per cent among those with a duration of less than a month to 2.1 per cent among those with a duration of 11 to 14 years. There were no recoveries among those with a duration of 15 years or more. The percentage showing any degree of improvement declined from 91.3 among those with a duration of less than a month to 37.5 among those with a duration of 11 to 14 years.

Since the outcome of treatment is related to both the duration of the psychosis before treatment and the age of the patient at the beginning of treatment, it will be of interest to note how recovery and improvement rates change with different combinations of the two variables.

Table 6 shows, for example, that of those aged 15 to 19 years at the beginning of treatment, who had been ill for less than six months prior to treatment, 37.1 per cent were discharged as recovered. The next class, consisting of those aged 20 to 24 years and with a duration of 6 to 11 months, had a recovery rate of 17.2 per cent. As we proceed along the diagonal, we find a downward trend in the recovery rate. Except for random fluctuations, it will be found that, in general, the recovery rate decreases as the age of the patient increases and as the duration of the disease increases.

We shall next consider the outcome of treatment in relation to type of dementia præcox. Reference to Table 7 shows that the catatonic type had the highest recovery rate (16.8 per cent), the simple type the lowest (2.1 per cent). Combining all degrees of improvement, very little difference is found among the simple, catatonic and paranoid types. The lowest rate was shown by hebephrenics.

However, these rates are influenced to a large degree by the duration of the psychosis before treatment. As the average duration differs in the several types, it is necessary to eliminate this factor. In order to do this, the recovery and improvement rates were standardized on the assumption that each type had the same distribution with respect to duration of the disease as all types

TABLE 5.

OUTCOME OF INSULIN TREATMENT ACCORDING TO DURATION OF PSYCHOSIS BEFORE TREATMENT.

TABLE 5.

OUTCOME OF INSULIN TREATMENT ACCORDING TO DURATION OF PSYCHOSIS BEFORE TREATMENT.

Duration of psychosis.	Total.			Recovered.			Much improved.			Improved.			Unimproved.			Died.		
	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.
NUMBER.																		
Less than 1 month	17	6	23	5	1	6	8	2	10	2	3	5	2	...	2	...	...	...
1-3 months	101	121	222	23	32	55	44	47	91	21	21	42	12	21	33	1	...	1
4-6 months	83	76	159	17	12	29	33	31	64	14	16	30	17	17	34	2	...	2
7-12 months	94	90	184	14	14	28	31	33	64	18	20	38	30	22	52	1	1	2
1-2 years	280	284	564	32	26	58	71	60	131	92	74	166	83	122	205	2	2	4
3-5 years	168	186	354	6	6	12	47	24	71	55	52	107	57	104	161	3	...	3
6-10 years	99	90	189	2	4	6	17	11	28	33	21	54	46	49	95	1	5	6
11-14 years	22	26	48	1	...	1	4	2	6	5	6	11	11	17	28	1	1	2
15-19 years	6	3	9	...	...	...	1	...	1	2	...	2	3	3	6	...	...	...
20 years and over	2	3	5	...	...	...	...	...	...	1	1	2	1	2	3	...	...	...
Total	872	885	1757	100	95	195	256	210	466	243	214	457	262	357	619	11	9	20
PER CENT.																		
Less than 1 month	100.0	100.0	100.0	29.4	16.7	26.1	47.1	33.3	43.5	11.8	50.0	21.7	11.8	...	8.7	...	...	...
1-3 months	100.0	100.0	100.0	22.8	26.4	24.8	43.6	38.8	41.0	20.8	17.4	18.9	11.9	17.4	14.9	1.0	...	0.5
4-6 months	100.0	100.0	100.0	20.5	15.8	18.2	39.8	40.8	40.3	16.9	21.1	18.9	20.5	22.4	21.4	2.4	...	1.3
7-12 months	100.0	100.0	100.0	14.9	15.6	15.2	33.0	36.7	34.8	19.1	22.2	20.7	31.9	24.4	28.3	1.1	1.1	1.1
1-2 years	100.0	100.0	100.0	11.4	9.2	10.3	25.4	21.1	23.2	32.9	26.1	29.4	29.6	43.0	36.3	0.7	0.7	0.7
3-5 years	100.0	100.0	100.0	3.6	3.2	3.4	28.0	12.9	20.1	32.7	28.0	30.2	33.9	55.9	45.5	1.8	...	0.8
6-10 years	100.0	100.0	100.0	2.0	4.4	3.2	17.2	12.2	14.8	33.3	23.3	28.6	46.5	54.4	50.3	1.0	5.6	3.2
11-14 years	100.0	100.0	100.0	4.5	...	2.1	18.2	7.7	12.5	22.7	23.1	22.9	50.0	65.4	58.3	4.5	3.8	4.2
15-19 years	100.0	100.0	100.0	...	...	...	16.7	...	11.1	33.3	...	22.2	50.0	100.0	66.7	...	...	...
20 years and over	100.0	100.0	100.0	...	...	...	...	...	...	50.0	33.3	40.0	50.0	66.7	60.0	...	...	...
Total	100.0	100.0	100.0	11.5	10.7	11.1	29.4	23.7	26.5	27.9	24.2	26.0	30.0	40.3	35.2	1.3	1.0	1.1

TABLE 6.

PER CENT OF DEMENTIA PRÆCOX PATIENTS RECOVERED.

Age at beginning of treatment (years).	Duration of disease prior to treatment.							
	Less than 6 months.	6-11 months.	1-1.49 years.	1.50-1.99 years.	2 years.	3 years.	4 years.	5 years and over.
Under 15	...	...	...	...	...	...	...	...
15-19	37.1	6.9	16.7	11.1	4.2	10.5	20.0	...
20-24	16.5	17.2	19.0	9.7	11.3	7.9	...	2.1
25-29	19.8	18.3	12.0	10.7	6.9	5.4	...	...
30-34	24.3	24.3	7.5	16.2	5.9	11.1	...	3.3
35-39	14.8	16.7	35.3	6.7	...	5.6	...	3.1
40-44	14.3	10.0	14.3	28.6	18.2	...	...	3.4
45-49	50.0	...	...	...	...	...	...	...
50 and over	...	...	...	...	...	...	...	...

TABLE 7.

OUTCOME OF INSULIN TREATMENT ACCORDING TO TYPE OF DEMENTIA PRÆCOX.

Types.	Total.			Recovered.			Much improved.			Improved.			Unimproved.			Died.		
	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.
NUMBER.																		
Simple	33	15	48	...	1	1	11	2	13	12	4	16	10	8	18	...	...	...
Hebephrenic	175	191	366	13	7	20	43	46	89	51	45	96	68	91	159	...	2	2
Catatonic	243	321	564	45	50	95	63	84	147	62	83	145	68	103	171	5	1	6
Paranoid	415	354	769	41	37	78	136	76	212	118	82	200	114	153	267	6	6	12
Others and unclassified	6	4	10	1	...	1	3	2	5	...	...	...	2	2	4	...	...	...
Total	872	885	1757	100	95	192	256	210	466	243	214	457	262	357	619	11	9	20
PER CENT.																		
Simple	100.0	100.0	100.0	...	6.7	2.1	33.3	13.3	27.1	36.4	26.7	33.3	30.3	53.3	37.5	...	...	...
Hebephrenic	100.0	100.0	100.0	7.4	3.7	5.5	24.6	24.1	24.3	29.1	23.6	26.2	38.9	47.6	43.4	...	...	...
Catatonic	100.0	100.0	100.0	18.5	15.6	16.8	25.9	26.2	26.1	25.5	25.9	25.7	28.0	32.1	30.3	2.1	0.3	1.1
Paranoid	100.0	100.0	100.0	9.9	10.5	10.1	32.8	21.5	27.6	28.4	23.2	26.0	27.5	43.2	34.7	1.4	1.7	1.6
Others and unclassified	100.0	100.0	100.0	16.7	...	10.0	50.0	50.0	50.0	...	...	...	33.3	50.0	40.0	...	...	...
Total	100.0	100.0	100.0	11.5	10.7	11.1	29.4	23.7	26.5	27.9	24.2	26.0	30.0	40.3	35.2	1.3	1.0	1.1

combined. On this basis, the simple type had a recovery rate of 1.9±1.3 per cent, the hebephrenics had a recovery rate of 6.8±0.9 per cent, the catatonics had a recovery rate of 14.5±1.0 per cent, and the paranoids had a recovery rate of 10.5±0.8 per cent. Because of the large probable error, the recovery rate for the simple type is without statistical significance. Among the other three groups, the catatonics had the highest recovery rate, the hebephrenics the lowest. If we consider all degrees of improvement, we obtain the following percentages: simple, 67.7±4.6; hebephrenic, 58.9±1.7; catatonic, 65.2±1.3; paranoid, 64.2±1.2. Unfortunately, the probable errors are so large that none of the differences can be considered of statistical significance. In order to be certain that improvement rates vary in accordance with the type of dementia præcox we require larger groups of patients.

Thus far we have shown that the condition of the patients with dementia præcox at the conclusion of such treatment contrasts very favorably with that obtained through non-specific methods. Before reaching any judgment as to the ultimate efficacy of such treatment, it is necessary to observe the condition of the patient at later intervals. For this reason each patient of the original 1039 cases was reinvestigated and the condition observed approximately one year later, after the termination of such treatment. As 13 patients had died during the course of treatment Table 8 shows the later outcome for the remaining 1026 patients. Of the 1026 cases, 132, or 12.9 per cent, were described as recovered at the time of the follow-up; 170, or 16.6 per cent, were much improved and 201, or 19.6 per cent, were improved. The total improvement included 502, or 49.0 per cent. The group described as improved had totaled originally 679, or 65.4 per cent. There was, therefore, a reduction of 176 patients in the group showing improvement after a period of slightly more than a year. There was a shift of only two patients in the total described as recovered, but the total of much improved decreased from 282 to 170 and the total of improved only was reduced from 263 to 201. We may conclude that half of the patients showed some degree of improvement after the lapse of a year. There was much shifting in the results, however. Thus, of the 134 patients described as recovered at the termination of the treatment only 73 were so described at

TABLE 8.

OUTCOME OF INSULIN TREATMENT OF PATIENTS WITH DEMENTIA PRÆCOX, SHOWING CORRELATION OF CONDITION AT TERMINATION OF TREATMENT AND CONDITION APPROXIMATELY ONE YEAR LATER.

Condition at termination of treatment.	Condition at time of follow-up.																					
	Total.			Recovered.			Much improved.			Improved.			Unimproved.			Unknown.			Died.		
	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.
NUMBER.																					
Recovered ...	71	63	134	38	35	73	9	12	21	12	5	17	11	9	20	1	...	1	...	2	2
Much improved	158	124	282	23	20	43	58	53	111	41	14	55	31	36	67	3	1	4	2	...	2
Improved	133	130	263	5	6	11	15	12	27	54	54	108	59	57	116	...	1	1	...	...	...
Unimproved .	156	191	347	2	3	5	4	7	11	10	11	21	132	162	294	4	5	9	4	3	7
Total	518	508	1026	68	64	132	86	84	170	117	84	201	233	264	497	8	7	15	6	5	11
PER CENT.																					
Recovered ...	100.0	100.0	100.0	53.5	55.6	54.5	12.7	19.0	15.7	16.9	7.9	12.7	15.5	14.3	14.9	1.4	...	0.7	...	3.2	1.5
Much improved	100.0	100.0	100.0	14.6	16.1	15.2	36.7	42.7	39.4	26.0	11.3	19.5	19.6	29.0	23.8	1.9	0.8	1.4	1.3	...	0.7
Improved	100.0	100.0	100.0	3.8	4.6	4.2	11.3	9.2	10.3	40.6	41.5	41.1	44.4	43.8	44.1	...	0.8	0.4	...	...	...
Unimproved .	100.0	100.0	100.0	1.3	1.6	1.4	2.6	3.7	3.2	6.4	5.8	6.1	84.6	84.8	84.7	2.6	2.6	2.6	2.6	1.6	2.0
Total	100.0	100.0	100.0	13.1	12.6	12.9	16.6	16.5	16.6	22.6	16.5	19.6	45.0	52.0	48.4	1.5	1.4	1.5	1.2	1.0	1.1

the time of the follow-up. Of the remainder, 21 were classified as much improved, 17 as improved, and 20 as unimproved. Of the original 282 who comprised the group of much improved, 111 remained in this category a year later, and 43 had improved to a degree justifying their being considered recovered. On the other hand 162 had relapsed, 55 being described as improved and 57 as unimproved. Of the 263 who had originally been considered improved 116 had relapsed during the ensuing year, but 38 showed much improvement, of whom 11 were considered recovered. Finally of the 347 originally reported unimproved, 5 later showed a complete recovery, 11 were much improved and 21 were improved. It appears, therefore, that though some patients showed various degrees of relapse in their mental status, following the termination of insulin therapy, an appreciable total continued to improve, a phenomenon also noted by Dr. Sakel who likened it to a process of ripening.

There is a great lack of suitable material for the establishment of a norm. The study which seems to approach closest in methodology is that by R. G. Fuller [5] who reported on the after-histories of 242 cases of dementia præcox discharged from the New York State civil hospitals prior to the introduction of insulin therapy. At the close of a period of 10 years after discharge from the hospital, he found that 43.8 per cent of the patients were living in the general community; 43.3 per cent were in a mental hospital and 12.8 per cent had died. We cannot assume, of course, that all those in the community were recovered or improved, so that it is highly reasonable to assume that less than 40 per cent of the group could be classified as improved in some degree. Furthermore, all cases had been selected by Fuller on the basis of discharge after a period of successful parole. They, therefore, constituted a favorable selection with respect to final outcome. Despite this fact, however, it is clear that the results obtained following the use of insulin must be considered significantly better than that for the control group.

On can draw certain conclusions from a study of these tables as to the type of case likely to be benefited most by insulin treatment. I believe that one is justified on the basis of our experience thus far to consider the following factors as of definite prognostic value.

1. Duration of Psychosis.

The rate of recovery seems to vary definitely with the duration of the illness prior to the treatment.

2. Type of Dementia Præcox.

The best results are obtained in the catatonic and paranoid types while the hebephrenic and simple types are not nearly so favorable.

3. The Personality of the Patient Before Development of the Illness.

A relatively well adjusted personality prior to the onset of the psychosis definitely affects the outcome of the treatment. This is seen especially in the follow-up cases. Those patients who were considered recovered at the completion of the treatment and whose age at the onset of the illness was between 25 and 34 retained their recovery to a much higher degree than those whose illness began between the ages of 15 and 24. The older group, therefore, was more fully developed emotionally, able to survive the difficulties of the adolescent period and in most cases there were definite precipitating factors at the time of onset. The younger group on the contrary usually developed a psychosis for no known or demonstrable reason and were unable to pass through the emotional adjustment during the adolescent period. While there is this difference between the two age groups one year after treatment, at the time that the treatment was completed the recovery rate was the same in each. In other words, the patients in the older group who do recover as a result of the treatment, when the treatment is instituted within the first six months after the illness has begun, are better equipped to retain their improvement.

We come finally to the question of parole and discharge. Desirable as it is to improve the mental status of a patient, this is not sufficient in itself. We must strive to improve the patient to such a degree that he is able to leave the hospital and assume a normal socialized status in the community. The degree of success in this direction may be considered in the data in Table 9.

Of the 1026 patients, 413, or 40.3 per cent, remained in the hospital after treatment had been terminated; 564, or 55.0 per cent,

TABLE 9.

CONDITION OF PATIENTS WITH DEMENTIA PRÆCOX DURING FOLLOW-UP PERIOD WITH RESPECT TO PAROLE AND DISCHARGE, CORRELATED WITH CONDITION AT TERMINATION OF INSULIN TREATMENT.

Condition at termination of treatment.	Total.			Number in hospital during entire follow-up period.			Number discharged upon termination of treatment.			Number paroled upon termination of treatment.			Number discharged after parole.			Number who relapsed on parole.						Number who relapsed after discharge.		
																Number of individuals.			Number of relapses.					
	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.
Recovered	71	63	134	...	...	...	1	4	5	70	59	129	47	39	86	18	13	31	19	14	33	6	6	12
Much improved	158	124	282	13	7	20	4	4	8	141	113	254	67	53	120	54	44	98	54	46	100	6	4	10
Improved	133	130	263	56	63	119	2	6	8	75	61	136	23	22	45	37	26	63	39	26	65	2	4	6
Unimproved	156	191	347	118	156	274	17	11	28	21	24	45	4	5	9	9	5	14	9	5	14	11	5	16
Total	518	508	1026	187	226	413	24	25	49	307	257	564	141	119	260	118	88	206	121	91	212	25	19	44

HARLEM VALLEY STATE HOSPITAL

PHARMACOLOGICAL SHOCK TREATMENT RECORD

(Developed by Frostig, Rossman, Cline and Schwoerer)

Name..... Diagnosis.....

Identification No..... Duration of Illness.....

Date	Insulin Day	Shock Day	Insulin Dose	Metrazol Dose	Hours	Loss of Consciousness	Hyperkinetic	Tonic tetanic	Babinski	Lid	Extensor spasm	Pupil	Medullary symptoms	Cornea	Termination	Comments
Total																
					1											
					2											
					3											
					4											
					5											
					6											
					1											
					2											
					3											
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					6											
4/25/39	20	12	120		1											
					2											
					3											
					4											
					5											
					6											

ILLUSTRATION

Record of One Day's Treatment

Total (over) Weight.....lbs.

Schematic Illustration of the Insulin Effect

	Cerebral Syndrome	Subcortical Syndrome Hyperkinetic Symptoms most prominent	Midbrain Syndrome Tonic-Tetanic Symptoms most prominent	Medullary Syndrome
0-2 hours	Drowsiness Sleepiness Sleep Defective Orientation Hunger Row Activation of Psychosis Loss of Contact Coma			
2-3 1/2 hours		Myoclonic Twitchings Clonic Twitchings Primitive Movements Choreic-Athetoid Movements Primitive Reflexes Hemibalismus Torsion Spasms Salivation Convulsive Seizure		
3-4 1/2 hours			Convulsive Seizure Tonic-Tetanic Spasm Dilated Pupils Tonic Torsion Spasm Babinski Oppenheim Rossolimo Hoffman Absent Lid Reflex Tonic Laryngo Spasm	
4-5 hours			Tonic Extensor Spasm	Tonic Extensor Spasm Dilated Pupils Rapid Pulse Magnus De Kleyn Diminished Reflexes Hypotonia Fixed Pin-point Pupils Slow Pulse Pallor Depression of Respiration Sluggish or Absent Corneal Reflex

were paroled; and 49, or 4.8 per cent, were discharged without being first paroled. Of those who were paroled or discharged, 250, or 40.8 per cent, subsequently relapsed. Of the total 1026 patients who were treated with insulin, 25.8 per cent had been discharged within a period of approximately one year after the termination of such treatment. According to Fuller,[6] 18.5 per cent of a large group of patients with dementia præcox treated before the insulin era had been discharged within one year after first admission, and 24.2 per cent had been discharged within two years. These figures do not appear to indicate very significant differences. But we must make allowance for the more favorable prognosis for first admissions and when this is done it appears that treatment with insulin makes possible a higher discharge rate.

The value of the insulin treatment is now definitely established. Every institution that has given it a fair trial has found it to be effective. There remains, however, the very great necessity of adopting not only a uniform application of the technique but also a standardized method of recording the results. Frostig was able to demonstrate by a study of his cases that the neurological signs during hypoglycemia developed as a rule in a definite order. He applied his findings to produce a chart for recording these signs in the treatment of acute cases. During the past year his plan has been extended at the Harlem Valley State Hospital by Frostig, Rossman, Cline and Schwoerer to include the chronic cases as well. We believe that we now have a simple chart that enables one to record the progression of symptoms during each treatment period in such a manner that one can see at a glance whether a patient has been adequately treated. Records obtained in this way will make it possible to critically examine and compare the results and provide information that will lead to more uniform success. The charts and the studies from which they are obtained are demonstrated in detail in papers to be published soon.

Summary and Conclusion.

1. One thousand seven hundred and fifty-seven patients suffering from dementia præcox have been treated thus far with insulin hypoglycemia in the New York State Department of Mental Hygiene, and 1140 patients have been treated with metrazol.

2. The recovery and improvement rate in the cases of insulin treated cases is significantly in excess of the rate in the control group. Metrazol, on the other hand, produced even fewer recovered cases than found in cases not treated by any shock therapy.

3. Recent reports indicate that metrazol, besides being not especially effective, also produces serious complications, notably fractured spines. While it is effective if used in selected cases in combination with insulin it is a dangerous drug and should not be used indiscriminately.

4. A follow-up study of cases approximately one year after completion of treatment indicates that a significant proportion of the patients tend to retain their improved state.

5. The prognosis in insulin treatment depends largely on:

(a) Duration of the illness before treatment.

(b) Type of dementia præcox.

(c) Personality prior to the onset of the illness.

6. The adoption of the uniform application of treatment and a standardized method of recording will make possible more uniform success.

BIBLIOGRAPHY.

1. Ross, Dr. John R.: The pharmacological shock treatment of schizophrenia. Am. J. Psychiat., 95: 769-779.

2. Malzberg, Dr. Benjamin: Outcome of insulin treatment of one thousand patients with dementia præcox. Psychiatric Quarterly, XII, 2, 528-553, 1938.

3. Pollock, Dr. Horatio M.: A statistical study of 1140 dementia præcox patients treated with metrazol. Psychiatric Quarterly, XIII, 3, 558-568.

4. Polatin, Dr. Philip, Friedman, Dr. Murray M., Harris, Dr. Meyer N., and Horwitz, Dr. Wm. A.: Vertebral fractures produced by metrazol-induced convulsions. J. A. M. A., Vol. 112, No. 17; pp. 1684-1687, April 29, 1939.

5. Fuller, Raymond G.: What happens to mental patients after discharge from hospital. Psychiatric Quarterly, IX, 1, 95-104.

6. Fuller, Raymond G.: Expectation of hospital life and outcome for mental patients on first admission. Psychiatric Quarterly, IV, 2, 295-323.

RESULTS AND OBSERVATIONS ON THE INSULIN-SHOCK TREATMENT OF SCHIZOPHRENIA.*

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Method.

Insulin-shock therapy has been given since December 1, 1936, at the Pennsylvania Hospital according to the technique originated by Sakel.

The 82 patients reported in this paper received an average of 44.3 days of treatment. Each patient had approximately 106 hours

TABLE I.

Analysis of 82 Completed Insulin Courses.

No. of patients.	No. of treatment days.	Hours of stupor.	Hours of coma.	Seizures.	Total amount of insulin.
82	3,633	8,692.3	164.82	58	344,400
		AVERAGE OF 82 PATIENTS.			
..	44.3	106	2.01	.7	4,200

in stupor and averaged only slightly in excess of two hours in coma during 44 days of treatment. The term coma is used by us to represent a state of unconsciousness associated with complete areflexia. Differences between our figure and those of other workers may very well be merely one of definition. These data were collected on charts similar to that recommended by Frostig.[1] They are presented in Table I.

Our group of patients averaged less than one convulsion per patient. From our experience with convulsive seizures during insulin therapy, we are unable to formulate any opinion as to their therapeutic value. Following a convulsion, certain patients were

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materially improved; others showed no favorable response. There was no definite trend in one direction or the other.

Fortunately no permanently grave complications of treatment have occurred. Those complications which did occur were of temporary duration and in but two cases indicated termination of treatment. In the first case, a right hemiplegia and aphasia occurred in a patient, age 58. This persisted for 10 hours. In the second case, insufficiency of the aortic valve developed during a period of insulin stupor. Other complications observed were the development of irregularities in cardiac rhythms ranging from extra systoles to auricular fibrillation. Periodic breathing of the Cheyne-Stokes type also occurred.

Although some of our remissions have shown a pattern of gradually increasing periods of lucidity as treatment progressed, there have been many interesting variations. Fig. 1 attempts graphically to depict some of these variations. Graph *A* demonstrates a case of acute schizophrenia in which massive abolition of psychotic material occurred following a convulsion. *B* shows the patchy type of response in which complete insight and recovery did not occur until two weeks after the cessation of treatment. Both of these patients remained well and have a high grade of insight into their illness. *C* demonstrates the occurrence of relapse after the first insulin series and remission after the second course of insulin into which metrazol convulsions were interpolated in the final two weeks. This patient has continued well for eight months.

Follow-Up.

The 82 cases of dementia præcox who received insulin-shock treatment from December 1, 1936, to July 10, 1938, have been followed to March 1, 1939. The latest case, therefore, has been followed for eight months and the earliest for 26 months.

It is realized that these cases are few as compared with the large number treated in the New York State Hospital services. In terms of percentages our cases divide as follows.

Of cases classified as recovered and much improved under the New York definitions, and who have held those satisfactory states for at least eight months, we have 31 or approximately 38 per cent. These patients have all been symptom free and able to adjust in the community at their prepsychotic level, although occasionally

slightly below or above it. Insight has varied from complete to partial; it has never been entirely lacking. Emotional relationships to family and friends have been easier in all cases.

Of cases classed as improved we have four or nearly five per cent. These cases showed slight or moderate responses to treatment which have been maintained.

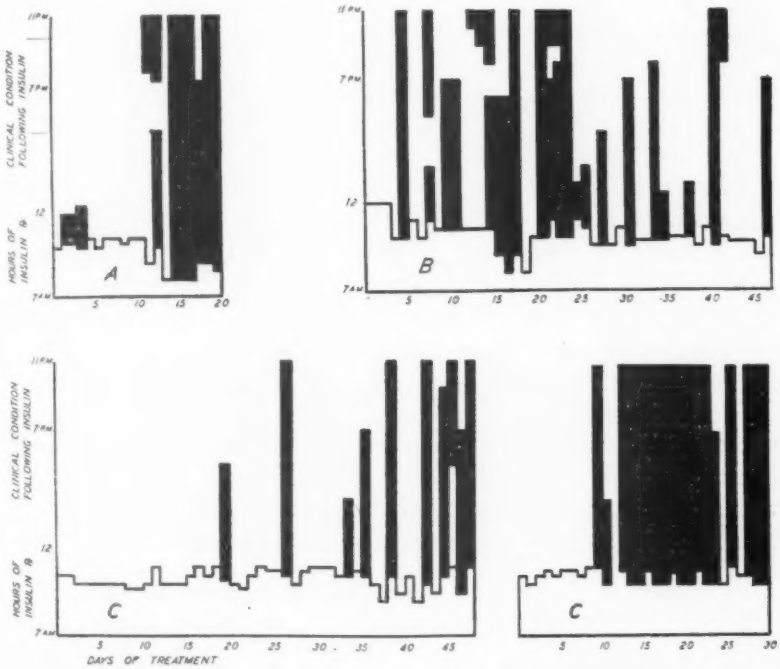


FIG. 1.—Clinical course of three patients A, B, C, during insulin treatment. Insulin given at 7 a. m. Hours of insulin reaction indicated in lower part of graph. Upper part of graph indicates patients daily clinical state following treatment. Black spaces indicate diurnal remission from psychotic symptoms. White spaces indicate no diurnal improvement.

Of unimproved cases there were 32 or 39 per cent who showed no response and 15 or 18 per cent who showed good responses and then relapsed before eight months had passed.

There were no deaths in the treatments and no patients seem to be worse mentally, physically or neurologically.

The best responses and those held longest are in cases with durations up to one year; these numbered 68 per cent of our cases.

Those cases treated towards the end of the first year of outspoken illness did as well as those treated earlier. Of cases two to 12 years in duration 32 per cent responded, most of whom relapsed. However, we have placed 10 patients in the doubtful list as to duration. Our experience makes us question the accuracy of the assigned duration of most cases. Usually there are different starting points for the beginnings of illness which depend on the testimony of untrained observers. In one case the family describes a

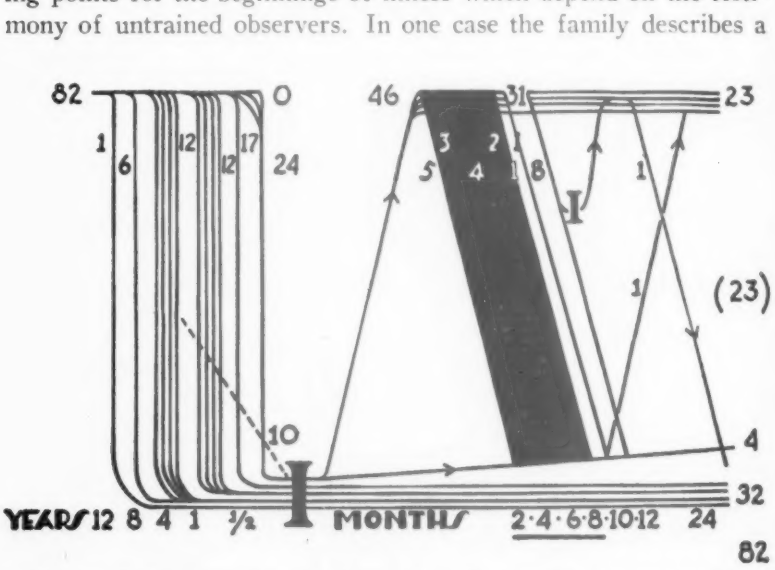


FIG. 2.—Follow-up results on 82 patients. Figures indicate patients. Top line shows number well. Bottom line represents those psychotic. Duration of illness shown in years in left portion of graph. Onset of illness represented by vertical lines. Insulin treatment indicated by letter I. Sloping lines show response to treatment. Relapses indicated by lines running from top towards bottom.

young man as "nervous" for six years, "very faddy" for three years, psychotic for two weeks. We have found it best to try to find the onset of glaring and unmistakable psychotic symptoms—the social turning point—and to measure duration from there.

Fig. 2 shows what happened in individual cases. The duration of illness is indicated in years, the response to treatment in months. The top line indicates the condition of the patients before the obvious beginning of the psychosis. Extremes in duration of illness were two weeks and 12 years. Twenty-four patients had a

duration of illness under Sakel's limit of six months; there were 19 with duration over four years.

Eighty-two patients fell from their baseline into psychosis. There they met insulin-shock treatment.* The results are shown in terms of getting back to that baseline. In 32 cases they did not climb from their low level, except in some cases for a few days. In four cases there was slight but maintained improvement. In 46 cases there was an immediate climb back to the normal baseline, or closely either side of it. But in eight months a total of 15 had fallen back into psychosis. As eight months is the only follow-up period to include all cases, our general statements stop here with 31 patients well or approximately 38 per cent of remissions.

As will be noted in the chart after the eight months' limit, 10 patients relapsed, most of them after a year's remission. Two patients in addition took a course toward recovery; one after a second course of insulin and one without shock treatment.

This of course indicates that follow-up of all cases for two years would show a remission rate which could not be higher than 28 per cent and might be considerably less.

Discussion.

As the diagnosis of dementia præcox varies from country to country and hospital to hospital, it is best to compare insulin-shock remissions with results before the time of shock treatments in the same hospital.

In 1927 a study of the prognosis in schizophrenia was published by Strecker and Willey:[2] the patients considered were 186 admitted to the Pennsylvania Hospital several years before. There was a percentage of 20 per cent "recovered."

In 1927 a study of the prognosis in mental disease was published by Bond and Braceland:[3] this was a five-year follow-up of all patients admitted to the Department for Mental Diseases of the Pennsylvania Hospital in 1927 and 1928. Patients with dementia præcox were sorted out of these, as were those with all

* In 19 cases, when in the last weeks of insulin treatment progress was not satisfactory, Metrazol was used as adjuvant in ways described by Bowman and Wortis, L. V. Meduna and Sakel. Metrazol was given on Wednesdays and Saturdays for two weeks while insulin was used on the other days.

other diagnoses. To keep the classification clear, many patients, 10 per cent of the total, were placed in an unclassed group. Dementia præcox cases made up 16 per cent of the total, diagnosis being made from the vantage point of five years after admission. Of 116 cases, 10 per cent had full remissions and 10 per cent improved. As was mentioned in the paper by Bond and Braceland the percentages of remissions could be increased to 40 per cent, if cases which suggested this diagnosis at admission were included. However, for the purpose of comparison with insulin-shock results, there is reason to use the 10 per cent plus 10 per cent figures for cases without chemical treatment because they are closest in time, in diagnostic customs, and in the care of the same senior physicians.

We have then to compare 10 to 20 per cent of old, with 38 per cent of shock remissions. (a) The older cases have lasted longer: the new cases having been followed for only eight months. (b) In the older remissions there is little to indicate what brought about improvement: in two cases hospital surroundings and supporting treatment resulted in gains of 20 and 46 pounds in weight and perhaps indicated emotional adjustments: in one case a patient was plunged with a devoted relative into the wilderness to live or die and responded splendidly. In the insulin-shock cases the remission has followed treatment immediately in all but two instances. (c) In the older remissions insight is not so prominent as it is in the insulin cases. (d) Of the older remissions there have been relapses after our regular follow-up period and the publication of the results. One patient relapsed 10 years after recovery with a very good record in between. If we watch relapses after shock treatments we must watch them in our control cases.

The extent to which valid comparisons may be made between our results and those from other hospitals is doubtful. The advantage of using our own cases as controls lies in the fact that we have a homogeneous group of cases judged by the same diagnostic criteria. Customs in the next hospital will vary somewhat: in other countries a great deal. We are certain that the diagnostic criteria at Burghölzi in Switzerland are such that many cases are classed as schizophrenia which would be labelled manic depressive in our own and in other American hospitals.

Action of Insulin.

Little is known of the action of insulin on the nervous system other than the fact that it reduces the oxygen utilization of the brain (Holmes) [4] (Wortis).[5]

The following experiments are concerned with studies of its action on the electrical activity of nerve and the relationship between these changes and the nerves excitability. This action of insulin has been studied by observing changes produced in the after potentials of the phrenic nerve of the cat by the injection of a convulsive dose of insulin into the animal. The nerves were obtained from cats who had been subjected to at least 10 days of treatment with convulsive doses of insulin. Each nerve was dissected out during a convulsive period and quickly mounted in a nerve chamber through which was passing gas mixture of 5 per cent CO_2 and 95 per cent O_2 according to the technique developed by Lehman.[6] The temperature of the nerve chamber was kept at 37° C. Ringer solution (pH 7.4) was used to keep the nerve moistened. The nerve was stimulated by a thyratron shock; the after potentials were observed on a cathode ray oscillograph.

The results of our experiments varied according to the severity of the insulin convulsions to which the animals were subjected. Under our conditions of recording the negative after potentials in our control nerves varied from 16 to 24 milliseconds. In the insulin experiments the nerves from those animals which showed increased reflex excitability during insulin coma, developed greatly prolonged negative after potentials ranging from 50 to 100 milliseconds in duration. Fig. 3 illustrates an instance of this. After 20 to 25 minutes in the nerve chamber this after potential began to decrease until it finally reached a normal value of 16 to 22 milliseconds after 30 to 40 minutes.

Since the excitability of a nerve parallels the course of its after potential (Gasser and Grundfest) [7] it follows that one of the effects of insulin in these experiments is to bring about a state of prolonged supernormal excitability as judged by the increased negative after potential. Clinically, neuro-muscular excitability curves mapped out on patients during insulin treatment support the view that insulin in large doses brings about a state of hyperexcitability. Fig. 4 shows such a curve and is typical of our findings. These

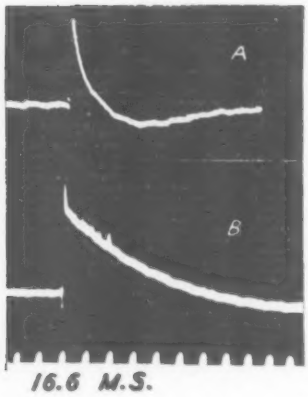


FIG. 3.—After potentials phrenic nerve of cat. *A*, Control from normal animal. *B*, From animal having convulsions resulting from insulin. Note increase in negative after potential.

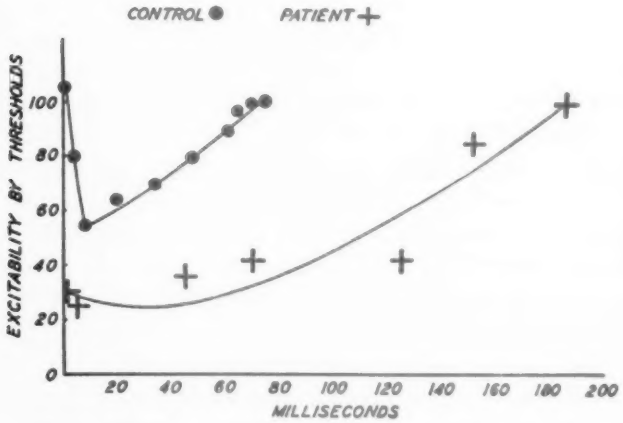


FIG. 4.—Neuro-muscular excitability curves on patients. Dots represent excitability on a normal control; crosses indicate excitability following insulin. Curves mapped by threshold method.

data were obtained by stimulating a flexor motor point on the forearm with a shock of threshold value then determining the percentage decrease in the strength of this shock when it was preceded by a conditioning shock at various intervals.

The mechanism of this insulin action is not known. Lehman [8] has shown that the type of after potential changes seen in these experiments can occur in normal nerves when there is a sudden shift in pH from a high to a lower value or on recovery from asphyxia.

It may be argued by making the assumption that similar changes in excitability take place centrally as observed in peripheral nerve, that the effect of insulin is to establish a state of increased excitability in the central nervous system. During this state of increased excitability nerve impulses could circulate which would ordinarily be blocked. To explain the clinical changes in patients on this basis implies a relationship between synaptic transmission, excitability properties and psychic processes.

Conclusions.

1. In every hospital using insulin-shock the patients treated should be followed for five years. Another group of patients, or other groups, as near alike as possible should be followed for the same time as controls. It seems certain that relapses after favorable responses will follow in all groups.

2. In the valuable paper just published by Malamud and Render [9] it is stated that a study of the course and prognosis in schizophrenia should include the criteria used by the author in diagnosis. We take a little exception to this statement because it might give encouragement to more varying personal standards for schizophrenia. After all, it is safer for psychiatrists to use the criteria put down in the official Statistical Manual,[10] and to work toward a definition which all can use.

3. Insulin-shock therapy is not a complete answer. But the transformation of the patient's condition is so immediate and favorable in a majority of cases that it will have place in, or provide suggestions for a future therapy, and this without regard to whether good results last for an afternoon or indefinitely. It is

only fair to remember that Sakel spoke of insulin-shock as the artillery in a general combined attack on the disease process.

4. Insulin in convulsive doses alters the metabolic processes of nerve.

BIBLIOGRAPHY.

1. Frostig, J. P.: Arch. Neurol. Psychiat., 39: 219, 1938.
2. Strecker, E. A., and Willey, G. F.: J. Ment. Sc., 73: 939, 1927.
3. Bond, E. D., and Braceland, F. J.: Am. J. Psychiat., 94: 263, 1937.
4. Holmes, E. G.: Biochem. J., 24: 914, 1930.
5. Wortis, S. B.: Arch. Neurol. and Psychiat., 33: 1022, 1935.
6. Lehman, J. E.: Am. J. Physiol., 118: 600, 1937.
7. Gasser, H. S., and Grundfest, H.: Am. J. Physiol., 117: 113, 1936.
8. Lehman, J. E.: Am. J. Physiol., 119: 111, 1937.
9. Malamud, W., and Render, Norman: Am. J. Psychiat., 95: 1040, 1939.
10. Statistical Manual National Com. for Mental Hygiene, N. Y., 39: 1934.

REGARDING SODIUM AMYTAL AS A PROGNOSTIC AID IN INSULIN AND METRAZOL SHOCK THERAPY OF MENTAL PATIENTS. (DEMENTIA PRÆCOX.)*

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In some earlier studies [1] it was found that the intravenous injection of sodium amytal in prenarcotic doses produces transient ameliorating effects in some psychotic patients. Since such an ameliorating response indicated that the disturbed functions of the brain of a patient with a psychosis could be favorably influenced by a chemical agent, it was decided to compare this reaction to the intravenous injection of sodium amytal with the subsequent therapeutic effect of insulin hypoglycemic shock and metrazol convulsant therapy.

Procedure.

A freshly prepared 10 per cent solution of sodium amytal was injected intravenously at the rate of approximately one cc. per minute for the first cc. and the patient was then questioned in order to determine whether any change in the mental state had occurred. If no change was manifest another cc. was slowly injected and the interview was continued. Patients who showed any amelioration usually did so after from one to three cc. In some cases which showed no amelioration with one to three cc. as much as five cc. was injected slowly over a period of about twenty minutes to one half hour while the patient was being interviewed. Only very occasionally did this prove effective where the smaller dose had failed.

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The amelioration was manifested by an increased accessibility or rapport and improvement in the mental state and behavior of the patient. This test was carried out before insulin therapy was instituted. The condition of the patient within one to two months after the completion of a course of treatment was used in evaluating the result of insulin hypoglycemic therapy for this study. The question of the permanency of the therapeutic effects in relation to the test will be analyzed at a later date after a larger number of cases have been collected.

Results.

In Tables I and II are given the data as to age, diagnosis, duration of illness, response to test and therapeutic results for patients who received insulin hypoglycemic shock therapy.

In Table III the data have been analyzed in two groups, namely, those in whom the injection of sodium amytal produced an ameliorating effect and those in whom no such effect occurred. The groups are further subdivided as to duration of illness and the therapeutic results obtained with insulin hypoglycemic shock treatment. The degree of response either in the test or therapeutic effect is not taken up in this table but is given in Tables I and II.

In Table IV are listed the cases which failed to respond to insulin shock therapy and who were subsequently treated with metrazol. The result of the sodium amytal test (performed before insulin treatment) and metrazol therapy is compared.

Discussion.

It can be seen from Table III that 77 per cent of the patients who responded favorably to sodium amytal responded favorably to insulin hypoglycemic therapy and 64 per cent of the patients who failed to respond to sodium amytal also responded unfavorably to insulin therapy.

All the 10 insulin failures treated with metrazol convulsant therapy failed to show any improvement with the latter treatment. Of these cases four had shown some amelioration from the injection of sodium amytal. It is of interest that of these four cases three had originally responded to insulin therapy and had subsequently relapsed.

TABLE I.

FEMALE PATIENTS.

Case number.	Patient.	Age.	Diagnosis (type).	Duration of illness.	Response to sodium amytal.	Result of Hypoglycemic therapy.	Remarks.
1	M.E.	35	Catatonic.	2 yrs.	−	−	Recovered several months after treatment.
2	D.M.	21	"	10 mos.	++	++	Relapsed after second course of therapy, did not improve.
3	B.K.	23	Hebephrenic.	6 yrs.	−	+	Relapsed and did not respond to second course of therapy.
4	D.F.	17	"	1½ yrs.	+	−	
5	B.H.	31	Paranoid	2 yrs.	−	−	
6	C.B.	36	"	9 mos.	−	−	
7	B.S.	32	"	1½ yrs.	++	++	
8	S.S.	31	"	7 mos.	+	++	
9	F.S.	19	Catatonic	3 mos.	++	++	Relapsed and recovered after second course of treatment.
10	N.S.	28	Hebephrenic.	4½ mos.	++	−	Improved in early stage of treatment, then relapsed.
11	J.Q.	33	Paranoid.	8 mos.	±	++	Three relapses, did not respond after third course of treatment.
12	M.N.	32	Catatonic.	1½ yrs.	−	−	
13	S.L.	22	"	6 mos.	+	+	Recovered after going home.
14	S.H.	35	"	8 mos.	++	++	Relapsed, improved at second course, relapsed again and improved after third.
15	G.C.	17	"	2 mos.	+	++	Relapsed and did not respond to second course of treatment.
16	S.C.	20	Catatonic	2 mos.	+	+	Relapsed and did not respond to second course of therapy.
17	V.C.	24	"	5 mos.	+	++	Relapsed after 1½ years.
18	G.Z.	27	"	3 mos.	++	++	
19	F.S.	41	Hebephrenic.	4 yrs.	+	+	Relapsed, improved after treatment.
20	M.Z.	25	"	2 yrs.	−	−	
21	R.W.	25	"	14 mos.	+	+	
22	D.C.	25	Catatonic.	10 mos.	+	−	
23	L.G.	27	Paranoid.	16 mos.	−	−	
24	M.M.	24	"	3 yrs. 8 mos.	−	+	Relapsed.
25	M.G.	16	Hebephrenic.	2 yrs.	+	++	Relapsed and improved with insulin and metrazol, final relapse without improvement.
26	B.K.	19	"	9 mos.	+	++	
27	C.G.	27	Catatonic ?	7 mos.	++	++	
28	J.H.	22	Not clear.	1 mo.	++	++	
29	K.F.	29	Hebephrenic.	1 yr.	++	++	

TABLE II.

MALE PATIENTS.

Case number.	Patient.	Age.	Diagnosis (type).	Duration of illness.	Response to sodium amytal.	Result of Hypoglycemic therapy.	Remarks.
1	E.W.	22	Paranoid.	10 mos.	−	+	
2	H.C.	19	Hebephrenic.	1 yr.	−	++	Relapsed, some improvement after second course, relapsed, returned to hospital.
3	D.S.	33	Paranoid.	5 mos.	−	−	
4	S.G.	39	"	5 mos.	±	++	(Patient had a prolonged coma.)
5	S.W.	16	Hebephrenic.	10 mos.	−	−	
6	G.S.	16	"	3 yrs. 9 mos.	−	−	
7	A.C.	20	Catatonic.	5 mos.	−	+	Finally recovered.
8	H.C.	28	Hebephrenic.	2½ yrs.	+	+	
9	H.W.	26	Simple.	2 yrs. 10 mos.	−	−	Improved one-half year later at time of discharge.
10	J.P.	16	Catatonic.	7 mos.	−	−	
11	D.T.	24	"	2 yrs.	−	−	Discharged unimproved, improved later at home.
12	B.W.	17	"	2 mos.	−	−	Improved several months after transfer to other hospital.
13	B.S.	27	Paranoid.	3 mos.	+	−	Improved under metrazol.
14	E.P.	18	Hebephrenic.	1 yr. 11 mos.	−	−	Relapsed.
15	R.B.	26	Paranoid.	2 yrs. 9 mos.	±	−	
16	J.F.	32	"	4 mos.	+	+	
17	C.R.	16	Hebephrenic.	9 mos.	−	−	
18	S.M.	23	Catatonic.	2 yrs. 1 mo.	−	−	
19	J.B.	15	Hebephrenic.	11 mos.	−	+	
20	H.G.	17	Catatonic.	6 mos.	+	+	
21	A.R.	30	Hebephrenic.	1 yr. 10 mos.	−	+	
22	M.S.	16	Catatonic.	5 mos.	±	−	
23	M.D.	26	Paranoid.	5 mos.	−	−	
24	T.O.	22	Hebephrenic.	2 mos.	+	+	
25	M.H.	18	"	1 yr. 8 mos.	±	+	
26	L.S.	28	Catatonic.	6 mos.	±	++	

RESPONSE TO SODIUM AMYTAL.

(−) = no response.
(±) and (+) = response as indicated by greater accessibility.
(++) = increased accessibility, improved mental state and at times awareness of delusional ideas.

RESULT OF HYPOGLYCEMIC THERAPY.

(−) = failure.
(+) = improved or much improved.
(++) = recovery.

In a recent publication Lemere [2] has suggested that the various agents used in the treatment of schizophrenia tend to strengthen the alpha rhythm of the electroencephalogram which he thinks has

TABLE III.

Duration of illness.	Group with positive response to sodium amytal.		Group with negative response to sodium amytal.	
	Positive therapeutic result. No. of cases.	Negative therapeutic result. No. of cases.	Negative therapeutic result. No. of cases.	Positive therapeutic result. No. of cases.
Up to 6 months....	8	3	3	2
6 months to 1 year..	9	2	5	2
1 year to 2 years...	3	2	6	3
Over 2 years.....	3	..	2	2
Total.....	23 (77%)	7 (23%)	16 (64%)	9 (36%)
Total for group.....	30		25	

TABLE IV.

INSULIN FAILURES TREATED WITH METRAZOL CONVULSANT THERAPY.

Case number.	Patient (female).	Sodium amytal test.	Number of Metrazol treatments.	Number of convulsions.	Therapeutic results.	Remarks.
10	N.S.	++	15	9	−	
5	B.H.	−	25	14	−	
21	R.L.	−	36	25	−	
22	D.C.	+	46	22	−	
3	B.K.	−	22	17	−	Improved and relapsed.
15	G.C.	−	21	19	−	
23	L.G.	−	21	15	−	
11	J.Q.	±	11	6	−	See table I, insulin relapse.
25	M.G.	+	11	5	−	Insulin relapse, see table I.
19	F.S.	+	18	7	−	Insulin relapse.

a tendency to be innately weak in schizophrenic patients and that all of these agents produce their effect through a similar mechanism. It is of interest, in this connection, that about 36 per cent of the cases who failed to respond to prenarcotic doses of sodium amytal subsequently improved under insulin hypoglycemic shock ther-

apy and 23 per cent of those who responded to the injection of sodium amytal failed to respond to insulin hypoglycemic treatment (see Table III). It is somewhat difficult to reconcile these cases with Lemere's hypothesis. It is intended to subject this phase of the work to further investigation with the use of electroencephalographic observations. The findings are of considerable interest from the theoretical standpoint and indicate the need for further studies of the mechanisms involved. However, from the prognostic standpoint it would appear that cases that respond favorably to the injection of sodium amytal in prenarcotic doses have a very good chance of responding favorably to insulin hypoglycemic treatment. In cases that do not respond to sodium amytal the chances for improvement are considerably reduced. This test which is comparatively simple to carry out appears to be of value as a prognostic aid in insulin shock therapy. The uncooperative state of the patient is no deterrent to carrying out this test since amelioration of such state by the injection of the sodium amytal is part of the test. This should be of advantage in cases where psychological tests [3, 4] which also have been found to be of prognostic value, cannot be carried out because of the uncooperative state of the patient. A combination of the various tests may prove of considerable aid in prognosis as to insulin shock therapy and perhaps also as to spontaneous recovery under the usual psychiatric treatment. The sodium amytal test is being carried out in the other psychoses at the present time. The data regarding the patients treated with metrazol while suggestive are too few for analysis.

Summary and Conclusions.

1. A total of 55 cases of dementia præcox were tested by the intravenous injection of prenarcotic doses of sodium amytal prior to treatment with insulin hypoglycemic therapy.

2. An ameliorating response to the injection of sodium amytal was obtained in 30 patients and of these 23 or 77 per cent had a favorable therapeutic response to insulin hypoglycemic treatment.

3. No ameliorating response to sodium amytal was obtained in 25 cases and of these 16 or 64 per cent failed to respond to insulin treatment.

4. The results of metrazol convulsant therapy in cases that failed to respond to insulin treatments are also compared with the sodium amytal test.

5. The response to the sodium amytal test appears to be of prognostic value in insulin hypoglycemic shock therapy.

6. Plans for further studies are indicated.

BIBLIOGRAPHY.

1. Harris, Meyer M., and Katz, S. E.: The effect of the administration of sodium amytal and sodium rhodanate on mental patients. Am. J. Psychiat., 12: 1065, 1932.

2. Lemere, F.: Effects on the electroencephalogram of various agents used in treating schizophrenia. J. Neurophysiol., 1: 590, 1938.

———: Electroencephalography. Psychiat. Quart., 13: 5, 1939.

3. Piotrowski, Z.: The prognostic possibilities of the Rorschach method in insulin treatment. Psychiat. Quart., 12: 679, 1938.

4. Bolles, M. M., Rosen, G. P., and Landis, C.: Psychological performance tests as prognostic agents for the efficacy of insulin therapy in schizophrenia. Psychiat. Quart., 12: 733, 1938.

IRREVERSIBLE OR HYPERGLYCEMIC INSULIN COMA.

Its Cause and Its Response to Blood Transfusion.*

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Hypoglycemic insulin coma, as the term implies, is associated with a low blood sugar, usually varying from 40 mg. per cent down to zero. It sometimes happens, however, that patients undergoing insulin shock treatment remain comatose even after a glucose tube feeding and even after enough glucose has been given by vein to raise the blood sugar to abnormally high levels. When this occurs, we speak of irreversible or protracted coma—a relatively common and dangerous complication of insulin shock treatment. In cases with a favorable outcome the coma may persist for hours, days or weeks, with a clinical picture which gradually merges into that of a catatonic stupor before the awakening. A status epilepticus may supervene, vomiting is common, fever, rapid pulse and respiration invariably develop, and the patients may finally die through respiratory failure or through a complicating pneumonia. Autopsy findings in such cases have usually revealed nothing to clearly indicate the precipitating cause of this coma or death. A summary of some recent case reports is contained in Table I.

In trying to find a cause for the phenomenon of irreversible coma, it is natural to suppose at first that these patients have simply been left in coma too long, and it is in fact easy to demonstrate, as Sherrill and MacKay [13] have done, that animals left in coma too long will no longer revive with glucose but will die. In patients undergoing shock treatment, however, irreversible coma was found to occur not infrequently in cases where a tube feeding was given an hour or less after the onset of coma, which is within

* Read at the Fiftieth Anniversary Celebration of The Johns Hopkins Medical School, May 5, 1939. From The Johns Hopkins Medical School, Baltimore, Maryland, and the Bellevue Psychiatric Hospital, New York.

TABLE I.

SUMMARY OF SOME RECENT REPORTS OF IRREVERSIBLE OR PROTRACTED COMA.

Authors.	Case.	Insulin dose.	Duration of coma till tube feeding.	Duration of coma till I. V. glucose.	Treatment.	Maximum blood sugar.	Maximum temperature.	Maximum pulse rate.	Maximum respiration.	Duration of coma.	Duration of confusion or stupor.	Psychiatric outcome.	Remarks.
Easton[1]	..	...	2 hours.	3 hours.	I. V. glucose, cardiac stimulants.	235 mg.	103.8°	140	34	49 hours.	7 days.	Marked improvement.	.....
Molony and Honan[2]	..	...	2 hours.	4 hours.	Insulin, saline.	360	102.2°	...	...	7 days.	7 days.	.....	2 percent glycosuria. Spinal fluid sugar 360 mg. percent. Gastric retention.
McKendree[3]	..	...	1½ hours.	1½ hours.	....	..	105.8°	158	60	4 days.	3 weeks.	.....	Spinal fluid sugar 108 mg. percent. Pulmonary edema.
Horwitz, Blalock and Harris[4]	I	50 units	1 hour.	....	....	..	102°	120	...	24 hours.	2 days ?	No improvement.	Adrenalin provoked seizure.
	II	75	1½ hours.	1½ hours.	....	295	106.6°	Rapid	Rapid	5 days.	2 months.	Recovery.	Adrenalin provoked seizure. Glycosuria.
	III	125	....	....	Betalin S, 1 mg.	..	101.8°	...	...	11 hours.	24 hours ?	Unchanged.	.....
Binzley and Anderson[5]	Case 14	90	3¾ hours.	4¼ hours.	I. V. glucose	196	102°	...	...	12 hours.	....	Recovered.	.....
	32	90	1¾ hours.	2½ hours.	I. V. glucose	290	103.2°	250	...	5 days.	5 weeks.	Recovered.	Glycosuria. Spinal fluid sugar 105 mg. percent.
	44	90	2¼ hours.	2¾ hours.	I. V. glucose	165	102°	160	Very rapid	10 hours.	....	Marked improvement.	.....
	50	65	1½ hours.	2 hours.	I. V. glucose. Coramine.	..	...	170	52	1 week.	1 week.	Died of bronchopneumonia.	.....
Lester[6]	..	240	1¾ hours.	1¾ hours.	I. V. glucose. Digitalis. Paraldehyde.	..	103.8°	Rapid	...	72 hours.	5 days.	Marked improvement.	Gastric retention.

Lester[6]	..	240	1 ¼ hours.	1 ¼ hours.	I. V. glucose. Digitalis. Paraldehyde.	..	103.8°	Rapid	...	72 hours.	5 days.	Marked improvement.	Gastric retention.

Finiefs[7]	Case 1	115	1 hour.	1 ½ hours.	....	..	...	...	...	10 hours.	....	.....	.....
	2	75	2 hours.	2 ¼ hours.	I. V. glucose. Morphine. Atropine. Lobeline. Coramine.	..	102.4°	120	...	14 hours.	....	.....	Gastric retention. Pulmonary edema.
James, Freudenberg and Cannon[8]	..	130	2 hours, 20 min.	2 hours, 50 min.	I. V. glucose. $CaCl_2$. Vitamin B_1 Spinal tap.	235	101°	120	...	5 hours.	5 days.	Recovered.	Spinal fluid sugar 300 mg. percent.
Lazell[9]	..	110	1 hour.	1 ½ hours.	I. V. glucose. Adrenalin. Spinal tap. Na luminal. Ca gluconate. Insulin.	166	101.6°	140	40	20 hours.	10 days (cerebellar fits).	Much improved.	.....
Freed and Wofford[10]	R. B.	75	2 hours.	2 hours, 40 min.	I. V. glucose. Atropine. Pheno-barbital. $CaCl_2$.	380	104°	150	60	24 hours ?	3 weeks.	Recovered.	Blood in spinal fluid.
Kastein[11]	..	140	1 ½ hours.	13 hours.	....	128	101°	140	...	20 hours.	8 days.	.....	.....
Timmer[12]	..	145	1 ½ hours.	No I. V. glucose.	Adrenalin. Digitalin. Caffeine. O_2	..	...	...	...	4 hours.	....	Died.	.....
Wortis and Lambert	M. L.	135	1 hour, 50 min.	3 hours.	I. V. glucose. Vitamin B_1. Adrenalin. Na amytal. Spinal tap. O_2. Blood transfusion.	280	105°	152	34	20 hours.	....	Recovered.	.....
	A. C.	130	1 hour, 40 min.	2 hours.	I. V. glucose. Blood transfusion.	514	101.8°	160	40	10 hours.	....	Improved.	.....
	J. J.	90	2 hours.	2 ½ hours.	I. V. glucose. Vitamin B_1. Blood transfusion.	110	101.8°	104	22	36 hours.	....	Recovered.	.....

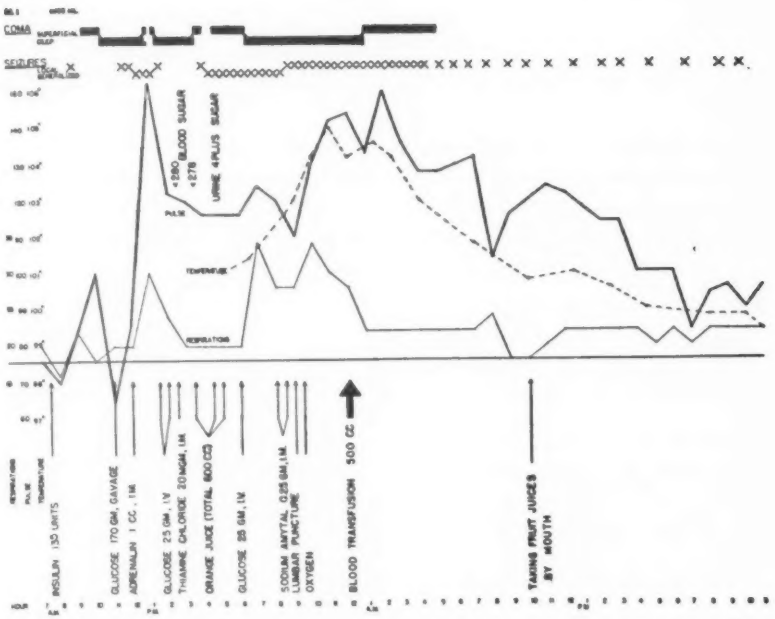
the usual permissible limit for prompt reversibility. One waited, the patient did not rouse, intravenous glucose was given a half hour or an hour later, the patient still did not rouse, and one was again confronted with the difficult problem of an irreversible coma.

The phenomenon seems to be related to another occasional troublesome consequence of tube feeding: slow awakening. Three years ago one of us [14] called attention to the cases in which the blood sugar remained low and the patient remained comatose after tube feeding until intravenous glucose was given, though it did not occur to us at that time that the patients might simply have had gastric retention. In a case of irreversible coma observed at Bellevue Hospital and reported in 1937,[15] it was found that the patient retained and vomited an entire tube feeding hours after it was given, though we again did not appreciate the full significance of the phenomenon of gastric retention.

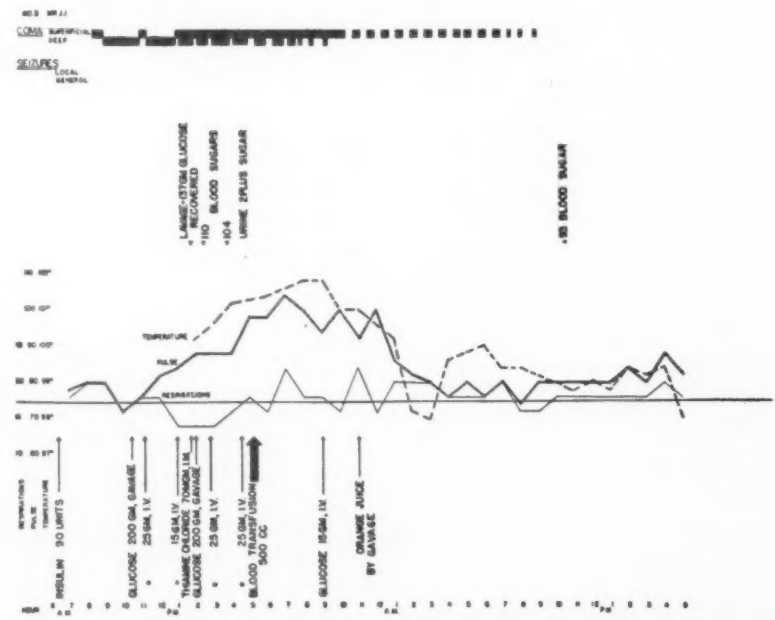
We have since confirmed the occurrence of gastric retention in a series of cases of protracted or irreversible coma, as well as in cases with slow awakening after tube feeding. In these cases the glucose solution passed by tube feeding was completely or almost completely retained in the stomach and could be recovered a half hour or sometimes even several hours later by again passing the tube and aspirating the stomach contents. There was usually some admixture of gastric juice but a determination of the sugar content in a series of seven cases invariably showed that little or no glucose had been absorbed or utilized. Intravenous glucose in such cases is thus often given too long after the actual onset of coma, when the organism is apparently no longer able to burn or utilize the glucose available. Hyperglycemia and glycosuria ensue and the further prolongation of coma, unless checked in time, leads to irreparable brain damage, status epilepticus and death.

Though various measures have been proposed and practiced, there has so far been no understanding of the basic physiology involved in the phenomenon of irreversible coma and no specifically effective measures found to combat it. Recovery in cases where this has occurred seems more likely to follow supportive and conservative measures than any specific remedy thus far proposed.

We have had occasion in three recent cases (see accompanying curves) not only to confirm this mechanism in the production of



CASE 1.—Miss M. L. Clinical improvement in this case was immediate after blood transfusion. See accompanying case report.

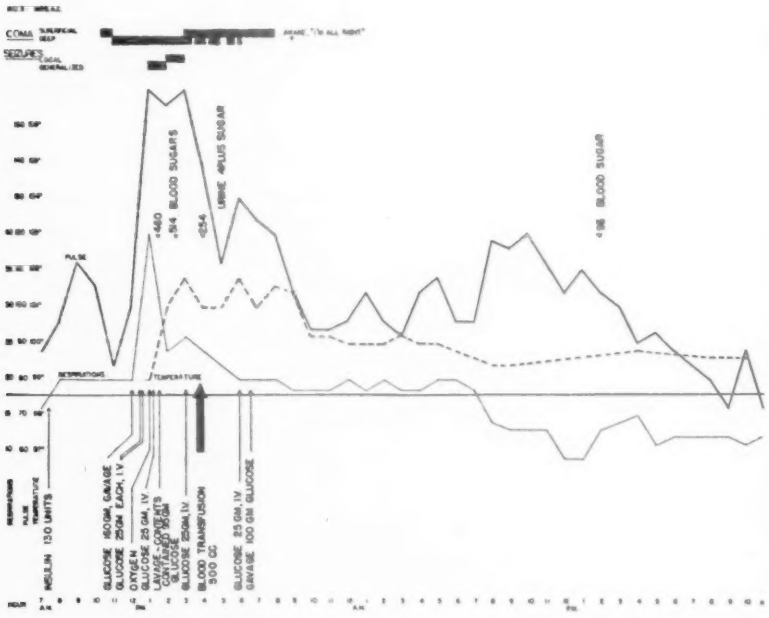


CASE 2.—Mr. J. J. A schizophrenic youth, whose illness developed about a year previously. The patient's condition during this protracted coma was never alarming, and he would probably have roused spontaneously in time, though transfusion appears to have facilitated recovery. Treatment was resumed in this case, and he was subsequently discharged as psychiatrically recovered.

irreversible coma, but have also been able to treat the condition with apparent success by blood transfusion, in accordance with the independent cursory suggestions of Kubo [16] in Japan and of James, Freudenberg and Cannon [8] in England. An illustrative case report follows:

An Illustrative Case.

Miss M. L., a 38-year-old school teacher, was admitted to the Phipps Clinic August 14, 1938, with a history of a gradually developing psychosis



Case 3.—Mrs. A. C. Improvement followed transfusion in this case, but the patient began to lapse into coma and a Babinski reappeared soon afterwards, until additional intravenous glucose was administered. There was no retention of the second tube feeding. The patient showed psychiatric improvement following this complication but treatment was not resumed. Slight weakness of the left side persisted for several days.

dating back about three years, characterized mainly by shyness, apprehension and frequent crying spells culminating in hallucinatory experiences, feelings of reference and of unreality, difficulties in concentration and occasional violence in the weeks preceding admission. After a period of four months in which all ordinary psychiatric procedures proved unavailing, insulin treatment was started December 6, 1938, and continued with gradu-

ally increasing doses until January 3, 1939, when she had already had 24 insulin treatments and 16 comatose reactions. Her course on January 3 and thereafter is outlined below:

Jan. 3,	7.30 A. M.	Patient is given 135 units of insulin.
	9.30	Patient is sleeping soundly, perspiring profusely.
	10.00	Patient does not respond to ordinary stimuli, is in deep coma.
	11.00	425 cc. of 40 per cent glucose solution is given by stomach tube.
	11.30	Patient does not respond.
	12.00 M.	Patient is responding. A few minutes later she says, "I don't feel so good. I'm cold." She ate lunch when spoon fed.
	12.20 P. M.	There are generalized twitching movements lasting 5-15 seconds, coming every 2-3 minutes.
	12.45	There is a severe, generalized convulsion lasting 30 seconds, accompanied by cyanosis and loss of consciousness, followed by slighter convulsions every few minutes.
	1.00	Another severe convulsion occurs. Patient is given 1 cc. of adrenalin intramuscularly, but does not regain consciousness.
	1.30	50 cc. of 50 per cent glucose solution is given intravenously.
	1.50	Patient is still in coma and having repeated convulsions. Intravenous glucose is repeated.
	2.05	The patient's condition is unchanged; 10 mgm. of thiamin chloride is given intramuscularly.
	2.30	Patient remains comatose. Generalized convulsions of tonic and clonic character are occurring every 10-20 minutes and lasting 1-2 minutes. Blood sugar is 280 mgm. per cent.
	3.00	Patient responds to questioning. Convulsions have ceased.
	3.30	Blood sugar is 278 mgm. per cent. Patient is taking orange juice by mouth but is vomiting most of it.
	4.20	There are twitchings of the extremities every few seconds.
	5.00	The twitchings continue, occasionally becoming generalized. Patient is again temporarily conscious. Rectal temperature is 101°.
	5.42	There is a severe generalized convulsion lasting two minutes. Patient is cyanotic and unconscious during the seizure.

	5.55	50 cc. of 50 per cent glucose solution is given intravenously.
	6.30	Glucose is repeated. There is a clonic seizure involving only the right arm and leg, lasting 45 seconds. Patient did not lose consciousness.
	7.00	Tonic and clonic seizures occur every 7-12 minutes, last 1-2 minutes, involve mainly the right side. Patient is in coma again.
	8.00	The seizures continue as above. Patient is still in coma. 0.25 gm. of sodium amytal given intramuscularly.
	8.15	Sodium amytal is repeated.
	8.30	The patient continues as above. Rectal temperature is 102.6°.
	9.15	The seizures continue. Lumbar puncture—10 cc. of clear fluid under normal pressure are removed. Patient is still in coma.
	9.20	Oxygen administration is started.
	9.30	There is another seizure, mainly on the right.
	10.00	The patient is quiet, still in coma. She moves left extremities but not the right.
	11.00	Patient is still in coma. Rectal temperature is 105°.
	12.00 M.	Blood transfusion of 500 cc. citrated blood is given.
Jan. 4,	1.15 A. M.	The patient is unresponsive. The pulse is of better quality and the color is better. (See chart.)
	1.30	The patient's general condition seems improved. There are occasional twitchings of the right leg and she is restless.
	3.00	There are occasional twitchings of the right leg.
	4.00	The patient appears to be conscious.
	7.00	The patient is fully conscious. She answers question with "yes" or "no," but does not talk otherwise. There are periodic twitchings of the right leg, marked weakness of the right arm and leg. There is no facial weakness.
	8.45	Blood sugar is 135 mgm. per cent.
	9.30	The patient is taking nourishment by mouth.
	5.00 P. M.	The twitchings of the right leg continue at frequent intervals. The patient still does not speak.
Jan. 5,	6.00 A. M.	The patient is talking coherently and relevantly. "What hospital is this?" There are still occasional twitchings and some weakness of the right leg. The right arm also is weak.
	7.00 P. M.	As above.
Jan. 6,	7.00 A. M.	The twitchings are much less frequent.
	7.00 P. M.	There is no further twitching.

Jan. 8, The weakness of the right extremities has disappeared. The patient is sitting up, looks fine. She says, "I'm glad I got the treatments because I'm better." She is in good spirits, carries on a normal conversation, and shows marked psychiatric improvement.

The patient received no further insulin treatment, was soon transferred to a quieter ward, and was discharged as recovered February 28, 1939. At the time of the present writing (May, 1939) she is still well.

Discussion.

It is possible that the improvement following the transfusion in our three cases is a coincidence, but the response in the severest of the cases (M. L.) seemed too marked to be merely accidental. Coma had persisted 14 hours in this case, and the patient's condition seemed threatening, but the clinical improvement following the transfusion was immediate. In the least severe of the cases (J. J.) the effect was sluggish; the patient was already beginning to rouse before the transfusion was given and his condition never appeared threatening. His response is nevertheless interesting, and an exacerbation or prolongation of his coma might have occurred if a transfusion had not been given. It is noteworthy that the vegetative functions—pulse, temperature, and respiration—generally responded before consciousness and the higher integrative functions were restored. Clinical impression, analysis of the curves, and reports of the value of transfusion from two other independent sources all tend to support the probability that blood transfusion is a valuable measure in the treatment of dangerously protracted coma.

Though we shall not undertake a discussion of the physiology underlying this condition, it may be suggested that certain manifestations of irreversible coma, such as the fever, may be attributable not merely to a central disturbance of heat regulation (for in therapeutic coma the temperature drops rather than rises) but also to an increased combustion of proteins in the presence of impaired carbohydrate utilization. It is interesting to note in this connection that Gjessing's [17] recent work has indicated a relationship between increased protein combustion and remission in schizophrenia.

Summary and Conclusions.

1. Irreversible or hyperglycemic insulin presents a typical picture consisting of a sustained high blood sugar (following intravenous glucose administration), fever, rapid pulse and respiration and frequently fits, vomiting or diarrhea. It is a serious complication occurring not only in shock treatment but no doubt in the management of diabetes as well. It may result in death or in permanent brain damage, but recovered psychiatric cases often show dramatic improvement in their mental state.

2. Hyperglycemic coma is probably due to over-prolongation of therapeutic coma beyond an hour or an hour and a half.

3. Over-prolongation of coma is often unintentionally allowed, because the presence of gastric retention makes termination by tube feeding ineffective.

4. As a practical consequence coma should generally not be allowed to last more than an hour or an hour and a half, especially in cases that tend to rouse slowly, and sugar should be given by vein in all cases that do not begin to rouse 15 or 20 minutes after tube feeding.

5. Treatment of irreversible coma by intravenous glucose and blood transfusion of 500 cc. of citrated blood appeared effective in three successive cases.

6. It would appear that the whole blood contains substances, other than oxygen or sugar, essential for brain metabolism, which have been destroyed or depleted by protracted coma.

7. The dramatic psychiatric improvement in certain cases following prolonged coma suggests that if a safe method for prolongation of coma could be devised, it would further extend the value of shock treatment.

BIBLIOGRAPHY.

1. Easton, N. L.: A case of protracted hypoglycemia. Am. J. Psychiat., 94: 853, 1938.
2. Molony, C. B., and Honan, M. S.: An unusual complication of insulin shock (hypoglycemic) therapy. J. Ment. Sci., 83: 630, 1937.
3. McKendree, O. J.: Insulin therapy and its complications in the treatment of the psychoses. Psychiat. Quart., 12: 444, 1938.
4. Horwitz, W. A., Blalock, J. R., and Harris, M. M.: Protracted comas occurring during insulin hypoglycemic therapy. Psychiat. Quart., 12: 466, 1938.

5. Binzley, R. F., and Anderson, J. C.: Prolonged coma in the insulin treatment of dementia præcox. Psychiat. Quart., 12: 477, 1938.
6. Lester, D.: A study of prolonged coma following insulin shock. Am. J. Psychiat., 95: 1083, 1939.
7. Finiefs, L. A.: Dangers and emergencies of insulin therapy of the psychoses. J. Ment. Sci., 84: 678, 1938.
8. James, G. W. B., R. Freudenberg, and Cannon, A. T.: Insulin shock treatment of schizophrenia. Proc. Roy. Soc. Med., 31: 578, 1938.
9. Lazell, E. W.: Status epilepticus of the cerebellar type occurring in insulin shock treatment of schizophrenia, with recovery. M. Bull. Vet. Admin., 14: 365, 1938.
10. Freed, H., and Wofford, C. W.: Subarachnoid hemorrhage during shock therapy for schizophrenia. Arch. Neurol. and Psychiat., 39: 813, 1938.
11. Kastein, G. W.: De insuline- en corvis-(cardiazol) Behandeling bij schizophrenie, I Het Verlengde Coma Bij De Insuline Kuur, Pathogenese en Therapie. Nederl. Tijdschr. v. Geneesk., 82: 991, 1938.
12. Timmer, A. P.: Een sterfgeval tengevolge van een behandeling met den insulineshock volgens Sakel. Nederl. Tijdschr. v. Geneesk, 82: 3088, 1938.
13. Sherrill, J. W., and MacKay, E. M.: Deleterious effects of insulin shock. Proc. Soc. Exper. Biol. and Med., 36: 515, 1937.
14. Wortis, J.: Experiences at Bellevue with the hypoglycemic treatment of schizophrenia. Jour. Nerv. and Ment. Dis., 85: 565, 1937.
15. Wortis, J., Bowman, K. M., Orenstein, L. L., and Rosenbaum, I. J.: Further experiences at Bellevue Hospital with the hypoglycemic insulin treatment of schizophrenia. Am. J. Psychiat., 94: 153, 1937.
16. Kubo, K.: Ergebnisse der Hypoglykämie-Shock-Behandlung der Schizophrenie. Schweiz. Arch. f. Neurol. u. Psychiat. (Ergänzh.), 39: 194, 1937.
17. Gjessing, R.: Beiträge zur Kenntnis der Pathophysiologie periodisch katatoner Zustände, IV Mitteilung. Versuch einer Ausgleichung der Funktionsstörungen. Arch. f. Psychiatrie, 109: 525, 1939.

EVALUATION OF THERAPEUTIC FACTORS IN PHARMACOLOGIC SHOCK.*

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INTRODUCTION.

Pharmacologic shock treatment with insulin and metrazol has won wide acceptance; however, it remains an empirical method. Despite intensive research no convincing evidence has been offered to explain its beneficial effects because of the physiological changes produced. Psychological factors have been suggested as important but adequately controlled studies along this line have been few. Therefore, we have organized our projects in such a way as to resolve certain doubts and answer certain questions, among which are these:

1. Is the physiological action of insulin a necessary requisite of the treatment?

2. Does this physiological action have to be carried to the point of producing coma in order to be therapeutically active?

3. Of what therapeutic potentiality is the treatment situation considered from the point of view of

   a. Increased attention to the patient on the part of attendant, nurse and physician,

   b. Closer attention to general physical care,

   c. More adequate dietary control because of the danger of after shock,

   d. Emotional attitudes toward the treatment on the part of the patient,

   e. Emotional attitudes of the attendant, nurse and particularly the physician, especially when such an attitude is one of optimism and enthusiasm engendered by the novelty of the procedure?

4. Will the duplication of the physical and psychological setting without the use of either insulin or metrazol produce similar results?

5. Are the psychological factors inherent in the therapeutic situation of importance, and if removed, insofar as possible, will the therapeutic results be altered?

6. Are these treatments a specific therapy directed against the schizophrenic process or are they nonspecific in their action and therefore likely to be of benefit to other groups of psychoses and even psychoneuroses?

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* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Ill., May 8-12, 1939.

METHOD.

In our work with insulin, therefore, we have treated our patients in groups of six, and, by a process of eliminating one factor in each group, we have attempted, albeit somewhat crudely, to determine what the importance of that factor might be. Thus, our first group of six patients received a course of insulin hypoglycemia carried to the point of deep coma combined with intensive resocialization activities.

A second group received insulin hypoglycemia, which for a period of time was not permitted to lapse into coma, and this same group later received sufficient insulin to produce coma, but in neither period received other than the usual ward attention except during the hypoglycemic interval. To this group may be added seven male schizophrenics treated by one of us (R. L.) in the W. J. Seymour Hospital. These patients also received sufficient insulin to produce deep and prolonged comas but otherwise received no unusual attentions.

A third group received what was supposed to be insulin, but actually this was physiological saline solution. For this group neither the physical setting of the insulin ward nor the clinical routine was altered. For the information of the ward personnel it was stated that a different type of insulin (protamine zinc insulin) was being used and that while the usual hypoglycemic reactions might not be encountered, nevertheless, unusual and dangerous reactions might occur at any time. Similarly, the medical staff, with the exception of the persons directly concerned, were kept in ignorance of this project. As a measure of the success in maintaining the degree of vigilance and attention ordinarily present in those who work on the insulin ward, it may be stated that on several occasions the physician in charge was summoned, both during the day and at night, to witness what was interpreted to be an unexpected hypoglycemic reaction.

The first group of patients treated with metrazol consisted of 20 schizophrenics. These reported to the treatment ward for the induction of a convulsion and were returned several hours later to their usual ward locations. Following this, we treated a second group of 20 similar patients, centralizing the group in one ward

where they received their treatment, and in addition intensive resocialization activities.

We then, because of certain theoretical considerations which will be discussed later, treated a group of nine patients with manic-depressive psychoses, depressed phase, or involutional melancholia, in the same way as the second group.

RESULTS.

INSULIN.

Table I represents a tabulation of patients treated with insulin, these being arranged in the order of maximum improvement. It will be noticed that a total of eight are considered as either recovered or markedly improved, and that this group consists entirely of patients who have received a combination of insulin hypoglycemia carried to the level of coma, plus intensive resocialization activities.

The five patients, in whom hypoglycemia was not carried to the coma level, displayed little or no improvement during this period, whereas, after the coma level was attained, four of the five displayed moderate to marked improvement for varying periods of time. At no time did this group receive intensive resocialization activities. Similarly, the group of seven male patients who received insulin hypoglycemia with coma, but otherwise received no special attention, produced only two cases of improvement, the remainder being unchanged.

One patient in this insulin table was diagnosed as a manic-depressive psychotic, manic phase. This patient has had three hypomanic episodes, each of which lasted a minimum of six months. On this occasion she arrived at the hospital approximately one month after the onset of her psychotic episode. She was observed for several weeks to determine the trend of her psychosis, which was found to be on the ascent. She then received 10 days of amytal narcosis following which she was still hypomanic. Twenty-three periods of hypoglycemic coma produced a gradual decrease during treatment in the hypomanic pressure of activity, elation and flight of ideation.

Practically all the patients treated with insulin showed some degree of improvement, chiefly along the lines of better ward

TABLE I.

INSULIN TREATMENT.

FEMALE DIVISION, ELOISE HOSPITAL.

No.	Pat.	Age.	Heredity.	Diagnosis.	Dur. psych.	Treatment.	Attitude to treatment.	Result.	No.
1	C.B.	29	0	Schiz. Par.	3 yr.	I+30 C+G.T.	Apprehensive-cooperative	Soc. Rec.	1
2	M.N.	36	++++	" Mixed	2 yr.	I+46 C+G.T.	Passive-cooperative	" "	2
3	E.H.	20	+	" Cat.	3 yr.	I+36 C+G.T.	" "	" "	3
4	I.L.	33	+	" Par.	10 yr.	I+46 C+G.T.	" "	" "	4
5	M.T.	25	0	Manic Dep. M.	3 mo.	I+23 C+G.T.	Antagonistic-resistive	" "	5
6	C.C.	37	0	Schiz. Par.	2 yr.	I+44 C+G.T.	Apprehensive-cooperative	" "	6
7	B.H.	31	++++	Schiz. Heb.	9 yr.	I+45 C+G.T.	Passive-cooperative	Marked Imp.	7
8	M.S.	23	0	" Cat.	2 yr.	I+53 C+G.T.	" "	" "	8
9	V.H.	27	0	" Par.	3 yr.	I+40 C+G.T.	" "	" " (T)	9
10	I.W.	38	0	" "	2 yr.	I+49 C+0	" "	" " "	10
11	F.D.	34	0	" "	3 yr.	I+39 C+0	Apprehensive-resistive	" " "	11
12	R.K.	31	0	" "	3 yr.	I+54 C+0	" "	" " "	12
13	E.S.	38	0	Schiz. Cat.	10 yr.	I+45 C+G.T.	Passive-occ. resistive	Improved (T)	13
14	M.W.	38	+	" Par.	6 yr.	I+40 C+G.T.	" cooperative	" "	14
15	T.L.	26	+	" Heb.	4 yr.	I+54 C+G.T.	" "	"	15
16	I.B.	34	++	" Par.	6 yr.	I+53 C+0	" "	"	16
17	T.L.	33	+	" Heb.	7 yr.	S+64 0+G.T.	" "	" "	17
18	R.S.	34	0	Schiz. Heb.	10 yr.	S+64 0+G.T.	Passive-cooperative	Slight imp.	18
19	M.Z.	36	0	" Par.	5 yr.	S+64 0+G.T.	Apprehensive-resistive	" "	19
20	M.R.	31	0	" "	2 yr.	S+64 0+G.T.	Passive-cooperative	" "	20
21	V.B.	23	0	" Cat.	7 yr.	I+37 C+G.T.	" "	" " (T)	21
22	L.J.	26	+	" "	2 yr.	I+24 C+0	" occ. resistive	" "	22
23	M.W.	30	0	" Heb.	5 yr.	I+ 8 C+G.T.	" " "	" " "	23

23	M.W.	30	o	" Heb.	5 yr.	I+ 8 C+G.T.				23
24	A.B.	24	o	Schiz. Par.	3 yr.	I+41 C+G.T.	Passive-cooperative	No imp.		24
25	M.S.	37	+	" "	3 yr.	I+42 C+G.T.	" "	" "		25
26	E.G.	39	+	" "	3 yr.	S+49 o+G.T.	Apprehensive-resistive	" "		26
27	G.G.	28	o	" Cat.	3 yr.	I+42 C+o	Passive-cooperative	" "		27
28	M.N.	34	++	" Mixed	6 yr.	I+23 C+o	" "	" "		28

LEGEND.

Heredity:
+—personality defects.
++—isolated occurrence of psychosis.
+++—multiple occurrence of psychosis.
++++—multiple bilateral occurrence of psychosis.

Treatment:
C —Coma.
I —Insulin.
S —Insulin substitute (physiological saline).
G.T.—Group therapy or intensive resocialization activities.

Duration of Psychosis:
Measured from actual onset of symptoms.

Social Recovery —Disappearance of psychotic symptoms with insight.
Marked Improvement—Disappearance of psychotic symptoms without insight.
Improved —Partial disappearance of psychotic symptoms with better ward adjustment.
Slightly Improved —Better ward adjustment.

adjustment, increased sociability and accessibility. This was also noticeable in the group which received physiological saline plus intensive resocialization activities, and we felt that this type of improvement was largely a function of the psychological factors associated with the therapeutic situation.

The attitude of the patient toward treatment, that is, whether fearful, apathetic, submissive or resistive, displayed no definite correlation with the therapeutic results. This finding is in marked contrast to the bearing of this factor upon the results obtained with metrazol as will be noted later. Although theoretically the absence of hereditary taint might appear to indicate a better prognosis, no definite correlations as to this factor could be established.

It is interesting to note that the weight gain per patient during the course of treatment was just as great in the group which received the insulin substitute (physiological saline) as in the groups which actually received insulin, thus substantiating the belief that the alleged nutritional benefits of insulin may be explainable upon the basis of increased attention paid to food intake, possibly because of the fear of hypoglycemic reactions. Although some reports stress gain in weight as a favorable prognostic factor, we were unable to establish such a correlation in our series.

Metrazol.

Table II tabulates the essential data for the patients treated with metrazol in order of therapeutic response. It may be pointed out that among the socially recovered and markedly improved patients are to be found a preponderance of those with affective psychoses, and those for whom metrazol was combined with intensive resocialization activities.

Here also little if any correlation could be established with regard to the age of the patient, whether schizophrenic, manic depressive or involutional, the hereditary background and therapeutic response. The correlation of attitudes toward the treatment is quite striking, the preponderance of apprehensive and resistive attitudes among the favorable therapeutic responses being rather marked, particularly in the affective psychoses. This raises the question as to the part that fear of the treatment may play in determining the therapeutic result.

TABLE II.

METRAZOL TREATMENT.

FEMALE DIVISION, ELOISE HOSPITAL.

No.	Pat.	Age.	Heredity.	Diagnosis.	Dur. psych.	Treatment.	Attitude to treatment.	Result.	No.
1	G.S.	47	+	Iv. M.	5 yr.	10/7 +0	Passive-cooperative	Soc. Rec.	1
2	C.W.	50	o	M. D.-D.	2½ yr.	6/3 +G.T.	Apprehensive-resistive	" "	2
3	B.L.	35	o	M. D.-D.	1 yr.	6/5 +G.T.	" cooperative	" "	3
4	B.S.	24	++	Schiz. Cat.	1½ yr.	20/15+0	Apprehen.; occ. resistive	" "	4
5	F.L.	30	++	Schiz. Cat.	1 yr.	10/5 +G.T.	Apprehensive-resistive	Marked imp.	5
6	M.B.	34	o	Schiz. Par. ?	1 yr.	11/5 +G.T.	" "	" "	6
7	J.J.	49	++	Iv. M.	1 yr.	8/4 +G.T.	" "	" "	7
8	S.M.	39	o	Schiz. Cat. ?	3 yr.	20/14+G.T.	" "	" "	8
9	G.F.	25	++	Schiz. Cat.	3 yr.	41/29+0	" "	" "	9
10	M.H.	30	++	" "	1 yr.	17/13+G.T.	" "	" "	10
11	H.G.	44	o	Iv. M.	1 yr.	10/7 +G.T.	Apprehensive-passive	Improved	11
12	A.C.	45	++	Iv. M.	3½ yr.	16/15+G.T.	" resistive	"	12
13	M.L.	39	++	Schiz. Cat.	3 yr.	5/3 +G.T.	" "	"	13
14	L.R.	46	o	Iv. M.	2 yr.	13/6 +G.T.	" "	"	14
15	M.K.	34	?	M. D. Mixed	10 yr.	22/6 +G.T.	" "	"	15
16	A.B.	34	+++	Schiz. Cat.	6 yr.	4/2 +G.T.	Passive-cooperative	"	16
17	A.F.	35	++	" "	4 yr.	35/24+G.T.	" "	"	17
18	J.F.	26	++	" Mixed	3 yr.	16/15+0	Apprehen.; occ. resistive	"	18
19	I.F.	34	o	" Par.	3½ yr.	16/15+G.T.	Apprehensive-resistive	"	19
20	L.St.	46	o	M.D. Manic	3 yr.	14/13+G.T.	Resistive	"	20
21	E.SA.	31	+	Schiz. Par.	2 yr.	17/15+G.T.	Apprehen. occ. resistive	"	21
22	G.L.	35	++	" Heb.	3½ yr.	22/16+0	Occ. resistive	"	22
23	C.M.	33	++	" Cat.	2 yr.	26/15+0	Passive-cooperative	"	23
24	M.S.	28	o	" Par.	2½ yr.	17/15+G.T.	Apprehensive-resistive	"	24
25	L.Sa.	34	+	" "	1½ yr.	22/15+0	Passive-cooperative	"	25
26	R.B.	32	++	" Mixed	2 yr.	29/22+0	Apprehen.-occ. resistive	"	26
27	L.Sv.	35	o	" Heb.	5 yr.	9/6 +G.T.	Passive-cooperative	"	27
28	T.L.	33	+++	" "	8 yr.	4/2 +0	" "	"	28

29	A.W.	34	o	Schiz.	Heb.	10½ yr.	33/24+o	Passive-cooperative	Slight imp.	29
30	A.H.	34	++	"	"	1½ yr.	36/22+o	" "	" "	30
31	E.W.	28	o	"	Cat.	2½ yr.	30/22+o	Apprehensive-resistive	" "	31
32	R.S.	27	++	"	"	5 yr.	25/24+o	Passive-cooperative	" "	32
33	E.S.	39	?	"	"	10 yr.	15/13+o	" "	" "	33
34	M.M.	31	o	"	"	6 yr.	18/15+G.T.	Apprehen.-occ. resistive	No imp.	34
35	A.C.	33	o	"	Par.	9 yr.	22/15+G.T.	Passive-cooperative	" "	35
36	L.A.	25	o	"	Cat.	3½ yr.	9/5 +o	Apprehensive-resistive	" "	36
37	H.R.	30	o	"	"	10 yr.	18/10+o	" cooperative	" "	37
38	M.R.	35	o	"	"	9 yr.	24/13+G.T.	" resistive	" "	38
39	S.M.	24	o	"	Par.	2½ yr.	20/15+G.T.	Passive-resistive	" "	39
40	M.P.	24	+	"	Heb.	2 yr.	8/8 +o	Apprehensive-cooperative	" "	40
41	G.L.	36	?	"	"	10 yr.	16/15+G.T.	Passive-cooperative	" "	41
42	D.H.	26	++	"	"	6 yr.	21/10+G.T.	" "	" "	42
43	S.C.	33	o	"	Cat.	10 yr.	19/10+o	Apprehensive-cooperative	" "	43
44	A.B.	23	++	"	Par.	2 yr.	24/20+o	Passive-cooperative	" "	44
45	V.B.	22	++	"	Cat.	5 yr.	12/11+o	" "	" "	45
46	I.L.	34	?	"	Heb.	6 yr.	34/22+o	Apprehensive-resistive	" "	46
47	F.B.	33	+++	"	"	7 yr.	33/25+o	Passive-cooperative	" "	47

CONTROL GROUP.

A control group of 30 patients was obtained by listing 90 consecutive admissions of female schizophrenics to the Eloise Hospital during 1936 and arbitrarily selecting every third patient on this list. Of these 30 patients nine have been paroled, of whom six represent recovered or markedly improved patients in the opinion of their attending physicians, giving a spontaneous remission rate of 20 per cent. However, of this group, four patients had their psychoses for less than one year, while in the insulin-treated group the shortest duration was two years, and the average duration approximately four years.

In contrast it may be noted that among those schizophrenic patients treated with the most favorable combination of insulin plus coma, plus intensive resocialization activities, seven out of 20, or 35 per cent, exhibited social recovery or marked improvement compared with 20 per cent in the control group. If we include those patients showing temporary improvement, the percentage would rise to 53 per cent.

The results obtained with metrazol in chronic schizophrenia are less striking. However, even here the number of recovered or markedly improved patients equals 16 per cent, a figure in excess of the spontaneous remissions which may be expected with a duration of psychosis of one year or more.

The nine patients with affective psychoses reveal a brighter picture. Eight of the nine treated were either improved, markedly improved, or recovered, the percentage of recovered and markedly improved patients in this group being 44.

It is interesting to compare this with the results obtained by one of us (L. S. L.) and Dr. L. J. Bailey with a group of 15 involutional patients treated with large doses of estrogenic substance. This treatment was carried to the point where the level of blood estrin was restored to the normal pre-menopausal level. In this group only two of the 15 patients, or 13 per cent, were improved.

DISCUSSION.

Our results have shown that the production of coma in the insulin treatment is essential. Neither intensive resocialization

activities alone, nor insulin hypoglycemia without coma, nor the combination of these produced comparable results. The actual reproduction of the hypoglycemic setting, using physiological saline instead of insulin, when combined with intensive resocialization activities was similarly ineffective.

However, we have found that the addition of intensive resocialization activities markedly increases the therapeutic potency of the insulin coma so that a combination of these factors, that is, insulin plus coma plus intensive resocialization, constitute the ideal therapeutic situation. Apparently the insulin coma is responsible for the first break in the schizophrenic armor, intensive resocialization then serving as a wedge to enlarge the opening.

In our opinion the ideal insulin routine is as follows: insulin in dosage sufficient to produce coma approximately two hours after administration, the optimum period of coma being not less than one, nor more than two hours. The occurrence of a lucid interval in pre-coma after previous production of comas is a favorable prognostic sign and an indication for interruption of hypoglycemia; if ignored, a favorable therapeutic opportunity may be missed.

Many complications may be avoided by lowering the coma-producing dose as the treatment progresses. Some type of conditioning by which the threshold for the production of coma is lowered by repetition apparently develops in the majority of patients. Frequently by such procedure the dosage in the latter part of treatment may be lowered as much as 50 per cent without any diminution in the intensity of the coma. We feel that the absence of fatalities in our series of cases is due to the recognition and utilization of this fact and, in addition, the arbitrary limitation of the period of hypoglycemia to a maximum of five hours.

Throughout the treatment psychological factors are of considerable importance. The general therapeutic setting in the pre-coma and emergence from coma lends itself at first toward the production of an attitude of infantile dependence. This attitude of dependence and trust, if wisely utilized by the attending physician, may serve as the focal point for the gradual addition of social responsibilities and performances. Indeed, this phase is important because if unrecognized and the acceptance of additional social responsibilities not forced at this time, the patient

may derive sufficient satisfaction from this infantile regression to prefer to remain fixated at this level. It is our feeling that some of the unsatisfactory therapeutic results are due to inability to wisely utilize this development.

Fear of impending danger may also play a part. Of interest in this regard is the reply of patient, M. M., who, when asked in what way he felt benefited by insulin, stated "Doctor, I realize that by giving me insulin, you have the power to take me out of the world and that if you did not give me something to bring me back, I would be gone. Therefore, I feel it necessary to cooperate with you and remain in your good favor."

In addition, the reactivation of psychotic symptomatology so commonly seen during the pre-coma and emergence from coma, during which panic and acute hallucinatory states and various regressive phenomena may be observed, may have some cathartic value.

The way in which the metrazol-induced convulsion benefits the schizophrenic is obscure. However, here also the difference in response between the combination of metrazol plus intensive resocialization activities, and metrazol alone, is striking.

It is our observation that fear of treatment is much more marked with the use of metrazol than with insulin, and that this fear of treatment is of considerable therapeutic potentiality. It frequently causes the patient for a time at least to make a better superficial ward adjustment in an attempt to avoid further treatment, even though the actual content of the psychosis itself remains unaltered. These effects of metrazol are, in our opinion, largely non-specific, and very similar to the striking, although usually transient, improvements observed in schizophrenic patients in response to a large number of accidental shocks as the result of extreme pain due to injuries, operative intervention for acute surgical conditions and acute febrile elevations due either to illness or non-specific foreign protein therapy.

We have also found metrazol valuable in the treatment of chronically disturbed patients regardless of type where our objective was better ward adjustment and not necessarily social recovery. Ten of the worst patients in the institution from this point of view were selected. They responded to the extent that seven

could be transferred from disturbed to dormitory wards and enter into a fairly complete range of occupational and recreational activities.

With regard to the results obtained in the affective psychoses, particularly in involutional melancholia, it is important that these results were more favorable in those patients whose psychoses revealed a strong sense of guilt and need of punishment. Even in schizophrenic patients the therapeutic response was related to the amount of guilt shown.

We were first impelled to treat the affective psychoses in this way because of a clinical experience of one of us (L. S. L.).

A 30-year-old married woman of Italian descent, resident in the hospital for two months, was continually agitated, expressing ideas of unworthiness, sin and body disturbance. She repeatedly urged that she be permitted to die or that some one kill her. One morning while an attendant stooped to pick up some soiled linen and deposit it in the clothes chute on the fifth floor, this patient vaulted over the attendant and threw herself down the chute. Fortunately, the lower floors had previously deposited their linen, so that she landed on a soft heap. When seen several minutes later on her return to the ward, she was physically unharmed, except for a few scratches. She begged to be forgiven for her act, but otherwise showed a rather striking disappearance of her agitation and delusional content. Several days later it was noted that she was apparently well. She was paroled and has adjusted satisfactorily for the past four years. The theoretic formulation in this case was that the patient had satisfied her need for punishment by this act, her survival being accidental and no fault of her own. With the satisfaction of this need for punishment, there was relief of the feelings of guilt and consequent amelioration of her psychosis.

On the basis of this clinical experience, several patients with similar psychotic content and spontaneous desire for punishment were selected, and after rather careful physical examination, and with much trepidation on our part, because of their age, metrazol was given. The results, as already indicated, whether the theoretical formulation be correct or not, are rather striking.

The initial enthusiasm on the part of the staff for both therapies is apparently not a significant factor, for the results with the most recently treated groups are, if anything, better than with the earlier ones; this, despite the fact that the personnel concerned remains the same.

A survey of the duration of schizophrenic psychoses at the

time of first admission to the Eloise Hospital confirmed our impression that the majority were of more than one year's duration. In fact, during the past six months only six female schizophrenics were admitted who had a duration of psychosis of less than six months, the total female schizophrenics admitted during the same period exceeding 100. Inasmuch as it is recognized that insulin and metrazol shock are therapeutically most efficient in patients whose psychoses do not exceed six months' duration, it is felt that the education of the public to this fact, with an earlier recognition of mental disease and prompt admission to the mental hospital would markedly increase our therapeutic efficiency.

CONCLUSIONS.

1. Seven of 20, or 35 per cent, of a group of chronic schizophrenic patients were benefited by insulin to the point of social recovery or marked improvement, as compared with a 20 per cent spontaneous remission rate in a control group of 30 patients.

2. Coma was found to be essential for the production of improvement, its effectiveness being increased when combined with intensive resocialization activities.

3. Metrazol was only one-half as effective as insulin in chronic schizophrenia, but justified its use because of its greater ease of administration and lesser cost. In chronically disturbed patients its use frequently resulted in moderate degrees of improvement resulting in better ward adjustment.

4. Paranoid schizophrenia responded best to insulin; agitated catatonia to metrazol.

5. Neither the age of the patient nor the degree of hereditary taint could be correlated with the therapeutic responses.

6. In involutional melancholia and manic-depressive psychosis, depressed phase, metrazol treatment appeared to constitute a direct attack upon the specific psychopathology of the disease. A theoretic formulation to explain this action was offered.

7. The relatively long duration of schizophrenia in first admissions to the Eloise Hospital was noted and the need for earlier recognition and more prompt admission stressed, so as to enhance our therapeutic effectiveness.

BIBLIOGRAPHY.

1. Cameron, E. D.: Further experiences in the insulin hypoglycemia treatment of schizophrenia. J. Nerv. and Ment. Dis., 87: 14-25, 1938.
2. Cheney, C. O., and Drewry, P. H.: Results of non-specific treatment in dementia præcox. Am. J. Psychiat., 95: 203-217, 1938.
3. Cohen, L. H.: The early effects of metrazol therapy in chronic psychotic over-activity. Am. J. Psychiat., 95: 327-333, 1938.
4. Gellhorn, E.: Effects of hypoglycemia and anoxia on the central nervous system. J. A. M. A., 110: 1433-44, 1938.
5. Orenstein, L., and Schilder, P.: Psychological considerations of the insulin treatment in schizophrenia. J. Nerv. and Ment. Dis., 88: 397-413, 644-660, 1938.
6. Read, C. F., Steinberg, L., Liebert, E., and Finkelman, L.: Use of metrazol in the functional psychoses. Am. J. Psychiat., 95: 781-786, 1939.
7. Ross, Jno. R.: The pharmacological shock treatment of schizophrenia. Am. J. Psychiat., 95: 769-779, 1939.
8. Rossman, I. M., and Cline, W. B.: The pharmacological "shock" treatment of chronic schizophrenia. Am. J. Psychiat., 94: 1323-1336, 1938.
9. Sakel, M.: Pharmacological shock treatment of schizophrenia. Nervous and Mental Disease Monograph, No. 62, 1938.
10. Schilder, P.: Notes on the psychology of metrazol treatment of schizophrenia. J. Nerv. and Ment. Dis., 89: 133-144, 1939.

OBSERVATIONS OF INSULIN SENSITIVITY.

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Individual sensitivity to insulin has long been recognized, but not until the advent of hypoglycemic shock therapy was the extent of variation among non-diabetics observable. Everyone interested in this therapy must be impressed with the wide differences in the coma dose within any group of individuals, but the changed resistance to insulin during the weeks of treatment is not so well appreciated. Shock from insulin may be considered present when there is apparent a mental, motor or physiologic response directly attributable to the effects of the drug. The sequence of the symptoms of shock is subject to individual pattern, and cannot be used as a guide for the degree of sensitivity. Nor is the convulsion a suitable indicator of optimum tolerance. Convulsions may occur early in treatment before the coma dose has been established. They may appear with a coma dose during the second and third hour of hypoglycemia, less frequently during coma, and after the patient is awakened. Convulsions may be inhibited by elimination of outside stimuli, by premedication with barbiturates and by increasing the dose of insulin. Likewise patients can be made predisposed to convulsions. For the purpose of this communication the development of coma in the fourth or in the beginning of the fifth hour is chosen as the measure for guidance.

Two types of sensitivity are distinguishable, as determined by the amount of insulin required to produce the first coma, and the necessary changes in dosage during the course of treatments. When the initial resistance exceeds 300 units, Braunmühl [1] calls this absolute resistance. To overcome it he has devised the *Zickzackmethode* which is successful in a high percentage of cases. The changed resistance manifest during the course of treatments he calls relative resistance. Representing the variation in initial resistance the writer has observed an instance of 500 units without deep coma,[2] and the case here to be described of coma with the first dose of 20 units. The course of the relative resistance may change direction more

than once; rise to more than twice the first coma dose, or drop to less than one-third. It seems unwise to begin treatment with an initial dose of 60 units [3] without some knowledge of the patient's sensitivity to insulin.

There is no measurable relationship between sensitivity to insulin and duration or chronicity of the psychosis. There is no conclusive support for the statement that "the optimum dose, once reached, is . . . . almost never being reduced in these chronic cases." [4] It has been observed on several occasions that the phlegmatic type of hebephrenic and paranoid schizophrenics undergoing a second course of insulin shock treatments show a higher initial tolerance.

The importance of relative resistance among treated cases may be judged from the fact that the first six graphs here presented are from the first 12 patients treated at this hospital. Fuller histories were given in an earlier publication.[5] To date 26 patients have completed treatment with the incidence of changed relative resistance far above 50 per cent. It is the exceptional case that does not require some adjustment of insulin dosage after several comas. Müller [6] has stated that "two-thirds of the cases exhibit hypersensitivity which may go so far that less than one-half the original dose becomes sufficient to produce the same clinical reaction as the latter."

Most workers have experienced difficulties at some time in arousing a patient from coma. Necropsies have established that in fatal cases brain pathology is always present. There is little doubt that in cases of difficult arousal the recuperative capacity of the brain has suffered through excessive insulin effect. This does not apply to cases where glucose by gavage fails and a small amount intravenously promptly awakens the patient. Of 13 deaths [7] in the first 1000 cases treated in New York civil state hospitals, the majority followed protracted coma. The early literature contained little reference to the question of sensitivity. In 1938 the July issue of the *Psychiatric Quarterly* contains two reports on prolonged coma. A total of 14 cases are reported for approximately 160 patients treated. Whether this complication can be entirely avoided may be answered in the negative, but the importance of changed sensitivity as a cause of such accidents is beyond question. Insulin shock therapy has already proven itself a valuable contribution to therapeutics in psychiatry. Our knowledge of its pharmacologic effects

in coma doses is incomplete, and every clinical observation of its activity adds safety to the new treatment.

This communication presents graphic illustrations of changed sensitivity with case material to indicate a probable relationship between excessive dosage and protracted coma. Graphs 6 and 7 illustrate the effective use of the zigzag method for raising the sensitivity to insulin. Graph 8 illustrates a case of unusually high sensitivity.

GRAPH 1 (S-239).—A single woman, age 27, had been a patient in this hospital 15 months without sign of remission. She was untidy, destructive to clothing, unclean in habits, mute or echolalic and assumed fixed positions. Even with prolonged tubefeeding she lost weight to emaciation. She went into her first coma with 110 units of insulin. The next day with the same dose she had a convulsion during the precoma period, but was afterwards allowed to pass into coma. The following day the insulin dose was lowered to 100 units and she reached coma at the end of the fourth hour after injection. For the next treatment she received 105 units, and a metrazol-induced convulsion while in the third hour of hypoglycemic shock. Thereafter there was a sharp rise in sensitivity which continued to mount as the treatments were discontinued. The last coma was induced with 50 units of insulin.

GRAPH 2 (S-538).—A married woman, age 38, acutely ill for four months. She was destructive, resistive and violent, soiled and smeared herself, was tubefed for days at a time and lost much weight. Her mood and activities were apparently governed by autistic experiences. After recovery she said that during her illness she thought her ideas were being stolen or censored, felt her bed charged with electricity, and believed her room was wired for dictaphones. She had the first coma with 70 units of insulin. The same dose the next day produced only stupor. Her dose of insulin was increased by five units with each successive treatment up to 105 units, despite the fact that she had coma with each dose above 70 units. She had a spontaneous convulsion during hypoglycemia with the 75, 80, 85, and 90 units doses. The rationale of increasing the insulin after a spontaneous convulsion is based on repeated observations that it is effective in eliminating the susceptibility to convulsion, more effective than pretreatment medication with barbiturates. As the graph indicates the subsequent course was without this complication. The only other convulsion in the course of the treatments was induced with metrazol. Before the close of treatments her coma dose had dropped to 35 units.

GRAPH 3 (S-510).—A single man, age 27, sick about six months, exhibited seclusiveness, mannerisms, auditory hallucinations and ideas of reference. He was untidy in dress and dull emotionally. His first coma was obtained with 150 units of insulin. Because coma appeared late, during the fifth hour, his dosage was raised gradually to 160 units for the next three

treatments. During the period of treatments before coma was obtained, as the dosage mounted, and during the precoma stage of his attained comas, the patient exhibited great excitement and straining. In such type of reaction it has been found that with treatment more than once a day with a subcoma dose or zigzagging the dosage until the sensitivity to the insulin is raised, coma on smaller doses of insulin is induced with less excitement and motor activity. The procedure illustrated in the graph proved effective. His last coma was obtained with 55 units. He could easily have passed to coma with 45 units had not the hypoglycemia been terminated when he reached light stupor in two and one-half hours.

GRAPH 4 (S-572).—A married woman, age 28, mentally ill eight or nine months; was clinically considered an agitated depression with paranoid trend and ideas of reference. She reached her first 12 comas on successive days of treatment with a maximum dose of 50 units for the tenth coma, and a minimum of 35 units for her twelfth coma. Thereafter her resistance to insulin mounted to 150 units for her maximum coma dose, two days before treatments were completed.

GRAPH 5 (S-452).—A married woman, age 39, was a paranoid schizophrenic with a duration of illness of two and one-half years. She had loosely systematized delusions of persecution based upon the fact of an illegitimate child during her senior year in high school. She believed that her history was broadcast over the radio, reported in the newspapers and performed on the stage and in the movies. Her life story was being commercialized. People in daily contact made disparaging remarks, scoffed at her or told lies about her life. She had episodes of unprovoked excitement with abusiveness and occasionally assaulted strangers. During quiet periods she was silly and erratic, emotionally and intellectually inadequate.

She reached her first coma with 75 units of insulin. On the two preceding days of treatment, 65 and 70 units respectively produced only stupor but with slow recovery from the hypoglycemic shock. For more than an hour her sensorium showed clouding and she felt "a little light." With the 70-unit dose she required glucose intravenously 45 minutes after drinking a neutralizing dose of sugar solution. Her awakening from the first coma was uneventful. When on the next day the dose was raised to 80 units there was difficulty restoring full consciousness. In addition to 150 gm. glucose by gavage she required 50 gm. glucose by vein. Even the next day, given as a rest day, she was unusually quiet, listless and felt weak. The next two comas were obtained with 60 and 65 units respectively. With the last she regained consciousness in 45 minutes and ate a good lunch. After lunch she was out for a walk on the grounds, participated in ball play and seemed to have enjoyed listening to a radio program. Four hours after termination of hypoglycemia she complained of feeling weak, that while in the gymnasium she had difficulties in following the ball, and that she felt as though walking on air. For three days after this without further treatments, she was nauseated and vomited most of her meals. She was in contact with her surroundings but

preferred to stay quietly in bed. Occasionally she complained of tiredness, weakness, dizziness and neuralgic pain in the back of her head. Blood sugar before breakfast on the third and fourth days was 103 and 107 mgm. per cent. By the seventh day she felt well enough to attend an evening social. She still tired easily and preferred inactivity. Treatments were resumed after a ten-day period of rest. She did not have a deep coma until the insulin dose gradually reached 110 units. Coma on consecutive days of treatment was not obtained until she was receiving 135 units and over. Her maximum insulin dose was 155 units, without any of the difficulties found earlier. At the conclusion of her treatments she had a coma on 100 units of insulin, and apparently her sensitivity was mounting.

GRAPH 6 (S-500).—A married woman, age 24, was mentally ill for nineteen months. In the beginning she was depressed, agitated and fearful; she believed herself infected with syphilis, that she had ruined and disgraced her family, and had cleanliness compulsions. Later she was paranoid about "strangers" robbing and ruining her family; her home was wired with dictaphones to spy on her. Her thoughts were being read and stolen. Voices accused her of ruining her own and her mother's lives. She repulsed everyone with tirades as "strangers who want to harm me and mine."

She had her first coma on 280 units. On the evening of the third day following, after a treatment with 270 units without developing coma, she had a secondary shock with loss of consciousness, diaphoresis and myoclonic activity. This was relieved by 100 gms. of glucose by gavage. After a day of rest treatments were resumed, and it was necessary to increase the insulin for satisfactory coma to a maximum of 310 units. Thereafter her sensitivity was raised by zigzagging the dosage. As the graph indicates the method was successful. Before treatments were discontinued she had on three occasions reached coma with 120 units. There were no recurrences of the secondary shock.

GRAPH 7 (S-647).—This case illustrates a high absolute tolerance and the effectiveness of the zigzag method in raising sensitivity. It also demonstrates absence of a predictable relationship between insulin dose and sugar level in the same individual.

A single man, age 23, a sturdy athletic type, a hebephrenic schizophrenic, first exhibited active symptoms of his illness three years ago. During his sophomore year in college he failed so completely that he was dropped from the school. He was home for months, seclusive and reticent. He unsuccessfully tried selling commodities by house to house canvass. Finally, in the summer of 1937 he reviewed his school work and was readmitted conditionally to college. In a few weeks his concentration failed; he was preoccupied, seclusive, noticed to be posturing, and was irrelevant and inappropriate in his replies to questions. Emotionally he was dull and disinterested.

Early in 1938 he was committed to a state hospital. He received a course of hypoglycemic shocks, including 50 comas. The coma dose was consistently 380 units of insulin. He relapsed one week after parole from the hospital,

and was admitted to this institution in November for the purpose of giving him another series of shock treatments. He reached the maximum dose of 400 units without obtaining a deep coma; he could be roused to drink his sugar solution. His blood sugar at 11.00 and 12.00 a. m. was 56 and 60 mgm. per cent respectively. Insulin doses were thereafter zigzagged as shown in the graph. During his first coma on 150 units blood sugars were taken at approximately the same hours of hypoglycemia, 11.00 and 11.55 a. m., and the values obtained were 53 and 50 mgm. per cent. Of the 19 comas produced in his series of treatments, 10 were obtained with 150 units of insulin.

Our last case is presented because it is believed that the patient showed a lower tolerance for insulin than any yet recorded in experiences with hypoglycemic shock therapy. Gillman and Parfitt [8] reported a case of coma on 17 units of insulin. This case is of special interest because it is also an excellent example of changing sensitivity. Their patient reached the first coma with 50 units, dropped to 17 units for the 15th coma, and by the 41st coma the dose of insulin had mounted to 120 units. James, *et al.*[9] mention a case of coma on 15 units of insulin. An article by Schatner and O'Neill [10] mentioned a coma on five units, but a personal communication from one of the authors [11] declared this a misprint for 15 units. Langfeldt [12] reported a case of unconsciousness and generalized convulsion forty minutes after an intravenous injection of 7 units of insulin. While a convulsion is not an indicator of optimum tolerance, the author pointed out that his patient had an "opium cure" immediately preceding the insulin injection, and this might have affected the sensitivity by depressing the production of adrenalin. We know of nothing to account for the unusual sensitivity in our patient.

GRAPH 8 (S-484).—A married woman, age 47, was actively psychotic for two years. Her first personality changes were observed after cæsarian section for the birth of her only child nine years ago. She became seclusive, lost interest in outside activities, distrustful of people's intentions, and doted over the child even at the expense of other obligations to her home. The active manifestations of her psychosis were precipitated when the child failed to appear at the usual moment after school. The patient screamed for police and neighbors to help, that her child was kidnapped. The services of a physician were necessary to quiet her, but she could not be reassured until the child was actually home. There was partial recovery from this episode in about a week. She continued quietly to voice the belief that there was a gang of foreigners plotting to kidnap the child; her husband was being followed and would be killed; she was being spied on and false

stories were being published about her. Two months later, after an exacerbation of her excited state, she was hospitalized. She heard voices, thought her food was being poisoned and felt she was spied upon. She was untidy, lost interest in all activities. Occasionally she was aggressive or did injury to herself in order to accuse the nurses of mistreatment. Dull and apathetic, she would show animus and active resistiveness when urged to participate in activities.

She reached coma on the first day of treatment with 20 units of insulin. During the first four weeks the coma dose fluctuated between 15 and 25 units, then for the next two weeks she had deep coma with 10 units of insulin. Quite abruptly her tolerance rose to 35 and 30 units at the conclusion of eight weeks of treatment. The only unusual happening before the rise in tolerance was a disturbing visit from her husband. He told her of his intention to divorce her, that the home she left no longer existed, and that he would take the custody of the child because she had been an inmate in a mental hospital. She had shown pronounced improvement both mentally and physically, and looked forward to an early release up to the time of this visit. Patient reacted to the news with depression, restlessness and return of the paranoid trend; believed her husband was a victim of sinister persons who forced him to act in this way toward her. She never regained the level of improvement shown at the end of six weeks of treatment. At the close of eight weeks of treatment she was considered well enough to try parole, but plans for this repeatedly failed, and each failure added color to the psychotic picture at the end of three more weeks she was too sick for parole, and shock therapy was resumed for another four weeks. She was paroled to the custody of a sister. In the second course of treatments her tolerance for insulin was much higher.

COMMENT.

The observation of relative resistance or changed sensitivity to insulin in the course of shock therapy is of major importance. The relationship between excessive insulin dose and difficulty in awakening the patient from coma seems certain. The effect of hypoglycemia on the brain whether due to anoxia or some other mechanism, results in pathological changes which may pass the stage of reversibility. The author [5] has mentioned several cases of paresthesia following shock treatment. Most workers have seen transient hemiplegias of various parts of the body. Recent experimental work by Accornero [13] confirms the impression that insulin in coma doses, even in non-fatal cases, produces edema and focal alterations in the brain.

SUMMARY.

The subject of sensitivity to insulin in the course of shock therapy was discussed in relation to this new treatment for mental diseases. A case of unusually high sensitivity was described, and the effective use of Braunmühl's *Zickzackmethode* for absolute or high resistance to insulin was illustrated.

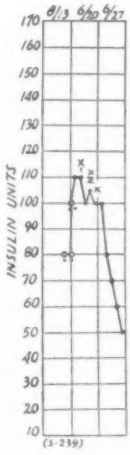
BIBLIOGRAPHY.

1. von Braunmühl, A.: Die Insulinshockbehandlung der Schizophrenie. Verlag von Julius Springer, Berlin, 1938.
2. Tillim, S. J.: A clinical consideration of hypoglycemic shock therapy. Medical Record, 148: 3, 112, Feb. 2, 1938.
3. Vander Veer, A. H., and Reese, H. H.: Treatment of schizophrenia with insulin shock. Am. J. Psychiat., 95: 2, 271, Sept. 1938.
4. Rossman, I. M., and Cline, W. B.: The pharmacological shock treatment of chronic schizophrenia. Am. J. Psychiat., 94: 6, 1323, May 1938.
5. Tillim, S. J.: Insulin and metrazol in the treatment of psychoses. Pending publication in The Psychiatric Quarterly.
6. Müller, M.: The insulin therapy of schizophrenia. Am. J. Psychiat., 94: Suppl., 5, May 1938.
7. Maltzberg, B.: Outcome of insulin treatment of one thousand patients with dementia præcox. Psychiat. Quarterly, 12: 3, 528, July 1938.
8. Gillman, S. W., and Parfitt, D. N.: Insulin sensitivity and sensitisation. The Lancet, 2: 13, 743, Sept. 25, 1937.
9. James, G. W. B., *et al.*: Insulin shock treatment of schizophrenia. The Lancet, 19: 1, 1101, May 8, 1937.
10. Schatner, M., and O'Neill, F. J.: Some observations in the treatment of dementia præcox, Psychiatric Quart., 12: 1, 5, Jan. 1938.
11. Schatner, M.: Personal communication, Feb. 2, 1939.
12. Langfeldt, G.: Insulin sensitivity and opium cure. The Lancet, 2: 1135, Nov. 13, 1937.
13 Accornero, F.: Histopathology of the central nervous system in insulin shock. Rivista di Patologia nervosa e mentale, 53: 1, pp. 1-96, Jan.-Feb. 1939.

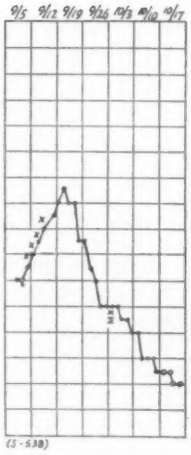
EXPLANATORY NOTE FOR GRAPHS.

(a) Circle indicates without coma; (b) circle and dot, semicomatose, required gavage for termination of hypoglycemia; (c) large dot indicates coma; (d) X, indicates spontaneous convulsions; (e) MX, metrazol-induced convulsion.

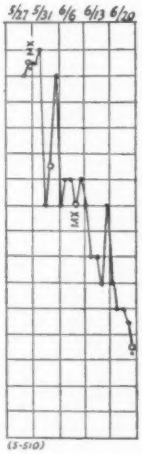
All graphs, except No. 1, begin with the first coma, or the maximum insulin dose used for the particular patient, and end with close of the treatments.



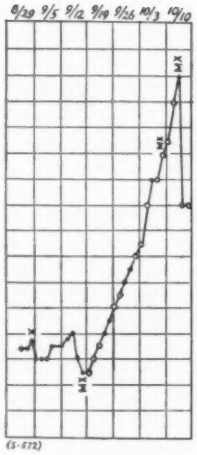
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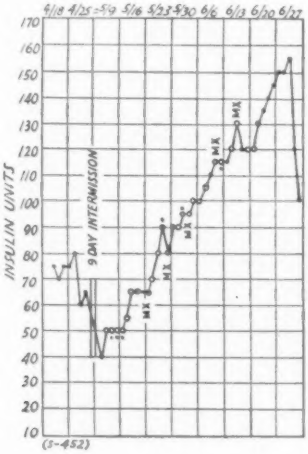
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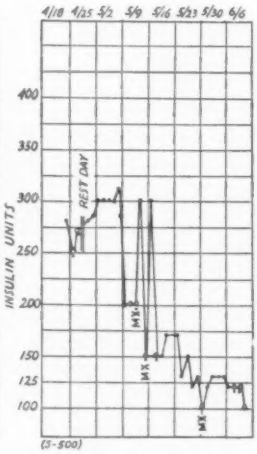
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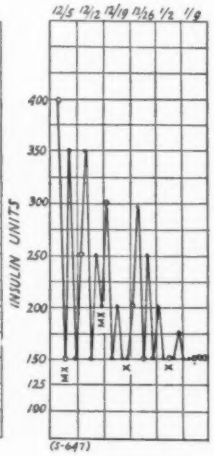
GRAPH 4.



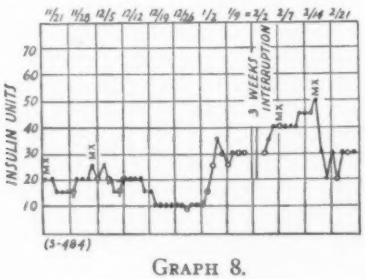
GRAPH 5.



GRAPH 6.



GRAPH 7.



GRAPH 8.

THE MECHANISM OF THE SYMPTOMS OF INSULIN HYPOGLYCEMIA.*

By H. E. HIMWICH, M. D., J. P. FROSTIG, M. D., J. F. FAZEKAS, AND Z. HADIDIAN.

The clinical symptoms resulting from the administration of insulin have been known since the introduction of that hormone for the treatment of diabetes. But the symptoms observed after insulin administration for diabetes as well as those described during spontaneous hypoglycemia are slight in comparison with those which occur during the treatment of schizophrenia. The dosage used in this treatment is larger, the time in which the insulin is permitted to operate longer, and an entirely different picture results. Sakel stressed that severe cerebral symptoms may endure for hours without threatening life. Angyal [1] and Frostig [2, 3] disclosed that the cerebral symptoms of hypoglycemia develop in a rather definite sequence. Frostig recorded the exact order and time of appearance and disappearance of each symptom. With his co-workers he was able to show that the sequence of the symptoms does not depend upon the dose of insulin, an increase of the dose speeds the cycle of symptoms, while a decrease slows its curve. But the order of the symptoms remains unchanged. An analysis of 6587 treatments on 195 cases (in preparation by Dr. Frostig) revealed that the optimum period for the duration of the insulin effect is approximately five hours. The following table presents the symptoms in the order in which they appear during such a 5-hour period. With the exception of loss of consciousness which developed in all shocks the symptoms which are in italics appeared in 50 to 90 per cent of the treat-

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Ill., May 8-12, 1939.

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ments; the symptoms in roman type in 20 to 50 per cent. Exceptions to this sequence of symptoms are rare, however in 2.8 per cent of the observations convulsive seizures bring the course of symptoms to a dramatic climax. These exceptions are likely to occur as the coma becomes more shallow. In such cases the symptoms regress to a previous phase of their sequence and do so in an order which is the reverse of that of their appearance.

From this table it may be seen that the effects of insulin hypoglycemia descend through the different layers of the brain, in a sequence which is determined phylogenetically. First the activity of the cortex becomes suppressed releasing the basal ganglia and the hypothalamus. As time proceeds these symptoms disappear in turn and new ones arise which may be associated with the midbrain. Still later mesencephalic signs give way to manifestations of parts of the brain stem caudal to the nucleus ruber, including the medullary centers. The disappearance of the corneal reflex is an indication for termination for if the hypoglycemia is permitted to continue beyond this point the cerebral changes may become irreversible.[4] The question of the exact localization of each symptom is a difficult one and not only so during hypoglycemia. But in our opinion there is no doubt of the general progress of the symptoms starting from cerebral cortex and going toward the medulla oblongata.

What physiological data are at hand to throw light on the mechanism of the symptoms? The physiological results reveal that the brain is unique among organs since it is the only one to oxidize carbohydrate predominantly.[5, 6] Though the brain is composed largely of lipoid matter, fats are not oxidized.[7, 8] Even in complete diabetes, when the rest of the organism obtains most of its energy from the oxidation of fat, the brain continues to oxidize carbohydrate.[5, 9] The cerebral supply of reserve carbohydrate in the form of glycogen is only a meager one.[10] The brain is, therefore, largely dependent on the sugar of the blood to satisfy its energetic demands.[11, 5] It is, therefore, understandable that hypoglycemia should reduce the metabolism of the brain.[12, 13, 14]

Cerebral metabolism depends upon foodstuff and oxygen. The volume of the oxygen used by the brain can be determined by comparing the oxygen contents of the arterial blood with that of the internal jugular vein. If metabolism of the brain is diminished as a result of hypoglycemia, the utilization of the oxygen of the blood

ABRIDGED TABLE OF SYMPTOMS

Hours	Symptoms		Localization
	Latent Time		
First	*Somnolence* *Hypotonia*	*Perspiration*	Depression of Cortical and Cerebellar Activity
Second	*Clouded Consciousness* Fine Tremor *Speech and Perceptual Disturbances*	Motor Excitement Psychotic Syndromes	
	Fall of Temperature *Diminished Exteroceptive Sensitivity*		
Third	*Loss of Consciousness* *Primitive Movements* *Forced Grasping* *Clonic Twitchings* Myoclonoid Twitchings Choreiform, Athetoid, and Hemibalistic Movements Torsion Spasms	Motor Restlessness *Pulse Rate Increased* *Exophthalmus* *Dilated Pupils* *Light Reaction Present* *Increased Exteroceptive Sensitivity*	Release of the Basal Ganglia and Hypothalamus
Fourth	*Tonic Spasms* Tetanic Postures Deepening Stupor Increased Tonus Coarse Tremor Torsion Spasms	*Depression of Pyramidal Functions*	
	Increased Proprioceptive Sensitivity. Diminished Exteroceptive Sensitivity		
Fifth	*Tonic Extensor Spasms* Postural Reflexes Coma	Pin Point Pupils No Light Reaction Pulse Rate Decreased Shallow Respiration	Release of Medulla Oblongata
	Diminished Sensitivity No Corneal Reflexes		

which passes through the brain must also be decreased. Previous investigations [15] disclose that the oxygen uptake of the brain is diminished during hypoglycemia from the normal average of 6.81 volumes per cent to an average of 2.57 volumes per cent when unconsciousness supervenes and then to 1.77 volumes per cent before the administration of sugar, a measure which may raise the utilization of oxygen to the normal level within a few minutes. Spontaneous awakening sometimes produces the same effect.

Other correlations were disclosed by the aid of the electroencephalograph. Hoagland, Rubin and Cameron [16] observed that alpha waves suffer a progressive decrease of their frequency and during hypoglycemia are slowly replaced by delta waves. As the effect of insulin deepens, the alpha waves finally disappear, while the delta index augments. These investigators [16] note a parallelism between the decline of the blood sugar curve and the changes in the electrical activity of the brain. The administration of carbohydrate causes a speedy return of the normal activity. Further electroencephalographic studies [15] reveal that a better correlation is afforded by the oxygen uptake than the blood sugar for the latter reaches its minimum approximately 2 hours after the injection of insulin, while the oxygen uptake decreases throughout the period of hypoglycemia. The present investigation is a cooperative undertaking prompted by the desire to compare the effects of insulin hypoglycemia by three different methods. Some of us (Himwich, Fazekas, and Hadidian) have been studying physiological changes including the metabolic and electrical while another member of this group (Frostig) has been observing the clinical symptoms of insulin hypoglycemia. Previous results obtained with these methods were consistent, in that they revealed an ordered and reproducible series of changes. It, therefore, appeared possible that a few crucial experiments might serve to correlate the physiological and clinical observations.

METHOD.

Patients with schizophrenia receiving coma-producing doses of insulin were chosen for this study. Before insulin was injected each patient received a careful clinical examination, then brain waves were recorded, and finally samples of blood were drawn from the internal jugular vein [17] and femoral artery. Clinical ex-

aminations were repeated every quarter of an hour. The brain waves were recorded by an ink writing undulator in the manner described by Hoagland and co-workers [16] with an input lead connected to the scalp over the occipital region. The two indifferent electrodes were firmly placed on the mastoid processes. Records were taken each one-half hour except when more frequent observations were required by the rapidly changing condition of the patient. Whenever marked alterations occurred in the electroencephalogram, blood samples were collected and analyzed for oxygen [18] and glucose [19] in order to estimate cerebral metabolism.

RESULTS.

Four series of simultaneous observations were made on two patients. The results are recorded on special charts. For brevity only the main group of symptoms are presented. They may be read with the aid of the following key:

KEY.

Time: hours after injection of insulin.

I = cortical syndrome.
II = general symptoms of the autonomic nervous system (perspiration and salivation).
III = loss of consciousness.
IV = symptoms due to the release of the basal ganglia (primitive movements, forced grasping, hyperkinetic syndrome).
V = sympathetic syndrome (hypothalamus).
VI = tonic-tetanic syndrome.
VII = tonic-extensor syndrome.
VIII = parasympathetic medullary syndrome.
BS = blood sugar in mg. per cent.
OU = oxygen uptake in vol. per cent.
A = alpha frequency per second.
D = delta index in centimeters.
--- = slight intensity of the symptoms.
≡≡≡ = moderate intensity of the symptoms.
+++ = great intensity of the symptoms.

Chart I, Patient E. B. 9/12/38, dose of insulin 196 units.

The course of the symptoms is as follows: patient becomes somnolent early and loses consciousness slowly. Shortly after loss of consciousness (III) at the end of the second hour primitive movements are observed. Forced grasping persists for an hour (IV), tonic phenomena appear early

(VI) and in the last hour tonic extensor spasms are interspersed (VII). Autonomic symptoms take their usual course exhibiting sympathetic symptoms (hypothalamus) (V) of exophthalmus, dilated pupils and rapid pulse during the third hour. Parasympathetic signs are present in the last hour (VIII). The clinical response may be characterized as poor in motor symptoms with a marked depression of consciousness. The patient awakes within 5 minutes after administration of sugar with no untoward symptoms.

Blood sugar decreases from 80 mg. per cent to 22 mg. per cent. It is striking that approximately the same level is maintained during the entire period of hypoglycemia. Oxygen untake shows a rapid decrease during the first two hours from 9.00 volumes per cent to 2.76 volumes per cent.

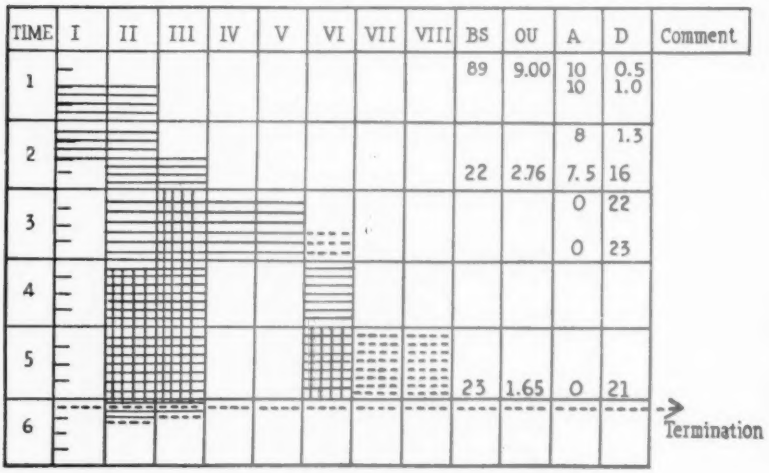


CHART 1.—Patient E. B. 9/12/38, dose of insulin 196 units.

At the end of the fifth hour a further decrease to 1.65 volumes per cent is observed.

The alpha frequency gradually diminishes till unconsciousness develops after which no alpha waves are recorded. The delta index augments rapidly during the first two hours, then the increase is slower. All these changes are reversed after the administration of sugar.

Summary.—The greatest decreases of blood sugar and oxygen uptake are recorded in the first two hours. Approximately at the time when consciousness is lost alpha waves disappear completely, the level of the blood sugar remains unchanged while a further diminution of oxygen uptake and increase of the delta index are recorded. The greatest changes in the clinical picture are associated with the alterations of consciousness and of the autonomic nervous system. Though the progress of the clinical symptoms is rapid, the patient enters into the terminal phase (VII) (VIII) only to a slight extent before the administration of sugar.

Chart 2, Patient M. K., 9/13/38, dose of insulin 175 units.

This "shock" shows a picture different from the previous one. Unconsciousness occurs later, after 2¾ hours (III). During the clouding of consciousness the patient becomes increasingly restless. Simultaneously with the loss of consciousness (III) various motor symptoms develop (primitive movements, forced grasping, myoclonoid twitchings, athetoid and choreiform movements) (IV.) Marked signs of sympathetic overactivity are observed at this time (V). Waves of increased muscle tonus appear repeatedly be-

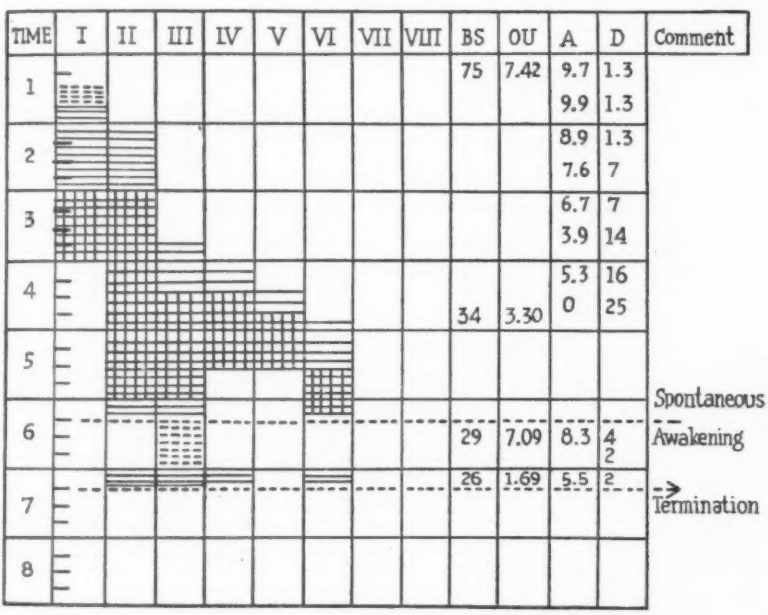


CHART 2.—Patient M. W., 9/13/38, dose of insulin 175 units.

ginning towards the end of the fourth hour (VI). By the end of the fifth hour they are the most prominent feature and at this time the sign of Babinski is elicited on the left foot. That the dose of insulin is too small to produce the greatest depth of coma in the postulated five hours is indicated by late appearance of unconsciousness, persistence of the symptoms of release of the basal ganglia and in the delay of the development of the tonic phase. For this reason sugar is not given despite the lapse of five hours. After 5¼ hours an important change is recorded. The patient commences to show signs of regression of the symptoms. The Babinski disappears, tonic phenomena give way to a state of hypotonia and consciousness returns (III) but only partially. Though the patient responds to ques-

tioning yet aphasia echolalia and perseveration are observed. This spontaneous awakening lasts for ¾ hour then consciousness is lost for the second time (III), subcortical motor symptoms reappear (IV), perspiration becomes profuse (II), while the Babinski and tonic spasms replace the few twitchings (VI). After 7¼ hours the "shock" is terminated by tube-feeding.

Blood sugar with a value of 75 mg. per cent reaches the level of 34 mg. per cent in the fourth hour, a level which is not changed during the spontaneous recovery. However, a parallelism in the slow development of the physiological and clinical changes is noted. While in the preceding observation the blood sugar of 22 mg. per cent was attained two hours after the injection of insulin, here the level of 34 mg. per cent is noted in the fourth hour.

The oxygen uptake shows the same slow decrease. The diminution of the oxygen utilization after four hours is 4.12 volumes per cent while on the preceding day the difference was 6.24 volumes per cent after two hours. Blood specimens taken while patient awakens spontaneously show an oxygen utilization of 7.09 volumes per cent which is almost equal to the initial one. Simultaneously, with the sudden deepening of the coma in the seventh hour, a speedy fall of oxygen utilization to 1.69 volumes per cent is observed, a fall which corresponds with the rapid progress of the clinical symptoms toward the midbrain phase.

A similar parallelism may be seen in the changes of the electrical activity of the brain for the early decrease of the frequency of the alpha waves is a slow one. These waves disappear entirely when unconsciousness develops. Meanwhile the delta index increases to 25 cm. Simultaneously with the spontaneous awakening a sudden rise in the frequency of alpha waves is perceived (8.3 per second) while the delta index decreases to 2 cm. The second and more sudden deepening of the "shock" is accompaneid by a fall of alpha frequency to 5.5 per second.

Summary.—The slow progress of the symptoms is accompanied by slow decrease of the blood sugar level, oxygen utilization and alpha frequency and similarly slow increase of the delta index. Regression of the symptoms during the spontaneous awakening is associated with an increase of the oxygen uptake, a rise of alpha frequency and a decrease of the delta index. However, no elevation of the blood sugar is recorded during the spontaneous awakening. The disappearance of the alpha waves occurs approximately at the time when consciousness is lost and basal ganglia phenomena appear.

Chart 3, Patient M. W., 9/14/38, dose of insulin 185 units.

Experiments of this day are devoted exclusively to a study of the correlation between consciousness and the electrical activity of the brain. The same patient examined previously is injected with a greater dose in order to hasten the progress of the symptoms and to prevent spontaneous awakening. Then small amounts of glucose are administered intravenously.

Cortical syndromes (I) and general symptoms of the autonomic nervous

system (II) develop in due time. At the end of the second hour consciousness (III) becomes clouded. Simultaneously with the loss of consciousness (III) violent motor symptoms develop as well as clonic twitchings, primitive movements, myoclonoid twitchings and other phenomena of release of the basal ganglia (IV). Sympathetic activity (V) also is marked with exophthalmus, acceleration of the pulse rate to 130 and dilated pupils (V). As the first tonic phenomena appear (VI) 2 grams of sugar are given intravenously. Immediate disappearance of all symptoms is observed with the one exception that consciousness is not regained (III). One half hour later perspiration and salivation (II) reappear and hyperkinetic phenomena are increased (IV). As tonic spasms reappear (VI) 4 grams of glucose are

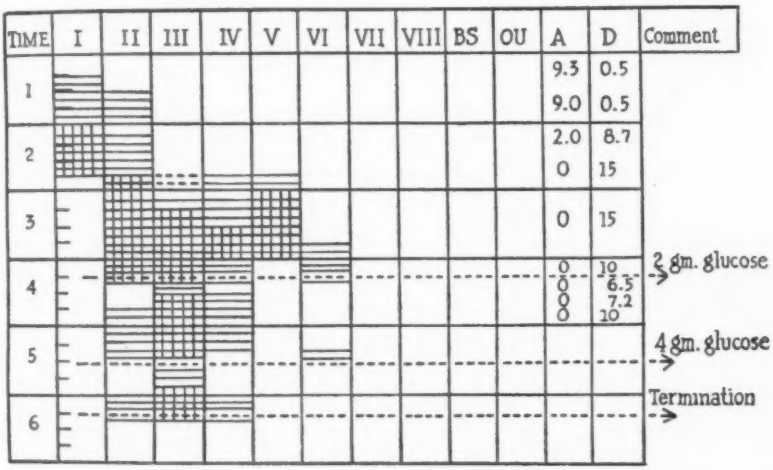


CHART 3.—Patient M. W., 9/14/38, dose of insulin 185 units.

given intravenously. The same reversal of signs is again reproduced as all symptoms with the exception of unconsciousness vanish. After half an hour the patient once more exhibits perspiration, salivation and motor symptoms; then the shock is terminated.

The decrease of the alpha waves is rapid. With the beginning of unconsciousness no alpha waves are recorded and the delta index rises. While injecting 2 grams of glucose, records are taken continuously but no return of alpha waves is seen. However, the delta index is decreased. During the injection of 4 grams of glucose external electrical disturbances prevent proper recording of electroencephalograms.

Summary.—With the exception of unconsciousness all symptoms disappear following the injection of 2 grams of glucose; nevertheless no return of alpha waves is recorded. The same clinical observations were made after the injection of 4 grams of glucose.

Chart 4, Patient M. W., 9/15/38, dose of insulin 195 units.

The next day the insulin dosage is again raised in order to study the more rapid production of the symptoms as well as the effects of larger amounts of sugar. The clonic twitchings and other motor symptoms due to release of basal ganglia (IV) develop more rapidly than on the previous day. Furthermore both motor and sympathetic symptoms (V) exhibit a greater intensity while consciousness (III) is depressed more profoundly. At the beginning of the third hour rapid myoclonoid twitchings become violent. After 2¾ hours a seizure occurs suddenly as twitchings became generalized. The patient turns pale, the pulse rate increases to 160 and

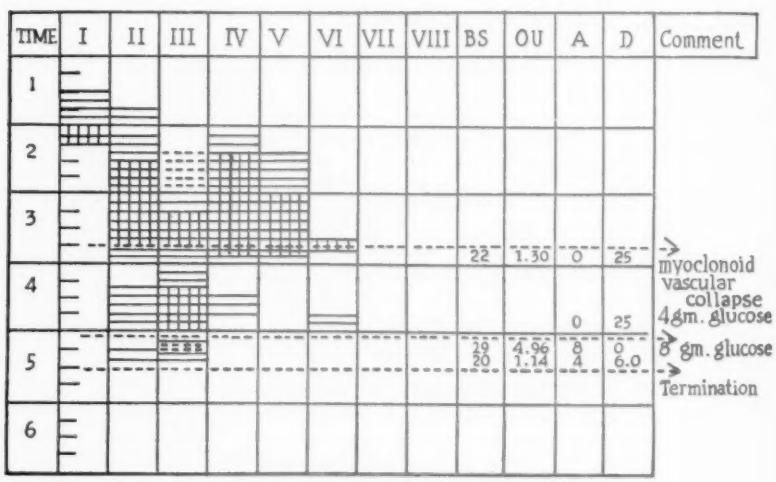


CHART 4.—Patient M. W., 9/15/38, dose of insulin 195 units.

becomes thready in quality. Tonic spasms with a tetanic posture of the hands terminate the fit.

Four grams of glucose are given intravenously and blood specimens taken. All symptoms with the exception of unconsciousness disappear (III). One half hour later perspiration, salivation (II) and basal ganglia symptoms (IV) reappear. As soon as tonic spasms (VI) are again observed 8 grams of glucose are given intravenously. Now all symptoms including unconsciousness disappear and contact is maintained for ½ hour. Then salivation, perspiration, motor symptoms and unconsciousness develop again. After 4½ hours the shock is terminated by tube-feeding.

Blood sugar is not significantly changed after the fit nor during the short period of contact. Immediately after the seizure the oxygen uptake shows the low value of 1.30 volumes per cent although 4 grams of glucose were given. In contrast after 8 grams of glucose the oxygen uptake rises to

4.96 volumes per cent while with the redevelopment of the symptoms a second fall to 1.14 volumes per cent is recorded.

The changes of the alpha frequency are well correlated with the biochemical and clinical observations. No alpha waves are present after the fit, and the delta index is high. After administration of 8 grams of sugar, however, return of alpha waves is seen with the frequency of 8 per second. With the subsequent deepening of the clinical symptoms the alpha frequency is diminished while delta index rises again.

Summary.—An increase of the insulin dose causes a more rapid progress of the symptoms than on the preceding day and evokes a seizure called myoclonoid vascular collapse (Frostig [2]).

The correlation between loss of consciousness and alpha waves is observed after the administration of 4 grams of glucose when neither consciousness nor alpha waves return and after administration of 8 grams when consciousness returns and alpha waves reappear. A parallelism is again noted between the oxygen uptake, electrical activity, clinical symptoms, but not with the blood sugar level.

DISCUSSION.

The present investigation has disclosed the simultaneous changes in the clinical symptoms, brain metabolism and cerebral electrical activity during hypoglycemia. The clinical changes make their appearance in a definite order beginning with the newer portions of the brain and descending through the various phylogenetic layers. Associated with these clinical signs is an ever-deepening depression of cerebral metabolism. Not only the oxygen consumption but also the variations of the electrical potentials indicate depression of cerebral activity. The administration of glucose which supplies the energy for brain metabolism reverses the electrical, biochemical and clinical changes.

A progression of symptoms similar to those occurring with hypoglycemia also result from anoxia though with the latter the rate of development of the changes is much increased. In this regard it is important to recall the results of Heymans and co-workers [21] who have shown that different portions of the brain exhibit a varying vulnerability to decreased supply of energy; acute anoxia affecting first the cerebral cortex, next the upper portion of the brain stem, and finally the medulla. By studies of electrical activity Sugar and Gerard [22] have also revealed that the various parts of the brain suffer a progressive depression from the cortex to the medulla during anoxia. Electrograms [23] disclose that during hypoglycemia

the activity of the cortex is depressed before that of the hypothalamus. Thus the progress of the symptoms during anoxia or hypoglycemia may be ascribed to a lesser sensitivity of the older regions of the brain.

A possible reason for the comparatively insensitivity of the older regions may be their smaller oxygen utilization.[24] It would seem that the older parts of the brain possess a smaller energy requirement and are, therefore, more resistent to anoxia or hypoglycemia than the younger parts with their higher metabolic rate. It is, therefore, understandable that, in general, the order of the symptoms moves in a direction which is opposite to the phylogenetic age of the cerebral layers.

A correlation is apparent between the cerebral disturbances of any given layer and the amount of carbohydrate available. Charts 3 and 4 disclose that appropriate doses of glucose may cause regression of the symptoms only to a certain point, for example to the basal ganglia stage but not to recovery of the cortical functions. It would appear that the supply of carbohydrate was too small to restore completely the metabolism of the parts with a higher metabolic requirement. It may, therefore, be concluded that the progression and regression of the symptoms moving in opposite directions through the layers of the brain depend upon the quantity of carbohydrate furnished to the brain. However, even when the amount of glucose injected is adequate to satisfy the energetic requirements of all parts of the brain, experiments on hypoglycemic animals reveal that the electrical activity of the hypothalamus is restored before that of the cortex.[23] A correlation exists between the rate of development of the clinical symptoms and that of the disappearance of the alpha frequency. Nevertheless this early correlation is no longer maintained when consciousness is lost for no alpha waves are recorded when sub-cortical release is complete. Assuming an error of 15 to 30 minutes because the clinical method of determining unconsciousness by loss of contact is inexact, the disappearance of the alpha waves is simultaneous with the development of unconsciousness. Furthermore, the administration of glucose causes the return of alpha waves only when consciousness is restored. In these experiments, therefore, the absence of alpha waves is associated with loss of function of the cortex. The delta index undergoes a gradual increase as consciousness becomes

clouded. Moreover, even after the alpha waves have disappeared the delta index continues to reveal a correlation with the metabolic and clinical changes. Similar relationships between the clinical symptoms and the alpha and delta waves have been reported by Gibbs, Gibbs, and Lennox [25] during natural sleep and after medication with various sedative drugs. Thus in the present investigation the simultaneous effects of insulin have been observed on blood sugar levels, cerebral oxygen uptake, electrical activity and the clinical symptomatology. As a result of an adequate dose of insulin blood sugar decreases and is maintained at a low level until termination of the hypoglycemia with sugar. Since the brain requires chiefly carbohydrate to maintain its function, the supply of carbohydrate to the brain becomes insufficient; it must, therefore, call upon its own meager carbohydrate stores. As these are gradually depleted [26] decreases of cerebral metabolism must follow, decreases which are evidenced by diminished oxygen utilization. The electrical activity of the brain and the development of the clinical symptoms are expressions of the decreased cerebral metabolism.

The sequence of events during hypoglycemia may therefore be considered as follows:

1. Hypoglycemia.
2. Decrease of the metabolism of the brain.
3. Changes of cerebral function.
   a. Electrical Activity.
   b. Clinical Symptoms.

SUMMARY AND CONCLUSIONS.

1. Simultaneous observations of clinical symptoms, cerebral oxygen utilization, blood sugar level and electrical activity of the brain were made on patients with schizophrenia receiving the insulin treatment.

2. Blood sugar decreases during the first hours after the injection of insulin and then remains at a low level until termination with sugar while the clinical signs continue to develop progressively throughout the entire course of hypoglycemia.

3. The march of the symptoms is correlated with a gradual decrease of cerebral oxygen uptake and the electrical changes.

4. The regression of the symptoms is accompanied by a gradual increase of the oxygen uptake and a reduction of the delta index. The sequence of the symptoms during regression is in an order which is the reverse of their appearance.

5. During hypoglycemia the alpha waves disappear approximately at the time when the functions of the cortex are completely suppressed and reappear with the restoration of cortical functions.

6. It is therefore, concluded that as a result of the hypoglycemia caused by the administration of insulin the metabolism of the brain is diminished. This reducton of cerebral metabolism is the cause of the clinical symptoms as well as the alterations of the cerebral electrical activity of the brain.

7. These conclusions concerning the correlations between the clinical and physiological changes are to be regarded only as a working hypothesis because of the small number of the experiments performed.

BIBLIOGRAPHY.

1. Angyal, L. v.: Über die motorischen und tonischen Erscheinungen des Insulin-shocks. Zeit. Neur., 157: 35, 1937.
2. Frostig, J. P. (Translated by Joseph Wortis): Sakel's pharmacologic shock treatment for schizophrenia. Arch. Neur. and Psychiat., 39: 219, 1938.
3. Frostig, J. P.: Experiences with the insulin therapy in schizophrenia with special reference to clinical problems. Am. J. Psychiat., 94: suppl., 220, 1938.
4. Cline, W. B. and Rossman, J. M.: (In preparation.)
5. Himwich, H. E. and Nahum, L. H.: Respiratory quotient of the brain. Proc. Soc. Exp. Biol. and Med., 26: 496, 1929 and Am. J. Physiol., 101: 446, 1932
6. Lennox, W. G.: Cerebral circulation. Arch. Neur. and Psychiat., 26: 719, 1931.
7. Goldfarb, W. and Himwich, H. E.: Ketone substance production and destruction in certain tissues of diabetic dogs. J. Biol. Chem., 101: 441, 1933.
8. Jowett, M. and Quastel, J. H.: Studies in fat metabolism, III. Biochem. J., 29: pt. 3, 2181, 1935.
9. Baker, Z., Fazekas, J. F., and Himwich, H. E.: Carbohydrate oxidation in normal and diabetic cerebral tissues. J. Biol. Chem., 125: 545, 1938.
10. Kerr, S. E. and Ghantus, M.: Carbohydrate metabolism of brain, II. J. Biol. Chem., 116: 9, 1936.

11. McGinty, D. A.: The regulation of respiration, XXV. Am. J. Physiol., 88: 312, 1929.
12. Dameshek, W., Myerson, A., and Stephenson, C.: The mechanism of the neurological symptoms in insulin hypoglycemia. Arch. Neur. and Psychiat., 33: 1, 1935.
13. Himwich, H. E. and Fazekas, J. F.: The effect of hypoglycemia on the metabolism of the brain. Endocrinology, 21: 800, 1937.
14. Himwich, H. E., Bowman, K. M., Wortis, J., and Fazekas, J. F.: Biochemical changes occurring in the cerebral blood during the insulin treatment of schizophrenia. J. Nerv. and Men. Dis., 89: 273, 1939.
15. Himwich, H. E., Hadidian, Z., Fazekas, J. F., and Hoagland, H.: Cerebral metabolism and electrical activity during insulin hypoglycemia in man. Am. J. Physiol., 125: 578, 1939.
16. Hoagland, H., Rubin, M. A., and Cameron, D. E.: The electroencephalogram of schizophrenics during insulin hypoglycemia and recovery. Am. J. Physiol., 120: 559, 1937.
17. Myerson, A., Halloran, R. D., and Hirsch, H. L.: Technic for obtaining blood from the internal jugular vein and the internal carotid artery. Arch. Neur. and Psychiat., 17: 807, 1927.
18. Van Slyke, D. D. and Neill, J. M.: The determination of gases in the blood and other solutions by vacuum extraction and manometric measurements. J. Biol. Chem., 51: 523, 1924.
19. Folin, O. and Wu, H.: A simplified and improved method for the determination of sugar. J. Biol. Chem., 41: 367, 1920. Suppl. 1.
20. Frostig, J. P., Persyko, J., and Persyko, H.: The quotient of blood sugar fall. A contribution to the question of insulin sensitization. Am. J. Psychiat., 94: suppl. 226, 1938.
21. Heymans, C., Bouckaert, J. J., Jourdan, F., Nowak, S. J. G., and Farber S.: Survival and revival of nerve centers following acute anemia. Arch. Neur. and Psychiat., 38: 304, 1937.
22. Sugar, O. and Gerard, R. W.: Anoxia and brain potentials. J. Neurophysiol., 1: 558, 1938.
23. Hoagland, H., Himwich, H. E., Campbell, E., Fazekas, J. F., and Hadidian, Z.: Effects of hypoglycemia and pentobarbital sodium on the electrical activity of the dog cerebral cortex and hypothalamus. J. Neurophysiol., 2: 276, 1939.
24. Dixon, T. F. and Meyer, A.: Respiration of the brain. Biochem. J., 30: 1577, 1936.
25. Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: Effect on the electroencephalogram of certain drugs which influence nervous activity. Arch. Int. Med., 60: 154, 1937.
26. Kerr, S. E., Hampel, C. W., and Ghantus, M.: The carbohydrate metabolism of brain, IV. J. Biol. Chem., 119: 405, 1937.

THE SIGNIFICANCE OF THE BABINSKI SIGN DURING HYPOGLYCEMIC TREATMENT OF SCHIZOPHRENIA.*

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Various phenomena are at our disposal to indicate the different stages of hypoglycemia induced by insulin during the treatment of schizophrenia. The delineation of a conscious and an unconscious stage, and then the demarcation of different phases within these, especially the latter, are most important questions. There is little controversy as to the characteristics of the onset of unconsciousness in which muscular clonus, twitchings, involuntary eye-movements or a fixed gaze are important symptoms. The period prior to the onset of unconsciousness is described by the terms sleepy, dizzy, drowsy or semiconscious. In addition we note the sympathetic manifestations and the various motor reactions which round out the syndrome of this period. The conditions, however, are quite different after the patient becomes unconscious as the signs available are more limited and the appearance or disappearance of various reflexes become valuable guides. Respiratory and cardiovascular symptoms are of significance chiefly to avoid pitfalls in treatment, and as indications for termination. The sympathetic symptoms such as sweating or variation in the size of pupils do not aid in understanding or defining the unconscious stage of hypoglycemia. The reflexes alone and possibly the state of muscular tonus have to be the basis for such an investigation.

Considering how widespread the insulin treatment of schizophrenia now is, the lack of a generally accepted scheme for determining the depth and duration of the coma looms large. It is not expected that each patient be treated according to some rigid scheme, but only that a basic objective plan be formulated which could be applied to the special needs of the individual patient. Just as in fever therapy, where the temperature is raised to a certain point and maintained for a definite time, in accordance

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with the dictates of experience, so in the insulin treatment one should induce a certain depth of coma in the patient and maintain this state for a given time. The scale in this case is the patient, and the movable mercury, the appearing or disappearing symptoms. Frostig was the first who devised principles of general validity for determining the landmarks of the coma, following the Viennese school. Subsequent workers modified these rules according to their own respective experiences.

It is a matter of general opinion that the appearance of the Babinski sign is significant for one of the deeper stages of coma. We consider a patient in coma upon the onset of unconsciousness as defined above. Gillman and Parfitt [1] state, "The criteria of coma are usually taken as absent corneal reflexes and extensor plantar reflexes." Since the corneal reflexes are lost only in the last phase of coma, the Babinski sign is evidently placed by these authors as a relatively late appearing reflex. According to Murray Rossman and William Cline [2] who divide the coma into three stages of light coma (out of contact but retention of superficial reflexes); moderate coma (loss of superficial reflexes, positive Babinski and Hoffman with sluggish corneal reflexes); and deep coma (loss of corneal reflexes), the Babinski phenomenon is also an indicator for a deeper coma and is taken to appear some time after the loss of consciousness. Goldman suggests the use of a clinical chart during insulin treatment in which the positive Babinski is noted as appearing after the onset of unconsciousness, during a period he designated as the third stage of hypoglycemia. Von Angyal [3] believes the Babinski is evidence of the subcortical phase of activity. Frostig [4] on the other hand discusses various symptoms which indicate the progressive depth of coma, and places the pathological reflexes at the head of these. Paul Hoch [5] in discussing the extreme

[1] Gillman, S. W., Parfitt, D. N.: The technique and complications of insulin therapy. J. Mental Science, 84: Sept. 1938.

[2] Rossman, M. and Cline, Wm.: The pharmacological "shock" treatment of chronic schizophrenia. Am. J. Psychiat., May 1938.

[3] Von Angyal, L.: Zeitschr. f. d. ges. Neur. u. Psych., 157: 35, 1937.

[4] Frostig, J. P.: Sakel's pharmacological shock treatment for schizophrenia, tentative directions and system of recording. Arch. Neur. and Psych., 39: 220-231, Feb. 1938.

[5] Hoch, Paul: Neurological observations in hypoglycemic states. The Psychiatric Quarterly, 12: 690-698, Oct. 1938.

variability in the appearance of reflexes mentions the possibility that the Babinski may sometimes occur in the somnolent stage or "before the skin and deep reflexes disappear, even before the patient is in coma."

The purpose of our investigation was to determine the relation between the appearance of the pyramidal signs and of the loss of the corneal reflexes, and their relative value as indicators for stages of coma. The present report is concerned with our findings as to the pyramidal signs and is the first in a series of reports on our observations during insulin treatment.

About 900 tests on 28 patients were performed during a period of at least five weeks for each patient. These patients were in various stages of their course in insulin treatment, from those just beginning to those ready to be discontinued.

We started to investigate the plantar reflexes during the unconscious stage, and found an extraordinary variability in the manifestations of this reflex similar to the findings of other authors. We obtained the response by lightly stroking a pin across the outer border of the sole. The types of responses observed ranged from the normal physiological plantar reflex to the dumb-sole; from the dumb-sole to fanning of the toes only; from fanning to a positive Babinski with fanning of the toes; and finally the classical Babinski.

In compiling our results the classical Babinski was considered positive, the doubtful or merely suggestive extensor responses as questionable even though accompanied by other suggestive pathological toe signs, and all other responses negative.

In 568 tests during the unconscious stage we found a positive response 453 times or 92.3 per cent; the response was negative 38 times or 7.7 per cent; and a questionable response 77 times, this latter figure being omitted from our calculations. That the positive Babinski occurs in most cases during the unconscious stage is clear from these results, but we could not confirm the appearance of the Babinski in relation to a definite time or depth of the coma. Almost invariably, with the onset of unconsciousness the Babinski was found already present. We rarely found its disappearance in the later stages.

It was evident, then, that appearance of the Babinski phenomenon does not have any significance for the depth of the coma and

is not to be depended upon for the classification of stages of hypoglycemia. On the other hand, this result compelled us to investigate the actual time of appearance of the Babinski reflex.

We therefore examined the same patients during the period preceding unconsciousness, when they were either semiconscious, drowsy, sleepy or completely conscious. In 316 tests we found a positive response 167 times, or 74.2 per cent; a negative response 58 times or 25.8 per cent; and a questionable response 91 times. The figure of 74.2 per cent positive responses would be somewhat increased were we to disregard those patients in our series who did not show the development of the positive Babinski at any time whatsoever during any of the stages of hypoglycemia. This group includes 10 per cent of all the patients. We saw the pathological toe sign in some patients who, just beginning their course of treatment and receiving minimal doses of insulin, did not exceed a stage of sleepiness. A positive toe sign appeared usually in the second hour after the insulin injection. In other words, one hour or more prior to the onset of unconsciousness the appearance of a positive Babinski is a rather common occurrence. These findings, of course, are related to our method of administering the insulin treatment in which we strive to induce the state of unconsciousness by the third hour after the injection.

CONCLUSION.

According to this investigation it is evident that the Babinski sign does not belong to a definite state of unconsciousness nor is it in any way exclusively connected with unconsciousness. In view of its frequent appearance during the conscious state the Babinski sign can no longer be utilized for the classification of various stages of the insulin coma.

In our experience on the other hand the loss of the corneal reflex is a very reliable symptom indeed for a deeper or so-called second stage of coma. We suggest the following scheme for the classification of the hypoglycemia:

Stage I. From injection to onset of unconsciousness.

Stage II. From the appearance of unconsciousness until the loss of corneal reflexes.

Stage III. From the loss of corneal reflexes until termination.

Unconsciousness should appear in the third hour after injection and the loss of corneal reflexes in the fourth hour.

Angyal, Frostig and others take it for granted that simultaneously with the appearance of the subcortical phase of the coma signs of pyramidal tract disturbances are manifested. It is likely and more in accordance with brain physiology—as is verified by the results of this research—that the Babinski sign appears concomitantly with the involvement of the cortex. Thus we should not be surprised to observe the presence of the toe sign simultaneously with, or usually even before the appearance of other motor symptoms on the onset of unconsciousness.

SUMMARY.

1. A positive Babinski was found in 92.3 per cent of cases after the onset of unconsciousness and in 74.2 per cent prior to the onset of unconsciousness in patients undergoing insulin treatment for schizophrenia.

2. The positive Babinski has no significance in determining a stage of coma during the insulin hypoglycemia.

3. The loss of corneal reflexes is suggested as a reliable sign for determining a deeper stage of coma.

[Dr. Max Sherman gave assistance in reading records and correcting the manuscript.]

FATAL COMPLICATION FOLLOWING METRAZOL TREATMENT FOR SCHIZOPHRENIA.*

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INTRODUCTION.

Metrazol therapy appears not to be as harmless as the earlier reports indicated. Aside from the relatively minor complications such as fractures, subluxations and dislocations of various bones and joints, fatal complications associated with metrazol therapy have been reported. Angyal and Gyarfas [1] reported one death which occurred one-half hour after the second injection of metrazol (7 cc.). Autopsy revealed aortic insufficiency and myocarditis. Biner [2] reported two deaths, one occurred four hours after the fourth injection and the other the night after an induced convulsion. Post mortem examination revealed bilateral hypernephroma and a goitre in one patient, and a pulmonary embolism supposedly from an old pelvic thrombophlebitis in the other. Hayman and Brody [3] reported the death of a 24-year-old male patient after the sixth injection, and at post mortem examination chronic endocarditis was recognized. The organic pathology found at autopsy in the above cases was not detected before metrazol treatment was instituted. Four deaths resulting from sequellæ or complications during the course of metrazol treatment, but not directly due to the treatment per se, have been reported by Ross.[4] From an analysis of more than 3000 cases V. Meduna and Friedman conclude that the mortality rate associated with metrazol (and camphor) therapy is 0.29 per cent.

CASE REPORT.

S. H., a 24-year-old farmer, was brought to the Wisconsin Psychiatric Institute by his father who stated that the patient had been acting queerly for the past four months. Following the departure of a 22-year-old brother to whom the patient had a strong emotional attachment, he became inac-

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Ill., May 8-12, 1939.

tive, associated less than previously with other people, and expressed "peculiar ideas." He spent much time day dreaming. Past history revealed that the patient was considered seclusive and "fidgety" by his school companions. Medical history indicated that the patient had measles and mumps in childhood. He had a wheezing and prolonged inspiration which occurred at irregular intervals and was associated with a strange mannerism during which the thyroid cartilage moved up and down. The father said that the patient had the peculiar respiration and mannerism associated with it "all his life."

Physical examination on admission showed a slight asymmetry of the chest with prominence at the left costo-sternal junction. He presented the peculiar behavior associated with respiration which was mentioned above. It was noticeable particularly during interview or when the patient appeared embarrassed. In view of his history, the peculiar breathing was interpreted as a habit. The blood pressure was 120 systolic and 70 diastolic. Blood count and urine analysis were normal. The blood and spinal fluid Wassermann were negative. The blood sugar was 88 and non protein nitrogen 28 milligrams per 100 cc. A lumbar puncture performed on October 29, 1938 showed an initial pressure of 80 mm. of water. The pressure rose to 300 mm. of water on jugular compression and fell promptly on release of pressure. The spinal fluid was normal.

Mental examination revealed a shy young man who was reticent to talk. He said that he was "all run down." He expressed the belief that the local bandmaster was trying to "slip something over" on him. He thought that people in the adjacent town were "making fun of me and were trying to play tricks on me." The patient stated that while helping fill silo a few weeks before admission he overhead a couple of boys say that they were "out to get some one" and the patient believed that they referred to him. He also believed that a policeman was looking for him. He was listless, spent most of the time in bed, and appeared engrossed in his own thoughts. A diagnosis of schizophrenia, paranoid type, was made and in view of the normal physical findings metrazol treatment was recommended.

On October 31, 1938 the patient was given, by speed injection, 5 cc. of metrazol (10 per cent aqueous solution) intravenously. The blood pressure, pulse, and respiration immediately preceding the injection of metrazol were 115/70, 64/min. and 18/min., respectively. After a latent period of 31 seconds, the patient had a generalized convulsion which lasted 55 seconds. Normal respiration did not appear at the end of the clonic phase and he was immediately given artificial respiration by costal pressure, and an oxygen bag was applied. Despite all efforts such as the administration of adrenalin, caffeine with sodium benzoate, coramine, the continued application of artificial respiration with excessive oxygen and finally the injection of adrenalin intracardially, the patient could not be revived. Autopsy was performed by Dr. John McCarter of the department of pathology of the University of Wisconsin, two hours after death.

Gross examination showed acute cardiac dilatation and congestion of the spleen, liver and kidneys. The thymus gland was not enlarged, and there

was no hypertrophy of lymphoid tissue. This would exclude the possibility of a status thymolymphaticus. The brain grossly was edematous and there was present herniation of the cerebellar tonsils and the medulla into the foramen magnum. After removal of the brain, examination showed a well-marked pressure collar extending about the cerebellar tonsils and medulla. Microscopic examination showed generalized brain congestion, edema and swelling or shrinkage of nerve cells particularly in the corpus striatum, thalamus and cerebellum. Cloudy swelling of nerve cells particularly of the hypoglossal and dorsal vagal nuclei was seen in the medulla. Minute perivascular haemorrhages were found especially in the medial part of the thalamus.

DISCUSSION.

The cause of death is not entirely clear although several explanations suggest themselves. (1) It is possible that the lumbar puncture performed 48 hours before the metrazol injection may have played an important part in the patient's death. The supposition is advanced that the lumbar puncture disturbed the cerebro-spinal dynamics and that this contributed to the herniation of the cerebellum and the medulla into the foramen magnum, producing pressure paralysis of the vital centers in the medulla and thereby causing death. (2) The fact that the patient had peculiar respiration suggests a possible defect in the respiratory mechanism. However, we were unable to detect any pathology at autopsy to account for his peculiar breathing. (3) Acute cardiac dilatation may have caused death. (4) A fourth possibility is that any severe convulsion regardless of its etiology may be a potential cause of death. This supposition is advanced because two patients in whom a diagnosis of idiopathic epilepsy was made died following a severe convulsion. The autopsies showed a well marked pressure collar in both cases.

Our experience and a survey of the literature suggest certain precautions in the administration of metrazol therapy. In addition to a complete physical examination, studies of the urine and blood, a diligent search should be made for existing pathology in the cardio-respiratory and skeletal systems. Hence x-ray of the chest, back and of the long bones, and electrocardiograms should be performed on all patients before treatment. There should be a long interval, perhaps one week, after lumbar puncture and the institution of treatment. We refuse to administer metrazol therapy to patients showing evidence of respiratory complications, cardiac disease either valvular or muscular, or bone and joint disease.

BIBLIOGRAPHY.

1. Von Angyal and Gyarfas, K.: Ueber die Kardiazolkrampfbehandlung der Schizophrenia. Arch. F. Psychiat., 106: 1, 1936.
2. Biner, O.: Results of the convulsion therapy in the psychiatric clinic in Berne. Am. J. Psychiat., 94: 167-172, May 1938 Supplement.
3. Hayman, M., and Brody, M. W.: Metrazol therapy in schizophrenia. J. A. M. A., 112: 310-311, Jan. 28, 1939.
4. Ross, J. R.: The pharmacological shock treatment of schizophrenia. Am. J. Psychiat., 95: 769-779, Jan. 1939.
5. Von Meduna L. and Friedman, E.: The convulsive-irritative therapy of the psychoses. J. A. M. A., 112: 501-509, Feb. 11, 1939.

CENTRAL NERVOUS SYSTEM CHANGES PRODUCED BY INSULIN.*

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The question of damage to the central nervous system of experimental animals from the daily injection of convulsive doses of insulin has been studied by several investigators.[1, 2, 3, 4] The use of large doses of insulin for treatment of schizophrenia has made this question of clinical importance. This work represents a further inquiry into the problem.

METHOD.

Cats which had fasted for 16 hours were injected intramuscularly with insulin in amounts sufficient to produce insulin shock. The dose of insulin usually averaged six units; one cat (B) was given a single dose of 60 units of insulin. As a rule the animals went into shock and developed convulsions within three hours. At the onset of convulsions the animals were given 25 per cent glucose solution either by stomach tube or intraperitoneally.

The experiments were conducted daily, Sundays excepted, over a period from 9 to 28 days. Each cat averaged one or more severe convulsions each treatment day. The diet of the animals consisted of milk, liver and commercial canned cat food.

The only animals included in this study are those which were purposely sacrificed at an elected time by means of carotid section. Their brains were placed immediately in 10 per cent formol solution. All other cats are excluded from this study limiting our series to nine animals. Histological studies were made by the following techniques: toluidine blue, Weil's myelin sheath stain and stains for fat, microglia, oligiodendroglia and astrocytes.

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

From the Institute of the Pennsylvania Hospital.

Aided by a grant from the John and Mary R. Markle Foundation.

RESULTS.

Two animals show no changes anywhere in the central nervous system. Five show scattered ischemic cell changes involving particularly the cells in the frontal areas. These changes are present in patches of cortex here and there and involve all the cell layers indiscriminately. They have a slightly greater predilection for the upper layers. The Purkinje cells of the cerebellum seem particularly susceptible to damage in all the affected cases. There are no striking changes in the neuroglia nor in the myelin architecture.

The detailed reports on these animals follow:

CAT 1 (EXP. 37). MILD ISCHEMIA OF GANGLION CELLS.

The meninges are normal. The cortical architecture is well preserved. In some of the cortical cells of the frontal region there is definitely paler staining. Apart from this there are no changes in the brain or brain stem.

CAT 2 (EXP. 29). GENERALIZED ISCHEMIA OF CORTICAL CELLS.

The meninges are clean and show no thickening or evidence of cellular infiltration. The cortical architecture is well preserved but the cells show diffuse changes which are present in all parts of the cortex and all the cortical layers are involved. A number of changes appear in the cells: (1) Some areas show typical ischemic changes in the ganglion cells. These cells are small, shrunken and have small dark nuclei and tortuous dendrites. (2) Other areas, and this is the most commonly seen change, show a pallor of the cytoplasm, with loss of the nuclear membrane and an indistinct nucleus. These cells are somewhat swollen. (3) Scattered cells are present with shrunken, small, pyknotic nuclei which stain a uniform intense blue color in which the cytoplasm is vacuolated. There is no evidence of change in the glial cells either in the cortex or white matter.

CAT 3 (EXP. 31). NORMAL.

No changes are seen anywhere in the nervous system. The cortex, basal ganglia, hypothalamus, medulla, pons and cerebellum show no changes in the cells, vessels, or meninges.

CAT 4 (EXP. 42). MILD ISCHEMIA OF GANGLION CELLS.

The meninges are clean. The cortex shows scattered cells which take a dark stain. These are shrunken, pyknotic, and in a few instances have begun to undergo neuronophagia. There are also scattered changes in the Purkinje cells.

FIG. 2.—Swollen, plump ganglion cells in cerebral cortex. Cat 2. Toluidine blue stain.

FIG. 1.—Ischemic changes in the ganglion cells of the upper cortical layers. Note the darkened, thin cells. Cat 2. Toluidine blue stain.

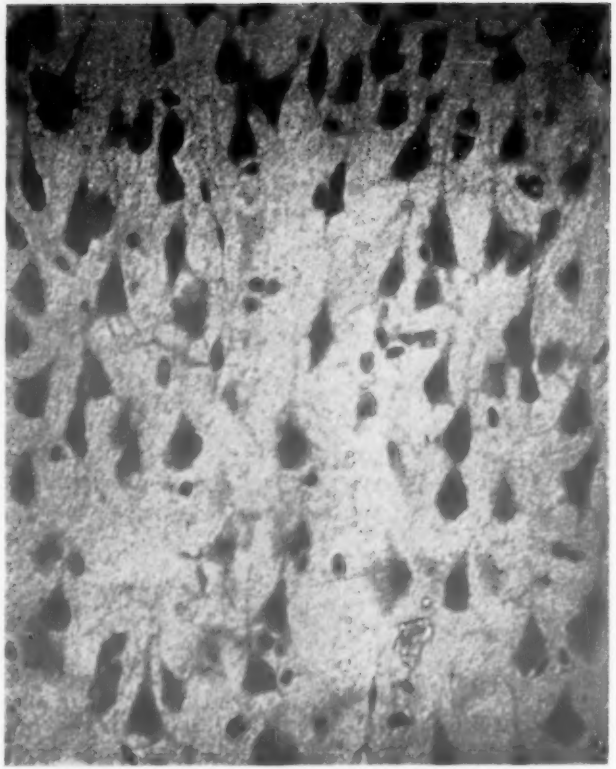


FIG. 3.—Ischemic cell changes in cerebral cortex. Cat 9. Toluidine blue stain.

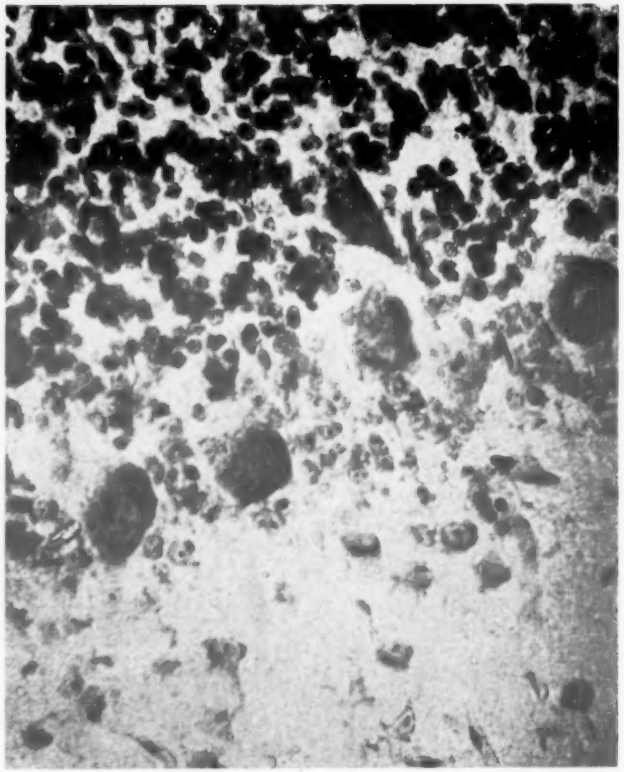


FIG. 4.—Diffuse changes in Purkinje cell layer. Some cells are pyknotic; one cell is almost completely gone. Cat 8. Toluidine blue stain.

Cat 5 (Exp. 43). Scattered Ischemia of Ganglion Cells.

The meninges are normal as is the cortical architecture. The ganglion cells throughout the cortex stain quite pale. Here and there, especially in the frontal cortex are cells which are shrunken, distorted, and stain uniformly dark. The nucleus of these cells, is small and pyknotic. These represent end-stages of the ischemic process, but apart from the pallor of the other cells there is no evidence of ischemia elsewhere. The Purkinje cells show uniformly a pyknosis of the nucleus and cytoplasm and irregular, distorted cellular outlines. There is no fat in the cells. The ganglion cells of the basal ganglia, hypothalamus, pons, and medulla are normal.

Cat 6 (Exp. 32). Normal.

No changes are seen anywhere in the nervous system. The cortex, basal ganglia, hypothalamus, medulla, pons and cerebellum show no changes in the cells, vessels or meninges.

Cat 7 (Exp. 41). Moderate Ischema of Ganglion Cells.

No changes are shown in the meninges or cortical architecture. Scattered everywhere through the cortex are cells which have small dark nuclei, and shrunken, dark, pyknotic cytoplasm characteristic of the end-stages of the ischemic process. Most of the diseased cells lie in the third ganglion cell layer. In some places whole sectors show changes of this sort. The cortex is for the most part healthy except for these scattered changes. The Purkinje cells are shrunken, dark and distorted, being more affected than the cortical cells. The Nissl substance in many of these cells is pulverized. The white matter in the frontal cortex shows beginning destruction of the myelin.

Cat 8 (Exp. 33). Scattered Ischemia of Ganglion Cells.

The cortex shows normal architecture. There are scattered cells throughout the cortex, but especially in the frontal areas with severe ischemic changes. These are shrunken, distorted and stain heavily; both the cytoplasm and nucleus are pyknotic. However, such cells contain no fat. Not all the purkinje cells are affected, but most of them are diseased as is apparent by their marked distortion, pyknosis and shrinkage.

Cat 9 (Exp. B). Generalized Ischemia of Cells of Cortex.

The meninges, showing no evidence of infiltration are clean and smooth. The cortical architecture is normal and there is no evidence of disarrangement. In the frontal cortical fields most of the cells show degenerative changes. These involve practically all cells, but they seem to be more marked in the lower cortical layers. These changes are less pronounced in the parietal and occipital cortex and are present to a mild degree in the medulla. They involve the purkinje cells in the cerebellum, but not the nuclei of the hypothalamic region.

TABLE 1.

Cat no.	Days of treatment.	Units of insulin. Max.	Units of insulin. Min.	Total hours of insulin effect.	Number of convulsions.	Examined days after last treatment.	Histological findings.
1 (37)	9	8	3	21	28	Directly	Mild ischemia of ganglion cells.
2 (29)	11	10	5	26	20	Directly	Moderate ischemia of ganglion cells.
3 (31)	12	8	4	35	25	2 days	Normal.
4 (42)	19	5	1	37	35	2 days	Mild ischemia of ganglion cells.
5 (43)	19	8	2.5	42	21	Directly	Scattered ischemia of ganglion cells.
6 (32)	21	5	0.5	37	56	5 days	Normal.
7 (41)	20	8	1	41	28	25 days	Mild ischemia of ganglion cells.
8 (33)	28	8	0.75	65	56	15 days	Scattered ischemia of ganglion cells.
9 (B)	1	60		6.5	Continuous	After 6.5 hrs.	Generalized ischemia of cortical cells.

The changes observed in the ganglion cells consist of a shrinkage of the nucleus which stains a uniform dark blue with toluidin blue. The cytoplasm is somewhat ballooned out in some cells, and shrunken in others. Usually it takes a lighter stain than normal, but in some cells it stains darker and is quite pyknotic. The Nissl substance is not seen nor is fat apparent either within the cells or vessel walls. The dendrites are abnormally visible in many cells.

In the medulla the cells of the nucleus ambiguous are swollen and pale. All these cell changes are of the ischemic type. The blood vessels are mildly hyperemic, but are otherwise normal.

The days of treatment, maximal and minimal dosage of insulin, hours of insulin effect, number of convulsion and time of examination as well as the histological findings are summarized in Table 1.

DISCUSSION.

Our findings on this group of animals are restricted to histopathological changes of the ischemic type. They are in agreement with the observations of Schmid [3] and of Weil and his associates.[4] They are not as severe as those noted by Tani [2] nor those reported by Steif and Tokay.[1] Variations in technique are sufficient to explain this difference.

The mechanism by which these ischemic changes are produced is conjectural. A possible explanation which is also subscribed to by Weil and his co-workers [4] is that it results from an inability of the brain to carry on its oxidative processes because of the action of insulin. In support of this are the experiments of Holmes,[5] Dameshek, Myerson and Stephenson [6] and Wortis [7] showing that insulin reduces the oxygen utilization of the brain.

BIBLIOGRAPHY.

1. Stief, A., and Tokay, L.: Ztschr. f. d. ges. Neurol. u. Psychiat., 153: 561, 1935.
2. Tani, N.: Zentralbl. f. d. ges. Neurol. u. Psychiat., 80: 30, 1936.
3. Schmid, M. H.: Ann. méd-psychol., 94: 658, 1936.
4. Weil, A., Liebert, E., and Heilbrunn, G.: Arch. Neurol. and Psychiat., 39: 467, 1938.
5. Holmes, E. C.: Biochem., J., 24, 914, 1930.
6. Dameshek, W., Myerson, A., and Stephenson, C.: Arch. Neurol. and Psychiat., 33: 1, 1935.
7. Wortis, S. B.: Arch. Neurol. and Psychiat., 33: 1022, 1935.

MENTAL DISEASE IN PERU.*

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The care and treatment of the mentally ill in Peru have passed through a series of progressive stages similar to those undergone in the development of psychiatric service in the United States, Canada and various European countries. A long period of neglect was succeeded by inadequate almshouse or asylum care, which in turn was replaced by so-called hospital care with some degree of medical attention. With the general advance in psychiatry came the latest stage which embraces scientific treatment in a modern hospital with a competent staff of psychiatrists, nurses and attendants.

The first hospital provision for the care of mental patients in Peru was made in two general hospitals of Lima: the one, St. Andrew's Hospital for men; the other, St. Anna's Hospital for women. In each of these hospitals a special section was set apart for the care of mental patients. These sections were called "loquerias." The first mention of these was made by Father Juan Meléndez in 1678. He wrote concerning the "loqueria" in St. Andrew's Hospital which was used for the violent insane. He mentions one of the unfortunate disturbed patients who was put in a cage with his hands tied and was cared for by a charitable woman who cleaned the cage and gave him food and drink. Later in St. Peter's Hospital the violent insane were separated from the quiet cases. The former were chained while the latter were simply locked in cells. In the early part of the 19th century insane persons were less cruelly treated. They were kept in clean cells, bathed and given a certain degree of individual attention.

Toward the middle of the century the "loqueria" of St. Andrew's Hospital became in part an independent unit. It had its own budget and was called "Hospital Para Insanos." A special physician was provided to care for the mental patients. In 1857, the public

* The writer is indebted to Dr. Honorio Delgado, a leading psychiatrist of Lima, Peru, for many of the historical facts set forth in this article.

welfare society * of Lima, which had charge of the hospital, appointed for this service Dr. José Casimiro Ulloa. Ulloa was an idealist. Upon investigation he found the "loquerias" of St. Andrew's and St. Anna's in bad condition and utterly inadequate for their purpose. He formed plans for needed improvements and sought aid in support of his projects. Fortunately, he was able to secure the active cooperation of the director of welfare, Don Francisco Carassa. With determination and persistence, the latter undertook the raising of funds for the erection of a suitable hospital for the patients who were unsatisfactorily cared for in the "loquerias." Success attended his efforts and eventually a large building was erected in the outskirts of Lima and was named "Hospital de la Misericordia." The new institution was opened December 16, 1859. It was considered a good asylum at the time. It had accommodations for 160 patients and soon after opening was caring for 153.

Naturally the new hospital used the means of treatment which prevailed at the time. These included surprise baths, baths for the disturbed similar to those used in the Salpêtrière, restraint chairs made in Paris, and wooden cribs or cages. There were also a variety of restraint apparatus, including iron bands for the feet and arms, to which chains were attached and fastened to the wall, strait jackets and barred cells. Many of these devices, silent witnesses of the ignorant cruelty of a bygone period, are still to be seen in the museum room of the Hospital Victor Larco Herrera in Lima.

The Hospital de la Misericordia was divided into four sections, the first for quiet cases; the second for those periodically disturbed; the third for mental defectives, epileptics and untidy cases; and the fourth for violent insane. This last section contained three strong cells such as were used in prisons.

Through the influence of Carassa and Ulloa the treatment of patients gradually improved. The policy of repression and restraint was modified and various activities were introduced to interest the patients and to provide an outlet for their energies.

Dr. Ulloa died in 1891 and was succeeded by Dr. Manuel A. Muniz, a physician who had studied in France and had observed the care of mental patients in other European countries. Muniz

* Sociedad de Beneficencia Publica de Lima.

insisted on the use of gentle measures in caring for the insane and abolished all kinds of punishment. Although the original hospital had been enlarged, it had become crowded and antiquated. Dr. Muniz made plans for the erection of a new institution but died before his plans could be realized. He was succeeded by Dr. Matto, who was sent to Europe to study the organization of asylums for the insane. A site for the new institution was selected at Magdalena del Mar, about four miles from Lima. The building of the new hospital, however, was delayed, and it was not opened until 1918. Dr. Matto having died, Dr. Hermilio Valdizan was appointed resident physician and afterwards director of the hospital. Dr. Valdizan, who was a man of exceptional ability, became a leader in the advancement of psychiatric teaching and practice in his country.

The new hospital was made possible by a generous gift from Victor Larco Herrera, a philanthropist of Lima. Under the direction of Dr. Valdizan the institution made rapid progress and became a treatment hospital instead of a custodial asylum. Following the death of Dr. Valdizan in December, 1929, Dr. Baltazar Caravedo was appointed director of the hospital. He has continued the progressive policies of his predecessor.

On March 27, 1939, following the Pan American Neuropsychiatric Conference at Lima, Dr. Caravedo entertained the conference delegates at the hospital and showed them its facilities and activities. As it is the only hospital of its kind in Peru, it represents the standards of psychiatric service now in use in that country.

The Hospital Victor Larco Herrera.

Bearing the name of its benefactor, Victor Larco Herrera, the Peruvian hospital for mental disease is located at Magdalena del Mar, a suburb of Lima, beautifully situated about four miles from the heart of the city. The year following its opening in 1918, the hospital cared for 817 patients. Since that time the number has increased from year to year until on the day of our visit, it had reached 1315.

The hospital is built on the cottage plan. There are nine cottages for continued treatment cases and a reception cottage. Both private and public patients are cared for. Male private patients occupy

one cottage and female private patients occupy two. The dependent patients occupy six cottages, three being devoted to the care of each sex. The reception building accommodates both males and females. In addition to the buildings mentioned, there is a small cottage for patients with infectious diseases; a cottage for the care of children of both sexes, most of whom are feebleminded; a cottage for physiotherapy; a mortuary; and an administration building. Separate homes are provided for the superintendent and steward. There is a nurses' home which also contains a sewing-room for the making of patients' clothing. No accommodations are provided for employees not engaged in ward work. A new cottage, which is nearly ready for occupancy, will care for the criminal insane.

In addition to the buildings mentioned, there are a well-equipped kitchen, a laundry, a bakery and various small buildings used for shops. Most of the cottages are one-story buildings. Some of those recently built, however, have two stories. The cottages as a whole constitute an attractive group but it is evident that strict economy was exercised in their construction. The kitchen is especially well adapted for its purpose. It is equipped with electric ranges and cookers which are said to give excellent service.

As the weather is warm throughout the year, no provision is made for heating any of the buildings.

The institution is well supplied with physicians, nurses and other employees. Including the superintendent, there are 12 physicians on the staff. Four of these are resident physicians. The others serve on part-time or are subject to call as their services are needed. One physician has charge of the laboratory and another of the physiotherapy cottage. A pharmacist and a part-time dentist are employed. The nursing force consists of the superintendent of nurses, two assistants to the superintendent, 58 ward nurses and 51 student nurses. Of the students, 32 are in the first year class, 11 in the second year class and eight in the third year class. In addition to the employees mentioned there are 148 others who are engaged in conducting the general work of the institution. Counting pupil nurses as employees, the ratio of all employees to patients is 1 to 4; excluding pupil nurses, the ratio becomes 1 to 4.8.

The salaries and wages paid employees are very small compared to those prevalent in the United States. The money unit is the

sol, which is worth about 20 cents in United States money. The superintendent receives an annual salary of 7200 soles. The physicians receive salaries ranging from 1200 to 3000 soles. The salary of the superintendent of nurses is 3600 soles and that of each assistant to the superintendent, 2400. The salaries of other nurses run from 1080 to 1800 soles each. Physicians who lecture in the school of nursing are paid from 240 to 600 soles annually. Pupil nurses receive 20 soles per month during the first year, 30 the second year and 40 the third year.

The annual per capita cost of maintenance, exclusive of housing, of patients in this hospital is very low being approximately $110 in the year 1937.

The movement of patients in the hospital is extremely active. The hospital population on the 31st of December, 1936, was 1123. The admissions during the year 1937 comprised 551 first admissions, 195 readmissions and 251 returns from parole, a total of 997. The departures during the year included 458 discharged patients, 264 paroled patients and 190 deaths, a total of 912, leaving a balance of 1208 patients in the institution. In addition there were 157 on parole at the end of the year.

The ratio of first admissions and of patients under treatment to the general population of the country cannot be accurately computed as no general census of population has been taken in recent years. It is estimated, however, that the population of Peru is nearly 6,000,000. If this is correct, the ratio of mental patients to general population in this country is much lower than in the United States. Dr. Caravedo, the medical director, estimates the rate of resident patients per 100,000 of population to be 17. In New York State the rate is about 500. We are told, however, that the Peruvian hospital receives all patients sent to it and does not maintain a waiting list. Why such a small proportion of the people of Peru require treatment in mental hospitals is not answerable from available data.

In 1937, the Hospital Victor Larco Herrera introduced the revised classification of mental diseases which had been adopted by The American Psychiatric Association in 1934. The uniform statistical system of the Association was brought into use at the same time. Through such action a comparison of mental diseases among first admissions in Peru and in the United States is now made

possible. Of the 551 first admissions for the year 1937, 41, or 8.7 per cent, were classed in the syphilitic group; 53, or 11.2 per cent, in the alcoholic group; 32, or 6.8 per cent, in the epileptic group; 24, or 5.1 per cent, in the senile group; 33, or 7 per cent, in the manic-depressive group; 185, or 39.1 per cent in the dementia præcox group. Thirty-two psychotic patients were reported as undiagnosed.

Of the total first admissions, 78, or 14.2 per cent, were reported as without psychoses. Of these, 36 were classed as mental defectives. Comparing the above percentages with those found for first admissions in New York State, we note much higher percentages in Peru in dementia præcox and alcoholic cases, and much lower percentages in arteriosclerotic and senile cases. Apparently, arteriosclerotic cases are not freely sent to the mental hospital in Peru, as only 6 of the 551 first admissions were diagnosed as belonging to this group.

The Hospital Victor Larco Herrera, although serving as a public mental hospital for the whole country of Peru, is not maintained by national appropriations. The hospital is under the control of the "Sociedad de Beneficencia Publica de Lima." This welfare organization supplies funds necessary for maintenance of the institution and for additions and improvements. However, the hospital receives from the councils of the provinces and from the various departments of the national government funds for the maintenance of certain patients for whom these branches of government are responsible. In addition, the hospital receives pay for the maintenance of private patients. From these various sources were received 243,000 of the 633,000 soles expended for the maintenance of the institution in 1937.

A patient is admitted to the hospital on the application of his guardian, custodian or legal representative to the Sociedad de Beneficencia Publica de Lima. The application must state the reasons for the proposed commitment of the patient and must be accompanied by a certificate of one physician to the effect that the patient is mentally ill and in need of treatment in a mental hospital. If payment for the patient's maintenance is to be made, a contract therefor is entered into by his legal representative with the welfare society. Patients may also be committed by court order, police

order or municipal council order by having prescribed forms filled out by authorized physicians and public officials. Provision is also made for the voluntary admission of suitable cases.

Treatment of patients in the Hospital Victor Larco Herrera has been modernized and at the present time compares favorably with that given in many mental hospitals of the United States.

When a patient is admitted to a hospital his personal and family history is compiled; he is given thorough psychiatric and physical examinations with such laboratory tests as may be indicated. Special treatment for physical disorders present is promptly instituted. After the patient has been under observation for a few weeks, his mental disorder is diagnosed and he is assigned to the ward most suitable for his treatment.

Seclusion and restraint are not used.

Patients are encouraged to work in the hospital industries and in the farm and garden but systematic occupational therapy has not been established.

Special treatment of schizophrenic patients by insulin and metrazol was introduced in 1937 and has been continued since that time. Favorable results from the use of both drugs are reported.

A commendable feature of treatment not ordinarily found in mental hospitals is the practice in use here of giving every patient a physical re-examination every three months and a psychiatric re-examination every six months. These examinations are of much value in adjusting treatment to the needs of the individual patient.

Social workers connected with the out-patient department are employed to find suitable places for patients who are ready for parole, and to exercise a certain degree of supervision over patients placed on parole. Patients who have recovered or have improved so that they may live safely in the community are either definitely or conditionally discharged. The conditional discharge is practically a trial leave of absence or parole, and must terminate at the end of three months by definite discharge or by return to the hospital. Court cases may be discharged only on the authorization of the committing magistrate. In 1937, the hospital discharged 76 as recovered and 261 as improved. The rates based on all admissions were 10.2 and 35.0 per cent respectively.

Mental Hygiene Activities in Peru.

Preventive mental hygiene work in Peru is of recent date and is not yet well developed. It consists of two principal activities; first, the publication and dissemination of information concerning mental hygiene, and second, clinic work for both adults and children. The general instruction of the population in mental hygiene is difficult because of the types of people that must be dealt with. More than half of the population of Peru consists of native Peruvians commonly called Indians. The remainder of the population is composed of white people, mostly of Spanish or Italian descent; mixed races representing an amalgamation of Europeans and natives; a considerable number of Negroes and some Chinese and Japanese. A large part of the Indian population does not utilize the services of trained physicians but still uses the primitive treatment methods that were developed during their tribal life. Sorcery and magic have a large place in such methods. Various herbs are used in connection with the magic rites of the medicine men. As nervous and mental diseases are not understood by these primitive people, they are treated by various forms of magic. Among part of the mixed population the ideas of the Indians with respect to the treatment of disease still prevail, but in the main the mixed peoples are more ready to engage the services of physicians when they become seriously ill. This part of the population, however, has very little knowledge of the nature of mental disease and is not inclined to seek the aid of physicians or psychiatrists when relatives become mentally afflicted. The white peoples, although they have had little opportunity to become familiar with psychiatric practice or mental hygiene, are more ready to accept and use new ideas and methods developed by medical science.

Through the efforts of Dr. Honorio Delgado and Dr. H. Valdizan, a propaganda campaign was inaugurated in 1919 for the teaching of mental hygiene to the medical profession. A series of conferences was held in which various aspects of mental health were discussed. These discussions were arranged by an organization known as "Seminario Psicopedagógico," and were attended largely by physicians and educational leaders. In 1922, the first conference on the Peruvian child was held. At this time Doctors Valdizan and Delgado proposed the creation in Peru of a national

mental hygiene league. It was hoped that such an organization would function like the mental hygiene committees of the United States and other countries. The league was organized and prepared, in its first year, a pamphlet on mental hygiene which was published by the "Sociedad de Beneficencia Publica de Lima." The later official recognition of the league is due largely to Dr. M. Caravedo, who since 1932 has published a mental hygiene periodical known as "Boletin de Higiene Mental."

To further aid in the dissemination of mental hygiene principles, Dr. H. Delgado wrote and published in 1922 a small book entitled "Some Aspects of the Psychology of the Child." This book contained an introduction by the late Dr. William A. White, of Washington, D. C. In 1933, Dr. Delgado published another book, entitled "The Mental Development of the Individual."

About 1920 mental hygiene clinics were begun by Dr. Delgado in the Hospital Victor Larco Herrera. These at first consisted of free consultations offered to out-patients by physicians of the hospital. During recent years the clinic work has expanded and at the present time the clinic visits each year number several hundred.

Considerable work has also been done for the prevention of juvenile delinquency. A children's court was organized in 1925 and an organization for the protection of children in 1928. In this work Mrs. M. G. Parks has had a large part. Some years ago she made a study of the work of the Bureau of Child Guidance of the National Committee for Mental Hygiene of New York and of the Judge Baker Foundation in Boston, and has adapted, so far as possible, the methods of these organizations in dealing with the children of Lima. The problem children referred to the protective society are carefully studied and are placed, so far as possible, in proper homes and are supervised for a period averaging more than one year.

The leading organizations of Lima are taking an active interest in child guidance and Dr. Delgado in 1920 formulated a child guidance program, at the request of the Minister of Justice and Education. The program was submitted to the Peruvian Congress but was not adopted.

The psychiatric leaders of Peru clearly recognize that the single mental hospital of Peru is not large enough to accommodate the

mental cases of that country that should receive hospital treatment. The hospital is now crowded beyond capacity and funds are not at present available for its enlargement. In order to meet the situation adequately, it is felt that a new hospital should be established in southern Peru, probably at Arequipa, and later a third hospital should be built to accommodate the people of northern Peru. It seems certain that with the gradual enlightenment of the population, the demand for hospital treatment of mental diseases will be greatly increased. It is also probable that with the psychiatric progress that Lima is now making, means will be found for the establishment of necessary institutions for the treatment of mental disorders.

BIBLIOGRAPHY.

Delgado, H.: La psychiatrie et l'hygiene mentale au Perou. L'Hygiene mentale, journal de psychiatrie appliquée, 8: 121-200, Paris, 1936.

Caravedo, B.: Memoria de la direccion de Hospital Victor Larco Herrera. Peru, 1937.

———: Victor Larco Herrera Hospital. Nosokomeion, X, 1: 64-67, Stuttgart, Germany, 1939.

THE PHANTOM LIMB.

Its Origin and Its Relationship to the Hallucinations of Psychotic States.

By ALFRED GALLINEK, M. D., New York City.

A "sensory ghost" is the term which Weir Mitchell applied to the phantom limb. Three centuries before Weir Mitchell it was known to Ambroise Paré that amputated parts of the body may be perceived in different modifications after the amputation.

The description given by Weir Mitchell is so exhaustive that later authors could not add much to it.

The Origin of the Phantom Limb.

The phantom limb consists of the hallucinations which a person experiences after complete or partial loss of an extremity. Its characteristics are the continuing feeling of the amputated limb, sensations of pain and motion frequently localized in it and the sensation of an approach of the distal parts of the amputated extremity to the proximal stump. Thus after amputation of the whole arm the phantom of the hand may originate at the stump of the shoulder. The phantom limb may be or become smaller than the real one, it may shrink. Its position may correspond to that just before loss of the limb, or its position may seem cramped and uncomfortable. The temperature of the phantom limb often seems to be different from that of the limb retained. Voluntary as well as involuntary and compensatory movements are possible. Thus an extremely peculiar play of movements may be perceived in the missing extremity. Wounds which were present in the actual limb may also appear in the phantom. Frequently the phantom does not appear until some time after the injury.

The explanations offered for the phenomenon of phantom limbs are just as divergent as the observations upon which the various theories are based. A rough analysis of these theories reveals that two main views can be differentiated, the cerebral, partly psychic and the peripheral theory. It must be emphasized that these vari-

ous theories cannot always be formulated strictly in accordance with either one of these fundamental views, but nevertheless the predominance of either the peripheral or the cerebral element is distinctly apparent in almost every paper on the subject. Thus acording to one school of thought the processes responsible for the origin of the phantom limb are chiefly of a cerebral or psychic nature, according to the other the center of gravity of these processes is supposed to lie in the periphery. This latter view point has recently remained somewhat in the background.

The cerebral theory was worked out above all by A. Pick. The frequent observation that the experience of the arm is much less pronounced than that of the hand is explained by the fact that the central representation of the hand is much more expansive than that of the arm. Pick believes that phantoms are missing in congenital defects or injuries of early childhood because the respective amputated part had never been represented in the body image. Schilder in his explanation very justly places the main emphasis on the body image. The suddenness of the loss plays an important rôle since the phantom limb is missing in slowly progressing losses such as freezing or tuberculosis of the joints.

Additional observations may be mentioned in support of the cerebral theory. The gradually diminishing size of the phantom limb is explained by the gradual dwindling of the body image of the adult which is supposedly replaced by the more resistant one of the child. The disappearance of the phantom limb after injuries of the posterior central convolution and the parietal region (Head) may also point to the cerebral character of the phantom. The influence of hypnosis and mescalin on the phantom may also be mentioned. Narcissistic inability to renounce the integrity of the body, the impossibility of adaptation to a sudden defect are not sufficient explanations of the phantom, for phantom limbs may originate without such a defect after a fissure in the plexus. This is further shown by the appearance of phantomlike sensations in cases of not amputated hemiplegic patients who instead of the paralyzed extremity experience another hallucinated one. There are certain relationships to the non-perception of the own defect, the anosognosia of Babinski.

Pitres favors the peripheral theory. He assumes that the phantom sensation originates chiefly from the excitation of the nerve

ends in the scar. Pitres, Souques and Poisot concluded from the disappearance of the phantom after anesthetization of the stump and the return of the phantom when the anesthesia was no longer effective that the genesis of the phantom must be peripheral. When the phantom has already disappeared it may be made to reappear through peripheral stimuli. The perception of a phantom limb already present may be intensified by such stimuli.

CASE MATERIAL.

CASE 1.—This patient was examined three years after an amputation in the knee and later at the middle of the left thigh had been performed.

Some time after the amputation the patient developed attacks of pain which he localized in the amputated leg. Simultaneously with the pains a phantom leg was felt, the presence of which was not even indicated in the painfree intervals. The phantom persists as long as the pain continues. With the disappearance of the pains the sensation of the leg also disappears. At times he feels an itching at the edge of the tibia and wants to scratch himself at that place. In order to ease the pains he tries to bend the phantom leg at the knee. When he wants to bend it, he has to laugh that he has forgotten that "the leg is gone." Before the phantom leg appears there is an indefinite sensation of pain which cannot be localized. The pain precedes the phantom leg, but both disappear at the same time. At times the size of the phantom leg seems to be the same as that of the other leg. Nevertheless, in walking (with crutches) he has a feeling as if it did not touch the ground. Frequently it seems to be shorter.

The patient is unable to say when he first felt the phantom leg, at any rate, not immediately after the amputation, but some time later. The pains in the phantom foot and the itching at the phantom tibia are accompanied by pains in the lower end of the stump. He knows nothing regarding the appearance and the color of the phantom leg; its spacial boundaries are similar to those of the normal leg, but at times seem somewhat indistinct to the patient. If he places a pillow against the stump he feels the pillow at the sole of his foot. Sometimes it seems to him that the foot begins right at the stump, but never when the itching occurs, as this is felt only at the tibia. The temperature of the phantom limb is that of the rest of the body.

The patient feels as though he could move about normally but, as stated above, when he is walking on crutches the phantom leg seems to be shorter and freely suspended. He feels similarly when walking with the prothesis which to him is not animated. No movements in correspondence to actual ones are present in the phantom leg. When the patient sits or lies down he always has the feeling as if the phantom leg were in a horizontal position. In bed it seems to be actually on the mattress, but when he is seated it appears to be extended horizontally, even if the other leg is in a normal position.

The pains in the phantom leg may be relieved by pressing or stroking the stump, but the pains and the phantom leg do not disappear entirely. If the

stump is pricked with a needle, this is felt on the calf. By freezing the stump with ethyl chloride one cannot make the pain and the phantom leg disappear, but on the contrary this procedure brings them out even more clearly. Only when the pains have completely disappeared the phantom limb also vanishes. It has become increasingly more indistinct as the pains subsided. The phantom limb never appears in the absence of pains.

CASE 2.—A man had sustained an injury to the right hand 18 years before the examination. At that time he had immediately lost consciousness and had been operated before he woke up. When he regained consciousness, the thumb, index and middle finger had been removed. After the wound was healed the patient frequently felt his three lost fingers, especially the thumb. It seemed to him as though he could grasp with his fingers. He could not indicate their position. The thumb appeared to be of normal size, the two other fingers seemed smaller. He had no sensations of direct or indirect movements. This phantom sensation appeared only in the presence of pains in the stump. The pains were felt only in the stump.

CASE 3.—Thirteen years before the examination the patient had had amputations through the left calf and the upper third of the right thigh. Phantom limbs had appeared on both sides immediately after the amputation. When the patient was told about the operation he would not believe it. Since that time, *i. e.*, for almost 13 years the phantom limbs have persisted and have remained completely unchanged.

On the right side (proximal third of thigh is retained) he feels his knee beginning at the scar. The lower part of the right thigh is absent in the phantom. He feels the limb distinctly from the knee down. On the left, where the amputation had been carried out at the middle of the calf, he feels calf and foot distinctly down to the heel. The toes are especially distinct and he feels as if he were walking on the tips of his toes. Sometimes the toes are cramped. He cannot indicate clearly the size of the phantom limb, apparently it corresponds to the norm.

He has no idea concerning the appearance and color of the phantom limbs, their spacial boundaries are like those of the rest of the body. With the exception of the missing lower part of the right thigh he feels no decrease in length, but in contrast to the feeling on the left side the toes on the right are missing. Sometimes, when he is not wearing his artificial limb, he feels bending and stretching movements in the right phantom knee joint; actual movements of the muscle stumps in the scar are mistaken for movements in the phantom limb.

When intensely at work or distracted he does not feel the phantom limbs; but in bed, with a tight cover over him, he feels them especially strongly. When he is covered only lightly the feeling is weaker. Stimuli applied to the stump are felt in the stump itself. If he lies in bed entirely uncovered, he does not feel the phantom limbs. As soon as the stumps are touched the phantom limbs immediately reappear. When the artificial limbs are applied the phantoms become very distinct. If a strong pressure is exerted upon the right stump, the patient, even when sitting down, feels as though he were

standing on his right leg. The leg seems to grow through the bed. With slighter pressure on the stump the phantom disappears from the foot to the knee. He then has a feeling of being pinched at the phantom knee.

Without sensory stimulation of the right stump (blanket, touch, clothes, artificial limb) the phantom leg is never present. As soon as it is covered it reappears, if the cover is removed it again disappears for the moment.

CASE 4.—Seventeen years before the examination an exarticulation through the left hip joint had been performed. When the patient awoke from the anesthesia he felt severe pains in the left (amputated) limb. He asked for a hot water bottle on his left leg, but was told that the limb had been removed. The patient would not believe it until he lifted the blanket and convinced himself with his own eyes. When he got up he fell because he had forgotten that one leg was missing and he was trying to walk without crutches.

From the time of the amputation the sensation of the phantom leg has remained unchanged. He feels the leg about half its natural length, but he very clearly distinguishes the foot, *i. e.*, the toes; the heel is less distinct, tibia and knee are only felt in the presence of pains. If he feels an itching on the edge of the tibia, he slaps the stump. This does not stop the itching, but the stronger sensation of the slap distracts from it. Often he feels the thigh and closely attached to it the lower third of the calf with the foot.

The phantom leg hangs down vertically. When the patient lies in bed the leg seems to go through the bed. When he sits on a chair it appears to extend through the chair. The toes are continuously spread out. The normal limb feels "completely, in its entire shape"; the left "rather without shape, as if bandaged tightly."

He has no idea regarding the aspect and color of the phantom limb. If he passes a stone in the garden he wants to avoid it and tries to pull up the phantom leg between the crutches with which he walks.

Patient complained of a constant pain. With increasing pains the phantom leg becomes more and more clearly defined, the ankles are only felt in the presence of the greatest pain, otherwise never.

DISCUSSION.

The phantom limb may appear immediately after the amputation, or some time afterward. The defect may be forgotten to such an extent that one patient wants to protect his missing leg in a fall or prevent it from being touched, another tries to bend his phantom knee, etc. We have observed the presence of the well known sensations of itching and of movements of the toes, the feeling of shrinking fingers, but it is characteristic that the thumb is not concerned. Stimuli applied to the stump are localized partly in the phantom, partly in the stump. The pain is relieved by press-

ing or stroking the stump; it is felt partly in the phantom and partly in the stump. To one patient the phantom limb seems to be thinner; in some cases it gradually fades out in the course of the years, in other cases it remains entirely unchanged.

Before a discussion of the essential factors the following remarkable points deserve mentioning:

The phantom limb may appear to be of normal size, not shorter, but, when walking the patient may lack the sensation of putting his foot on the ground. The absence of definite ideas about aspect and color of the phantom limb is also striking.

Of further interest is the relationship to the artificial limb which may be animated and identified with the phantom limb. However, this need not necessarily be so. The position of the phantom limb is altogether remarkable, it is felt to be largely independent of the position of the body and of the healthy limb.

It is also of interest that the heel is that part of the foot which appears to be most indistinct.

The fact that slapping the stump temporarily relieves unpleasant sensations such as itching, etc., in the phantom limb is comparable to the fact that a pain of long standing may not be felt during the occurrence of acute pain.

The essential factor in all cases is the relationship of the phantom limb to sensory stimulation in the stump. Thus in Case 1 we are mainly interested in the fact that the phantom limb only appeared after peripheral pains and preceded it and that the sensation persisted as long as the pains remain. This patient never had a phantom leg without pains; thus in this case peripheral stimulation is an absolutely necessary requirement for the formation of the phantom. Exactly in the same way in Case 2 the phantom fingers were only felt in the presence of pain; both pain and phantom appeared and disappeared simultaneously. In Case 3 tactile peripheral stimulations, such as the bedcover, clothes and the artificial limb, instead of pain represented the sensory factor. Case 4 is the only one of our cases where the phantom limb was always present. This might be in contradiction to the view of the necessity of peripheral sensory stimulation for the formation of the phantom limb, if the continuous pains in this case would not represent such continuous stimulation. When the pain became very severe the entire limb became more distinct.

The etiologic importance of sensory stimulation is further shown by other facts. Thus in Case 1 paresthesias over the tibia produced the appearance of this part in the phantom, while otherwise the foot appeared to begin directly at the stump.

We believe that it is evident from our cases that peripheral sensory stimulation of some kind—not necessarily pains—is a significant factor in the formation of the phantom limb, and we would assign an important rôle for such stimulation. Even in cases where phantom limbs were felt without amputation the peripheral factor must be considered important, as one will have to assume its presence in lesions of the plexus, for example, just as much as in any amputation.

By placing the main emphasis on the peripheral factor we do not wish to deny the significance of cerebral and emotional mechanisms in the formation of the phantom, and we would emphasize that the phantom must be brought about by a process involving the entire nervous system.

Peripheral stimulation from the stump is eventually carried into the same regions which would be reached by peripheral stimulation from the limb if this were still present; if we may say so—they encounter its old engrams at the place where the "body image" (Schilder) is localized. It is no pure chance that its localizations coincide to a large extent with those of the highest cortical sensory "centers." The primitive stimuli emanating from the periphery are the raw material for the evolution of the phantom.

We have never observed a phantom after amputation of the mamma, penis or facial disfigurement—though this is occasionally mentioned in the literature. This is not in agreement with psychoanalytical or purely psychological theories for the explanation of the phantom. The narcissistic inability to renounce the integrity of the body should be even more pronounced after amputation of the mamma, penis or face than after the loss of the extremities. There can be no doubt that the libidinous significance and cathexis of face, mamma and genitals must be greater than that of the extremities, but nevertheless there are no phantoms after loss of these parts. It seems that the engrams of easily movable parts of the body can be more readily reproduced, than those of less easily movable parts.

The cortical sensory centers of the most movable parts of the body are the carriers of the body image. Afferent sensory stimuli from these centers give rise to the phantom limb which is a peculiar partial hallucination. Peripheral stimuli are the blood which the sensory ghost must drink in order to be awakened to its phantom existence.

The Relationship of the Phantom Limb to the Hallucinations of Psychotic States.

The phantom limb is a hallucination occurring in persons with a perfectly clear mind. What is its relationship to the hallucinations of psychotic states? We have seen that the phantom limb is felt as absolute reality. On the other hand a clear minded person knows that in spite of its high degree of reality this sensation is illusional even though some patients have to convince themselves of the illusional character of the sensation with their own eyes over and over again. The normal amputated person has two forms of perceptions, a subjective and an objective one. By the subjective perception the phantom limb is perceived as a real one, by the objective one this impression is corrected, chiefly on the basis of visual impressions. These two forms of perception apply not only to the phantom as such, but also to the position of the phantom limb and to its relation to the surrounding space. By the subjective perception it may grow through solid objects such as the bed on which the patient lies though objectively the patient knows that this is impossible.

CASE MATERIAL.

L. B. was 62 years old at the time of examination in the summer of 1937. He had been an inmate of the Manhattan State Hospital since 1929. Previously he had been an inmate of Central Islip State Hospital for two periods of several months duration in 1923-24 and 1927.

Many years before he was hospitalized he had wanted to invent dirigibles and to present them to the President. Already at this time he anticipated the second coming of Christ. In later years he felt that he was influenced by spirits who accused him of masturbation, gonorrhœa and syphilis. In 1929 he cut off his right hand because this was "the offending hand and God had told me to take it off."

Since then he hears God's voice every day giving him messages. He also has visions of God. He firmly believes that God possesses his whole body

and that God's voice speaks out of his own. God has endowed him with special powers and entrusted him with a special mission. The voice of God is a small voice, like a woman's voice. The patient himself can be a man or a woman, he is not limited. On the ward he is quiet and cooperative, although he claims that he is kept unjustly at the hospital, because his ancestors came to this country to enjoy religious freedom. He is happy since he cut off his hand.

Asked about his phantom hand he says: "I know, you doctors call it a phantom hand. But it feels different from the way other amputated persons feel." He feels the hand drawn up and is able to move the fingers. The hand feels numb, as if asleep, and cold, but he has no impression of its color. The phantom hand feels as real as the left hand, but he feels a peculiar constant pain in the phantom hand. (Up to this point the patient's description of his phantom hand does not differ at all from the descriptions given by mentally sane amputated patients.) He believes that his missing hand is really present, but rendered invisible by God. Just as there is a visible and an invisible Christ there are visible and invisible hands. On the wrist he feels two bracelets and on the first and second joint of each finger he feels rings, so that he wears 10 rings on the phantom hand. (Actually he wore only one ring when he cut off his hand.) God has given him the rings "Yes, sir, absolutely, in fact, all of them." God adorned the phantom hand with these rings about a year after he had cut off his hand and the phantom hand had made its appearance. The rings are made of metal, "they are gold," and because they are metallic he feels them even more strongly than the hand.

DISCUSSION.

We had expected that the hallucinating mental patients would experience the "normal" hallucination of the phantom limb differently from their pathological hallucinations. When hallucinating patients who hear their voices over the radio are questioned about these voices they will be found to be able to distinguish between the real radio and the hallucinated radio voices. They attribute just as much reality to the hallucinations but there is an indefinable difference. Such an ability of differentiating does not seem to exist between the phantom experience and pathological hallucinations. In the above described case the phantom limb has all the characteristics which it has in the case of normal amputated persons. In addition to these characteristics, however, additional symptoms have been added, which originate from the psychotic experience of the patient and are of the same type as his other hallucinations. The 10 rings on his hand appeared only a year after the phantom. The psychotic hallucination has been super-

imposed on the natural one, but though the two symptoms originate from different roots they are felt and interpreted in the same way. The interpretation is entirely within the range of the psychosis.

Subjective and objective perception which are entirely separate from each other in the normal amputated patient have become fused in the hallucinating mental patients.

The author expresses his appreciation to Dr. W. E. Merriman, superintendent of the Manhattan State Hospital and to Dr. M. P. Lonergan, director of clinical psychiatry, at the Manhattan State Hospital for their kindness in permitting him to examine the last described patient.

BIBLIOGRAPHY.

Head, Henry: Sensory disturbances from cerebral lesions. Brain, 34: 187-189, 1911.

Mitchell, Weir: Injuries of nerves and their consequences. Lippincott and Co., Philadelphia, 1872.

Paré, Ambroise: Quoted after Weir Mitchell.

Pick, A.: Ein Beitrag aus der Kriegsmedizin. Neurologisches Zentralblatt, 34: 257-265, Leipzig, 1915.

Pitres, A.: Étude sur les sensations illusoires des amputés, Annales Médico-Psychologiques, 5: 1-18, 117-192, Paris, Masson and Co., 1897.

Schilder, P. F.: The image and appearance of the human body. Psyche Monographs, K. Paul, Trench, Trubner and Co., London, 1936.

Souques and Poisot: Origine périphérique des membres amputés. Revue Neurologique, 13: 1112-1116, Masson and Co., Paris, 1905.

THE OTHER SIDE OF HALLUCINATIONS.*

PART II. INTERPRETATION.

BY JONATHAN LANG.

INTRODUCTION.

While fully realizing that his opinion based on nine years of direct personal experience of hallucinations contains subjective distortions, the writer feels that his views on the phenomena possess sufficient value to be recorded. He shares the position taken by Karl Mannheim in his book *Ideology and Utopia* that the integration of diverse perspectives offers a more accurate guide to actuality than the concentration upon a single perspective. Hence he offers this statement of the viewpoint of one who has actually experienced hallucinations in the hope that it may to some extent complement the viewpoint of the psychiatrist and so help in clarifying the actuality behind hallucinations.

I. THE SELF AND THE HALLUCINATION.

If there is anything that I can say with certainty concerning the problem of hallucinations, it is that hallucinations are not the product of the activity of the conscious self of the experiencing individual. The conscious self acts merely as a spectator. It does not anticipate; it does not initiate; it does not control the hallucinations. To the conscious self, hallucinations come as phenomena which it experiences. They reach it as already organized configurations. They are, to the self, phenomena which arise outside its range.

Further, a series of more than 50 experiments which I have carried out, each time with negative results, have convinced me that the conscious self *cannot* produce hallucinations (using the term in the specific sense of a spontaneous false stimulation of the sensory nervous system). While I have been able to produce vague affects and vague eidetic images, I have never been able to produce a stimulation of the exteroceptive nervous system identical with that arising from normal external stimuli such as has occurred in the hallucinations which I have experienced.

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* This is the second part of an article on hallucinations written by a patient of mine, whose case is one of schizophrenia of about nine years' standing. The first part, describing hallucinatory phenomena as a personal experience, was published in an earlier issue of this JOURNAL (94: 1089). This part deals with the patient's interpretations of these phenomena and of the formation of delusions on a hallucinatory basis. The author's name, as given here, is fictitious.—AARON J. ROSANOFF.

Hence hallucinations are, to the conscious self, experienced phenomena over which it possesses no control. At the time of their incidence, the self cannot distinguish them from normal sensory stimuli except through the presence of clues in the surrounding situation which indicates such separate identity. They reach it as already organized sensory configurations. The self not only has no part in their organization, but also has no awareness of any factor in the organism to which it belongs which participates in the formation of hallucinations. Consequently it views them as the product of some external factor. When the surrounding situation does not contraindicate, the self assumes that the hallucination signifies the actual external presence of the experienced phenomena. It accepts the hallucination as the experiencing of normal external stimuli. When the surrounding situation indicates that the phenomenon does not have normal external existence, the self still cannot accept the hallucination as the product of its own organism. It tends to the belief that the phenomenon is the result of the influencing of its organism by some external agent.

In complex cases, such as my own, the further conviction develops that the separate incidences of hallucinations and allied phenomena are not the products of separate agents, but rather the repeated manifestation of a single agent. Circumstances present in the experience force this conclusion upon the self. In my own experience, factors of continuity and temporal contiguity and of relevancy, have impelled me to this view. The approximate continuity of the thoughts-out-loud spanning nearly the whole period of wakefulness, day after day, points in this direction. The reference of the thoughts-out-loud to hallucinations which have taken place in the past and their anticipation of hallucinations and other events which subsequently occur, also tend to increase the impression. And their adherence to a central theme—the attempt to produce a messianic fixation—also helps convince me of the continuity of the agent behind the hallucinations. The reference of the thoughts-out-loud to other hallucinations in close temporal sequence—either shortly before or immediately after—supplies temporal contiguity. The accuracy with which the thoughts-out-loud often predict hallucination provides relevancy. The manner in which the thoughts-out-loud have accurately described shifts in the locus of hallucinatory pain, the way in which they have described hallucinations as they are taking place, and the occasions (not very frequent) when the thoughts-out-loud have accurately predicted the nature of hallucinations which have subsequently occurred, lend strength to the impression of an association and relevancy between the thoughts-out-loud and other hallucinations. The interlocking of these factors of continuity, temporal contiguity and of relevancy has forced the conviction of a central controlling agent behind the hallucinations and the thoughts-out-loud.

To sum up, the self of the experiencing individual has very little choice in the concept which it forms concerning hallucinations: the data of the experience determine the self's belief. The self cannot deny having experienced an intricate phenomenon. It knows that it itself did not produce the hallucination, and it believes that they do not arise from the activity of some other

factor of its own organism. The self is forced to the conviction that hallucinations are the product of some external agent acting upon the individual's organism.

II. Objective Factors.

From my nine years of direct experience, I am inclined to the view that this subjective attitude rests upon an objective foundation. While I realize that difficulties exist, I believe that it is possible for me to isolate some approximately objective factors which support such a conclusion.

In the first place, hallucinations first appeared in the history of my case without antecedents. They appeared spontaneously and without warning. The original incidence—an auditory hallucination of a voice claiming to be that of God—came without preceding connections. While my general condition at the time was depressed, due to a hard year at college, it did not contain any factor of a seeking of divine aid. In fact my attitude toward the existence of a God who made direct contact with man, was one of skepticism. The hallucination had no basis in the patterns of my conscious thought. The hallucinations did not commence as the end product of a serial change of single factors; they started as the spontaneous appearance of an already organized configuration.

Except in so far as one form of hallucination has been anticipated by other hallucinations, this absence of antecedents has held true throughout my experience. Hallucinations appeared without anticipation in the conscious field. Often they lacked any connection with the conscious content present at the time of their incidence.

Secondly, the experiential nature of hallucinations to the self has its objective as well as subjective value. The occurrence of the hallucinations in the central conscious (intrapsychic) field as already organized configurations, indicates their origin on the afferent side of that field. The lack of knowledge of the self concerning the hallucinations previous to their incidence, precludes the possibility that the hallucinations arise from conscious or post-conscious processes. As the central awareness—volitional factor of the conscious field, the self would necessarily possess knowledge of activity in the conscious field.

Thirdly, the inability of the self to produce hallucinations experimentally has its objective as well as subjective implications. It rests upon an experimental basis which the reader can test for himself. This experimental inability precludes the possibility that the hallucinations arise from self-initiated processes. (In this connection, the reader should remember that in this paper the term "hallucination" implies a spontaneously appearing complexly organized stimulation of the sensory nervous system identical with normal stimulations except in its lack of a normal initiating agent. The reader should be careful not to mistake affects and eidetic images for hallucinations.)

Fourthly, the intricate pre-existing organization which hallucinations possess before reaching the central conscious field, objectively suggests the

existence of some organizing factor. Before reaching the conscious field, an hallucination already demands (1) the selection of a specific sensory pattern with sometimes the synchronization of processes of more than one sensory system, (2) the addition of specific locational factors, and (3) sometimes the provision of an anesthesia for factors actually existing in the external configuration which are incompatible to the hallucinatory pattern. It is difficult to understand how such a complex organization arises except through the operation of some form of organizing factor.

Fifthly, the innervation of some factor of the afferent side of the nervous system possessing the ability of providing external locational factors, indicates the activation of the exteroceptive nervous system. The external locational factors do not arise in the central field as the product of faulty judgment or reference by the self. They are definitely integrated into the hallucinatory pattern when it reaches the central conscious field. Such incidence of external locational factors requires the overcoming of a basic safeguard of the psychophysiological mechanism of the human organism—the afferent-to-efferent principle of neural conduction, without which no distinction would exist between sensation and imagination. It is upon this distinction that adjustment to the environment depends. In overcoming this safeguard, the hallucinatory process involves the defeat of one of the major protections of the psychophysiological mechanism. It necessitates the operation of some special factor possessing the power to overcome this special resistance.

Sixthly, the hallucinatory process in so far as it involves the innervation of a part of the sensory nervous system of the human psychophysiological mechanism is not a mystic nothing-something. It must function as an energy discharge stimulating the nervous system.

III. Psychiatric and Psychological Theories Concerning Hallucinations.

Turning to the theories concerning hallucinations in the literature of psychiatry and abnormal psychology, one quickly notes differences. A formal discussion of the various competitive theories of psychiatrists and abnormal psychologists lies beyond the scope of this paper. The writer will, however, attempt some comments upon various theories from the perspective of the actual experiencing of hallucinations.

In the first place, his experience contradicts the view that hallucinations arise as the end of a series of stages of deterioration of perceptive processes from an antecedent stage of illusions.[2, 3] As far as my own case is concerned, no illusions preceded the hallucinations; the hallucinations emerged as an already intricately organized phenomenon without any antecedent illusions.

Secondly, his experience does not favor the explanation of hallucinations as due to lesions of the brain in thalamus or other regions.[4] The variety of the hallucinatory patterns, the absence of repetitions and the complexity of the specific incidences, sometimes with the involvement of more than one

sense, and occasionally with anesthesia for factors of the actual external configuration which compete with the hallucinatory pattern, suggest the inadequacy of such a simple explanation.

Thirdly, his experience does not coincide with psychoanalytical stress upon homosexual content as the dominant characteristic of hallucinations.[5] In his own experience, homosexual content was lacking until a suggestion was made by a psychiatrist in the eighth year of the experience. Such content was absent during the eight previous years, and it occurred only sporadically subsequently to the suggestion. In this connection, reference should be made to the frequent influence of suggestion from other persons upon the verbal content of the thoughts-out-loud. This change in response to suggestion has been experienced by the writer on several occasions. It was particularly apparent at one period during which the writer was having treatments from a "spiritual healer." The thoughts-out-loud presented short playlets of struggles between good and evil spirits in keeping with the view of the case advocated by the "spiritual healer." The writer is inclined to question the reliability of verbal content of the hallucinations as an indicator to underlying mechanisms except in cases in which special precautions are taken to avoid all possibility of suggestion.

Fourthly, the writer's experience has made him skeptical of the explanation of hallucinations as results of "projection." With regard to those advocates of the "projection" hypothesis who seem to hold that "projection" of the hallucinations is a quasi-deliberative process in the central conscious field in which the self, through faulty reference "projects" the hallucination,[6, 7] the writer has already presented his dissenting argument. With regard to the view that the process of "projection" takes place outside the central conscious field, but still wholly through the operation of factors of the organism,[8, 9, 10] the writer would point to the difficulty of locating and isolating any such factor in the psychophysiological mechanism. He would draw attention to the circumstance that such factors as "libido," "defense mechanism," or "autocorrective principle" which have been advanced by various schools as factors producing "projection" have as yet only the status of ideological theories. For the present, reasonable grounds for doubt certainly exist.

IV. The Hypothesis of Psychic Infection.

The existing uncertainty of the etiology of hallucinations leaves open the way for the suggestion of an alternate hypothesis. Before entering upon such an attempt, the writer would like to emphasize two aspects of his attitude which otherwise might be misunderstood. In the first place he wishes to make clear that he does not claim to present a definitive explanation; he merely offers an hypothesis which seems reasonable in the light of his nine years of personal experience. He fully agrees that its establishment must await the experience and experiment of others. Secondly, the writer does not question the pathological nature of hallucinations whether of religious

or other types. From his experience of hallucinations primarily religious in type, the writer entirely endorses the psychiatric viewpoint as to the pathological nature of hallucinations—Petrim Sorokin [11] and the mystics to the contrary notwithstanding. The issue and the only issue which the present writer is raising is that of the nature of the pathogenic factor responsible for the phenomena.

For such a discussion of alternative hypotheses of the development of hallucination, the interference with the afferent-to-efferent principle, in the opinion of the writer, forms the critical problem. This disturbance of normal function formed the chief characteristic of hallucinations as they have been experienced by the writer during the past nine years. As a major departure from normal processes, this disturbance seems to postulate some factor not normally present in the human organism. One can provisionally outline the general nature of this factor as an energy factor possessing the capacity of overcoming the afferent-to-efferent principle. The question of the origin and development of this factor is an open one. It is, of course, possible that it may arise as a pathological development of factors normally present. On the other hand, it is also possible that it may represent the intrusion of some originally external agent. Two considerations incline the writer to adopt the second of these alternatives: first, the already mentioned difficulty of locating a definite factor of the psychophysiological mechanism capable of producing the given result; second, the experience of investigators in the field of somatic diseases.

In somatic diseases it has been shown that major departures from the normal processes of the organism are more often due to the intrusion of an originally external agent (a germ, a virus, etc.) than to the spontaneous generation of an agent within the organism. The work of Pasteur and others showed that the commonly held belief of their predecessors that the spontaneous development of a pathogenic factor within the organism was the universal cause of pathological conditions, was a mistaken one. The possibility that the process of infection from without may also operate in psychopathological conditions is one which demands attention. It should not be forgotten that the present dominance of the theory of spontaneous generation of psychopathological conditions may be delaying the advance of psychopathology in the same manner that the spontaneous generation theory of somatic pathological conditions delayed the development of somato-pathology.

The complexity of the factors involved in hallucinatory phenomena and the difficulty of determining them precisely suggest that search for some form of psychic infective agent may lead to valuable results. As to the nature of this postulated psychic infective agent, only vague surmises are at present possible. In so far as the human organism is a physico-chemical mechanism, one is probably safe in holding that this factor must be a physico-chemical agent rather than a mystic nothing-something. Perhaps it may be nothing more complex than a type of micro-organism not yet isolated or some form of virus. However, the complexity of resulting hallucinatory phenomena

indicates that the agent or agents may be something more complex. Since the psychological sphere seemingly deals more specifically with energetic aspects of physico-chemical processes, it is likely that the hypothesized agent may differ from that of somatic diseases in that whereas somatic diseases arise chiefly from chemical activity within the organism induced by the infecting agent, the psychological diseases arise chiefly from physical activity within the organism induced by the infecting agent. The psychic infecting agent may be some form of energy system possessing the capacity of entering the organism and stimulating the nervous system in such a way as to produce hallucinations. Specifically there is the possibility that the agent may be some form of energized gas which has drifted down into the lower atmosphere from the ozone belt and which enters the body through the respiratory tract and lodges in the brain or cerebrospinal fluid.

Such an hypothesis would explain only the final stages of a psychosis, in particular the incidence of hallucinations. Before this possible psychic infective agent can act, a predisposing psychophysiological state is probably necessary. The psychic infective agent presumably acts only to extend a trend preëstablished by deficiencies or excesses in the structure of the organism. The psychophysiological state of the organism may probably determine the general nature of the pathological phenomena; the psychic infective agent, the degree to which the condition develops.

Conclusion.

The problem of hallucinations looms as an important one for the understanding of the nature of psychopathological conditions. From the viewpoint of actual experience, hallucinations form the epicenter from which the psychosis spreads. The clarification of the hallucinatory process will lead far toward the understanding of psychopathology.

Nine years of actual experience have led me to the belief that the crucial factor of the hallucinatory phenomena is the overcoming of the afferent-to-efferent principle. In my experience hallucinations involved complex stimulations of complex patterns of the sensory nervous system. These stimulations were not and could not have been produced by the self of the individual.

The complexity of the patterns of the hallucinations suggests the existence of some form of organizing factor. Already existing knowledge as to the physico-chemical nature of the nervous system implies that this factor must at least operate as a physico-chemical agent—say some form of energy system. Difficulties in locating and isolating such a factor within the human organism suggest the possibility of the entrance of an external agent. The question is raised as to whether the prevalent theory of spontaneous generation of psychopathological phenomena may not be retarding the clarification of the actuality behind hallucinations. The possibility that hallucinations may be produced by the intrusion of some psychic infective agent is one that requires attention.

BIBLIOGRAPHY.

1. Mannheim, Karl: Ideology and utopia. New York, 1936.
2. Morgan, John J. B.: The psychology of abnormal people, pp. 96-97. New York, 1928.
3. Conklin, Edmund S.: Principles of abnormal psychology, p. 73. Second rev. ed., New York, 1935.
4. Menninger, Karl: The human mind, p. 212. New York, 1930.
5. Rosanoff, Aaron J.: Manual of psychiatry and mental hygiene, pp. 34, 524, 569. Seventh ed., New York, 1938.
6. Prince, Morton: Clinical and experimental studies in personality, pp. 399-400. Cambridge, Mass., 1929.
7. McDougall, William: Outlines of abnormal psychology, pp. 344-351. New York.
8. Bleuler, Eugene: Textbook of psychiatry, pp. 59-68. New York, 1924.
9. White, William A.: Outlines of psychiatry, pp. 80-88. Fourteenth ed., Washington, 1935.
10. Strecker, E. A., and Ebaugh, F. G.: Practical clinical psychiatry, p. 29. Philadelphia, 1935.
11. Sorokin, Pitrim: Social and cultural dynamics. 1: 130-134. New York, 1937.

Proceedings of Societies.

THE AMERICAN PSYCHIATRIC ASSOCIATION

PROCEEDINGS NINETY-FIFTH ANNUAL MEETING.

PALMER HOUSE, CHICAGO, ILLINOIS,

MAY 8-12, 1939.

TUESDAY MORNING SESSION.

MAY 9, 1939.

The ninety-fifth annual meeting of The American Psychiatric Association convened in the Grand Ballroom of the Palmer House, Chicago, Illinois, at ten forty-five o'clock, President-Elect William C. Sandy presiding.

CHAIRMAN SANDY.—In the temporary absence of the President, it becomes my honor and privilege to open the ninety-fifth annual meeting of The American Psychiatric Association.

If those present will please rise, the Rev. Herbert W. Prince, Rector of the Episcopal Church, Lake Forest, Illinois, will give the invocation.

REV. HERBERT W. PRINCE.—Let us pray.

We yield Thee high praise and thanks, O God, for the choice vessels of Thy grace and the lights of the world in their assembled generations. In the long process of the development of the human mind, Thou hast step by step revealed the infinite treasures of Thy Mind to Thy children. As man has learned to reach his capacity, Thou hast filled it. In the fundamental laws of being, which is philosophy, and in the fundamental laws of knowledge, which is science, and in the application of those laws, which is art, religion, Thou hast made Thine infinite mind known to our finite minds. We cannot see the end but only the steps along the way. As we, Thy sons and daughters, in humble and reverent obedience explore these steps and do Thy will, do Thou disclose to all of us the infinite resources of Thy will and Thy mind, for Thou art the Fountain of all grace, the Master Light of all our seeing, the Master Mind of all our being, that we may by Thy grace and by Thy light be illumined to see Thee in all Thy beauty and seeing Thee to come to know Thee Whom to know is life forevermore. Amen!

CHAIRMAN SANDY.—*Ladies and Gentlemen:* In the absence of the Honorable E. J. Kelly, Mayor of Chicago, who has been called elsewhere, we have the great pleasure and the honor of hearing from the Honorable John

J. Sonsteby, Chief Justice of the Municipal Court of Chicago, who will welcome us to this city.

In our short conversation after my introduction to Chief Justice Sonsteby this morning, I learned he has been very much interested in our activities for years and really he might be considered a member of our family. We are, therefore, very happy to hear from Chief Justice Sonsteby.

CHIEF JUSTICE JOHN J. SONSTEBY.—*Mr. Chairman and Guests of the City of Chicago:* I regret exceedingly that Mayor Edward J. Kelly is not here personally to greet you, but he was called to Washington.

In coming on his behalf, I welcome you to Chicago, the city that is probably destined to be the largest city of the United States.

We have a great many things here that ultimately will advertise themselves and show that Chicago is the center of so many of the activities of our country, not the least of which is the work in which you are engaged.

In selecting me to represent the Mayor, he probably had in mind the fact that for quite a number of years I have been in rather close touch with your work. As a member of the American Prison Association and the National Conference of Social Work for about 28 years, I remember the days when psychiatry wasn't even considered sufficiently important to be on the program of either of those organizations.

As an honorary member of the 33rd Degree of the Scottish Rite Masonry of the Northern Jurisdiction, I of course have been somewhat associated with and have come to know the work that we have done as part of our contribution to American life, namely, the beginning of this intensive research to ascertain the causes, if not the cure, of dementia præcox. You, of course, know something of that work, some of the results that are now becoming known and which will help materially in the years to come, both in the study of and the cure of, this most dangerous, shall I say, disease or condition.

I became Chief Justice of the City of Chicago in December, 1930. About a year after that, I organized the Psychiatric Institute of the Municipal Court of Chicago. Dr David B. Rotman, who may be here this morning, is Director of that Institute. He, as one of the faculty of Rush Medical College, keeps us in touch with all of the clinics and organizations in Chicago and the United States dealing with this subject. In that institute we handle each year over 2,500 defendants, something that is not known generally. Of that number, a very large percentage are so mentally unbalanced as to require institutional care. In the case of the feebleminded, we commit directly to the state institutions. If defendants require the care of the other institutions, we send them to the Psychopathic Hospital of Cook County and there, after another examination, over 99 per cent of those we send are committed to state institutions.

We are increasing the work of this department year by year. Last year we made quite an intensive study of the cases that came to us through the automobile safety court. In other words, Dr. Sharp, our chief psychologist, took the cases from that branch of our court and made an intensive study

to ascertain the causes that brought these defendants into court. Of course, she found amongst them dementia præcox cases and other mentally unbalanced cases; also the stuttering type, and so on. So that out of that study we really got some very valuable information which has been made available to the profession.

We have made an attempt to coordinate our work in this Institute with the other clinics and institutes of the city. We are very happy to have the new state institution and hospital, the Neuropsychiatric, begun and to have the cornerstone laid today. The laying of that cornerstone, I take it, is delaying the presence of Mr. Bowen and the others who would have preceded me on this program.

This Institute has been the dream, you might say, of Dr. Singer and Mr. Bowen during the last ten years. Now that they have gotten it started, it is going to mean a tremendous advance in the work in the City of Chicago, because the various institutes and clinics will have some place to send children and others requiring hospitalization as well as study.

We are advancing in the City of Chicago. The last ten years have shown tremendous advance in this particular work and I may say we have had the support of Mayor Kelly and the City Council in the work we are doing, particularly in the Municipal Court of Chicago.

I said a little while ago you have not heard much of our Institute. We do not advertise it generally. We have refused to let newspaper reporters come in. We don't let them come in and make photographs. We figure that the persons involved and the families involved have enough load to carry without having publicity in all the newspapers in addition. Of course, that doesn't make the newspapers very friendly toward us, so we get very little write-up regarding the work of the Institute. But once a month I send a release to all the papers and they do publish the number of cases we handle and what is done with them.

The major crimes, such as murder, rape, robbery and holdups, have decreased from 45 to 60 per cent in the last ten years in the City of Chicago, whereas they have increased throughout the entire United States. That is because of the coordination between the Police Department, the Courts, and all the other law-enforcing agencies. We have shown to the world what can be done when the law enforcement agencies, properly directed, work together with the one object in view.

So I speak on behalf of the Mayor and the citizens of Chicago when I welcome you today and hope your visit will be a very pleasant one, and that out of the work you do here, from the papers you will hear read and from the discussions that will follow, there will be a valuable increase in benefits that your various communities will get from the fact that you have been here with us. Thank you!

President Richard H. Hutchings assumed the chair.

PRESIDENT HUTCHINGS.—I would like to call upon Dr. Douglas Singer to make a few remarks. He is Chairman of the Committee on Arrangements. I am asking him to speak out of place for a particular reason.

DR. H. DOUGLAS SINGER.—*Mr. President, Ladies and Gentlemen:* Our Committee on Arrangements has asked me to express to you the very deep appreciation that we feel for your visit to us here. I have been looking forward to this for a great many years. I sincerely hope we won't have to wait so many years before we see you here with us once more.

On behalf of the profession of this city and district, I have asked Dr. Arthur H. Curtis, who is the President of the Chicago Institute of Medicine, to express his pleasure at your visit. Dr. Curtis!

DR. ARTHUR H. CURTIS.—*Mr. President, Mr. Chairman, Members of The American Psychiatric Association and Guests:* On behalf of the Institute of Medicine, I welcome you to Chicago. Perhaps I may presume also to be the unofficial spokesman for Chicago's medical profession as a whole, for they are well aware of and are interested in your meeting today.

Permit me also to greet you on behalf of American gynecology, for our gynecological ancestors were past masters in the art of healing mental twists and cyclical turns a generation ago when psychiatry was still in its swaddling clothes. You psychiatrists have usurped a most generous percentage of the gynecologists' bread and butter patients, those longest lingering, habitually repeating patients; but you have not stopped there. You have also eradicated some of our most cherished ailments such as reflex pain of mysterious pelvic origin, those gynecological headaches and other intangibles long recognized solely due to ovarian disorders and uterine displacements. Yet in spite of all this, we extend to you the friendly hand of welcome.

A closing word. To speak less flippantly and indeed most seriously, the medical profession should congratulate you on the intellectual and scientific growth which has characterized your specialty above all others. We wish you a most profitable time in Chicago.

DR. SINGER.—Thank you, Dr. Curtis!

I would like to say that in our problems among the psychiatric hospitals in this state there has been one group of physicians to whom we have felt we can always turn for help, and that is the group known as the Institute of Medicine of Chicago. Dr. Curtis has not said anything of what that body has done for the State of Illinois in the care and development of its institutions. I feel it is a great pleasure myself to tell you that it has just recently completed a survey of the whole medical service in the Illinois institutions, not only the state hospitals but also in the penal institutions of the state. Their report, I believe, is not quite ready for publication, but I have had some indications of what it contains. They have spent many months of work visiting every one of the institutions, sometimes in quite large groups, and I know that the report they are going to make on this subject is one that is going to be of the very greatest help to us in developing our medical services in the state.

I have long had the feeling—of course, I am not alone in this—that while we as psychiatrists have been particularly interested in introducing psychiatry into medicine, what we needed to do as a corollary of that situation was to

introduce a great deal more of medicine into psychiatry. It is for that reason that I have always felt we should be grateful for the assistance that the Institute of Medicine has been able to give us.

It is also my prividege to introduce to you to represent the State of Illinois, our Director of Public Welfare, a man who has been heart and soul in state institution work ever since I came to the state in 1907, when I found him already here at that time as secretary to the governing body which was then known as the State Board of Charities. Our Governor would have been here to greet you were it not for the fact that he is not well. He has designated Mr. Bowen to represent him, and I believe that nobody more suitable for that purpose could have been found. Mr. Bowen, Director of Public Welfare!

The Honorable A. L. Bowen read his address of welcome.

A. L. Bowen.—It is not often that this very old and distinguished association—one of the first in the American field of medicine—honors a layman with a place upon its program. I am seriously conscious today of the high privilege I enjoy as a lay official of Illinois in welcoming you to Illinois for your 1939 deliberations.

The welcome I extend is but the reflection of the welcome that Governor Henry Horner offers to you. But for a serious illness, from which he is making gratifying recovery, he would be here today to make this address of welcome on behalf of the state.

During his long service as Probate Judge of Cook County, he made a nationwide reputation by his devoted interest in medico-social problems. His concern for the mentally afflicted was shown often by his wise handling of the individuals who appeared before him and by his leadership of community action, against all forms of human unhappiness.

His administration as Governor of this state has been characterized by a tremendous program of institutional rehabilitation, enlargement of facilities for care of mental cases, improvement of personnel, development of the best technique in care and treatment—culminating in the decision to erect now the Psychiatric and Neurological Institute and Hospital, whose cornerstone we laid this morning.

Few men in public life in this country have shown the intelligence and human sympathy that he has shown in dealing with those conditions which have called you into the psychiatric field of medicine. It is his regret that he cannot be here this morning and he has asked me to extend to you the greetings and welcome of Illinois that he would find so much pleasure in delivering to you in person.

Psychiatry and neurology are making fast progress not only in developing their sciences but also in establishing a confidence and place in the public conscience. The future of medical education and practice is in psychiatry. How rapidly this sentiment is spreading among our people, perhaps, the lay administrator of great eleemosynary institutions is better able to judge than even you who devote your time and energies to your own circumscribed practice of medicine.

The world is seeking cures for suffering that is not physical. The world is sensing that our human machine is not divisible; it operates as one unit. A defect in one part affects all the other parts and throws it out of smooth, noiseless gear. More and more certainly the physician of today heads toward his inevitable goal as doctor of human behavior.

It has been our habit to regard our physician as a remedial agent when our behavior has betrayed disorder in the tangible flesh of which our bodies is made. It is pretty well established now that our doctor's responsibiltiy extends into the realm of intangible elements which enter into man's constitution. And I believe that soon we shall go even further and expect of our physician, aid, suggestion, remedy, treatment and cure for all the ills that beset us in our quest for happiness and are inherent in our own physical, mental, social and moral makeup.

Medicine, using that term in its broadest sense, is one of the hopes, one of the bright spots in this quest of mankind. It is recognizing its duty and its opportunity. Its horizon enlarges as it views the scene. What is the field of medicine beyond this frontier, where we hesitate today? Its field is the whole world-wide field of human behavior, far transcending its old accepted conception of responsibility to flesh and blood alone.

For those who today complete their education and training in a modern school of medicine there dawns the day when the practicing physician will be an unsought, back number who has not had training and experience in psychiatry and neurology. The nurse who has not had it likewise will be almost useless. Both institutional and private practice will offer satisfactory recompense. Public sentiment is driving political administration out of our public institutions and substituting the professonal ideal. Our public schools demand specialists. Police departments, the courts and many other important public agencies dealing with human behavior find themselves almost helpless without such advice as you are now able to give, even in such limited volume.

The future man of medicine is to be not a doctor of medicine but a doctor of human behavior, a promoter of human happiness. Whereas, heretofore, his field has been that little area circumscribed by the human body, hereafter his field will be that human unit we call man, in all his relations and close contacts with his fellowmen. He must be able to adjust daily and hourly to conditions changing more frequently and more violently than they do today. Through it all, man must continue to be social or he shall perish. In that different civilization which some of us are beginning to prospect, a man's relation to other men will determine whether he is ill or well. A fever will be a minor event in his career. A day's work well done without the ill effects of sand in the gears will be the test of health. A day's work, poorly done, with irritated and roughened edges as night comes, will be the true symptom of human sickness. Shall medicine in all its implications, education in all its phases, social welfare in all of its fields join hands in one effective force to meet the demands adequately that we already sense and must realize are going to increase a thousandfold in 25 years?

The modern medical school and medical man understands that medicine of 25 years hence will be so unlike medicine of today that they cannot be recog-

nized as having even been related. We marvel today at the feats of medicine and surgery and stand appalled at the precision and celerity of the excision of an appendix, the recalling of the dead to life, the patching up of the old human machine into a fair operating unit. But these feats and these marvels are destined to pale into insignificance when they are put alongside the accomplishments of medical science 25 years hence. Now we deal with the ills of human flesh. Then we shall deal with the ills of mal-adjusted human beings, the ills of a disturbed and distraught human life, the ills of a social order torn and disrupted by the unsatisfactory behavior of its disoriented members. In the presence of such an inspiration how can I have patience with the pessimistic thought of the day that there is no future for our youth, or that this profession is overcrowded? The day of heroics on the vacant plain, of adventure in the unexplored forest, of discovery in unknown lands has passed, but the day of adventure, of heroics and of brilliant exploits in the mysterious and glamorous realms of human behavior lie just ahead and are as real as life itself.

Again be assured, Doctors of Human Behavior, of Illinois' cordial welcome to you on this auspicious occasion when the world so appealingly extends its hands to you and you are able to offer to the world a new hope and a new star leading to the manger of human happiness.

PRESIDENT HUTCHINGS.—Thank you, Doctor.

I will now call upon the President-Elect, Dr. William C. Sandy, to make reply to the distinguished speakers who have welcomed us in the name of the Association.

PRESIDENT-ELECT SANDY.—*Chief Justice Sonsteby, Mr. Bowen, Dr. Curtis and Dr. Singer:* On behalf of the officers and members of the American Psychiatric Association, may I express our very deep appreciation of your cordial words of welcome. It may be interesting to remind you that this is the third occasion upon which our Association met in Chicago, the first being the 47th meeting in 1893, the second the 74th meeting in 1918, the latter of course during very strenuous times and our program then being characterized by many military titles. Probably very few of us had, therefore, experienced the advantages of your city as a convention city in these meetings, but we have become otherwise informed about the varied and splendid facilities such as for education, hospitalization, recreation, and so on.

We are very glad to have these further words of welcome, especially from those who are so much interested and more or less intimately connected with our own activities. May I, therefore, for the Association thank you again for your expressions of gracious hospitality.

PRESIDENT HUTCHINGS.—Thank you, Dr. Sandy!

Report of the Committee on Program, Dr. Roscoe W. Hall!

Dr. Hall announced several changes in the program.

PRESIDENT HUTCHINGS.—What is your wish in reference to the report of Dr. Hall? Shall you adopt it?

DR. HENRY I. KLOPP.—I make a motion we adopt it.

The motion was seconded and carried.

PRESIDENT HUTCHINGS.—The Secretary now has a report of the Council which he will present.

Secretary Ruggles read the report of the Council.

SECRETARY RUGGLES.—The Council met at the Palmer House Hotel, May 8, 1939, at 5 p. m. Fifteen members were present. The President, Dr. Hutchings, presided.

The Secretary reported changes in the membership as follows:

Two Fellows and one Member sent letters of resignation which could not be accepted. It was recommended that the JOURNAL be discontinued to 13 Members and 14 Fellows whose dues were two years in arrears. The Council voted to drop from the rolls of the Association, 1 Associate Member, 4 Members and 4 Fellows, three years in arrears, three notices and a special letter having been sent.

The Council recommended for approval the following for Life Membership, who were elected to Active Membership in 1909, with 30 years service:

Dr. Dana F. Downing, Richmond Hill, L. I., N. Y.
Dr. J. S. De Jarnette, Staunton, Va.
Dr. Samuel W. Hamilton, New York, N. Y.
Dr. Thomas P. Prout, Summit, N. J.
Dr. D. J. McCarthy, Philadelphia, Pa.
Dr. J. Moorehead Murdoch, New Kensington, Pa.
Dr. J. Milton Scanland, Napa, Calif.
Dr. G. Kirby Collier, Rochester, N. Y.

Dr. D. Ewen Cameron presented the report of the Board of Examiners in place of the Chairman, Dr. William E. Gardner, on which the Council voted to recommend for election 64 applicants for Associate Membership, 150 applicants for Membership, 33 to be transferred from Associate Member to Member and 14 to be transferred from Member to Fellow.

The Council also voted the reinstatement of 1 Member and 4 Fellows.

The Council approves the recommendation of the Board of Examiners for furthering determining qualifications for Fellowship.

The Council accepted and approved for printing in the proceedings the reports of the following Committees namely: Executive, Special Committee of which Dr. Chapman is Chairman, Psychiatry in Medical Education, Standards and Policies, Ethics, Public Education, Research Committee, Committee on Arrangements for 1939 and 1940 meetings.

The Council recommended that the Chairman of the Committee on Psychiatry in Medical Education be authorized to seek financial assistance from some of the interested foundations toward the inauguration of regional institutes for instruction in psychiatry in order that younger staff members in various hospitals may be helped in meeting the requirements of the American Board of Psychiatry and Neurology. The Council recommended that any

arrangements for such financing and the personnel of such institutes be approved by the Executive Committee.

In considering the report of the Special Committee of which Dr. Chapman is Chairman, it was voted by the Council that a resolution of the Massachusetts Psychiatric Society that a careful study of the Constitution and By-laws of the Association with particular reference to the election of officers be referred to this Committee and that they should report back to Council at the December 1939 meeting.

The Council also gave authority to this Committee to create the machinery for and set up an Editorial Board to supervise the compilation of a biographical directory of the Members and Fellows of the Association and it further recommended that a sum not exceeding $2500 be appropriated for the accumulation of the data necessary for such directory so that accumulation of data in the offices of the Association may proceed at once.

The Council adopted the resolution of the same Committee authorizing the incoming President to appoint a military mobilization committee, with power to represent the Association to the military authorities and to draw up a comprehensive plan for the mobilization of psychiatric facilities for the military and civilian needs of the population.

This Committee recommended that the annual dues of Fellows should be raised from $10.00 to $15.00. The Council voted against this recommendation but referred the matter of Fellowship dues back to the Committee for further study and report.

The Executive Committee in its report stated that at the December meeting the Chateau Frontenac Hotel in Quebec City, Canada, was selected as the site for the 1940 meeting of the Association and the date fixed as June 10–June 14, 1940.

The Council adjourned at 10.35 p. m.

PRESIDENT HUTCHINGS.—You have heard the report of the Council. It is in order to adopt this report and ratify the proceedings, if it is your wish. Do I hear a motion?

DR. WILLIAM L. RUSSELL.—I so move, Mr. President.

The motion was seconded, was put to a vote and carried.

PRESIDENT HUTCHINGS.—Dr. Ruggles, will you report as Secretary and Treasurer?

Secretary Ruggles presented his report.

SECRETARY RUGGLES.—I shall first give the summary of the membership of the Association. At present, the total membership is made up as follows:

19 Honorary Members
60 Life Members
4 Corresponding Members
860 Fellows
985 Members
105 Associate Members

Total membership on May 15, 1938, was 1880.

Total membership on April 15, 1939, was 2033, making a net gain of 153.

The report of the Treasurer is based upon the findings of the certified public accountant and represents the period from May 15, 1938, to April 14, 1939, or 11 months. This report will appear in full in the proceedings. In order to conserve time, I shall abstract it.

The cash balance on May 15, 1938, was $38,404.19. Total cash receipts were $18,094.56, making a total of $56,498.75. During the same period, cash disbursements amounted to a total of $28,977.77, leaving a cash balance on April 14, 1939, of $27,520.98.

The JOURNAL account showed on May 15, 1938, a balance of $370.76. The total cash receipts during the period from May 15, 1938, to April 14, 1939, were $11,674.34, making a total of $12,045.10. Disbursements during the same period were $11,855.17, leaving a cash balance on April 14, 1939, of $189.93.

PRESIDENT HUTCHINGS.—We must act upon the report of the Secretary-Treasurer. Will you accept it?

DR. FREDERICK W. PARSONS.—I move we accept the report.

The motion was seconded and carried.

PRESIDENT HUTCHINGS.—The Chair will announce the appointment of a Committee on Resolutions, with the request that they make their report to the Association at noon on Friday of this week. I will ask the following Fellows to act as the Committee on Resolutions:

Dr. William L. Russell, New York.
Dr. Glenn E. Myers, California.
Dr. H. Dawson Allen, Jr., Georgia.

The next order on the program is the memorial for deceased members. I will ask the audience to stand while the Secretary reads the list of members who have passed on during the year.

The audience stood during the reading of the names of the deceased members by Secretary Ruggles, as follows:

Samuel Ginsburg, M. D., Bronx, N. Y., died Dec. 29, 1937.
John W. Stevens, M. D., Nashville, Tenn., died Mar. 18, 1938.
Paul C. Lybyer, M. D., Ogdensburg, N. Y., died Apr. 23, 1938.
Leon E. Duval, M. D., Ionia, Mich., died June 17, 1938.
David R. Clark, M. D., Detroit, Mich., died July 3, 1938.
Frederick Peterson, M. D., New York, N. Y., died July 9, 1938.
John Levy, M. D., New York, N. Y., died July 11, 1938.
Charles L. Vaux, M. D., Newark, N. Y., died July 23, 1938.
W. Scott Farmer, M. D., Nashville, Tenn., died Aug. 5, 1938.
Frederick Tilney, M. D., New York, N. Y., died Aug. 7, 1938.
W. Frederick Dey, M. D., New York, N. Y., died Sept. 9, 1938.
G. Lef. de Bellefeuille, M. D., Outremont, P. Q., died Sept. 21, 1938.

Jo M. Ferguson, M. D., S. Carrollton, Ky., died Sept. 21, 1938.
Louis J. Bragman, M. D., Binghamton, N. Y., died Oct. 12, 1938.
Lionel L. Cazavanette, M. D., New Orleans, La., died Oct. 15, 1938.
Charles F. Applegate, M. D., Los Angeles, Calif., died Nov. 27, 1938.
George F. Inch, M. D., Ypsilanti, Mich., died Nov. 29, 1938.
J. Allen Jackson, M. D., Danville, Pa., died Dec. 1, 1938.
Richard H. Hutchings, Jr., M. D., Wingdale, N. Y., died Dec. 14, 1938.
Donald Gregg, M. D., Wellesley, Mass., died Jan. 6, 1939.
D. Percy Hickling, M. D., Washington, D. C., died Jan. 9, 1939.
Thomas L. Houlton, M. D., Omaha, Neb., died Feb. 1, 1939.
Monroe A. Meyer, M. D., New York, N. Y., died Feb. 27, 1939.
Lesser Kauffman, M. D., Buffalo, N. Y., died Mar. 11, 1939.
Arthur H. Harrington, M. D., Providence, R. I., died Mar. 12, 1939.
Frank P. Norbury, M. D., Jacksonville, Ill., died Mar. 15, 1939.
Charles S. Woodall, M. D., Brandon, Vt., died Mar. 26, 1939.
Lewis M. Walker, M. D., Cambridge, Mass., died Apr. 4, 1939.

PRESIDENT HUTCHINGS.—Dr. Sandy, I turn the meeting over to you.

President-Elect Sandy assumed the chair.

CHAIRMAN SANDY.—*Members of The American Psychiatric Association, Ladies and Gentlemen:* In accordance with an old custom, it is now my honor and privilege to present one who needs no introduction to this audience, President Richard H. Hutchings, who will give his annual address. Dr. Hutchings!

President Hutchings read his presidential address, following which the audience arose and applauded.

CHAIRMAN SANDY.—By regular custom it is not permitted to have the President's address discussed. We are greatly indebted, however, to President Hutchings for his contribution, and I am sure that we would like to have some comments from some of the Fellows. I shall, therefore, call upon Dr. Karl Menninger, of Kansas, for a few remarks.

DR. KARL A. MENNINGER.—*President Hutchings, Mr. Chairman:* When a man has worked in his chosen field for over 40 years and has done so with a consistency of effort, a devotion of purpose and a cogent application of intelligence and foresight and open-mindedness, it is a significant event that he should be selected from among his colleagues as President of the organization representing his life work. And it is a significant moment in that event when this man, this leader, concentrates his ideas, his ideals, his vision of the goal of his own life and that of his fellow members in that verbal crystallization that we call a presidential address.

You have just heard the message of President Richard Hutchings. To those of us who have walked with him and worked with him and looked for his friendly face and presence at meeting after meeting for many years, it is just what we expected. It was like the man who wrote it and delivered it. It was characterized by clarity, charm, dignity and intelligence, but, above all, by a youthful, forward-looking spirit.

This, I think, is also characteristic of Dr. Hutchings, charm, dignity, intelligence and a young heart. He has always been known for his open-mindedness, for welcoming new ideas, for applying new methods. He was one of the first American psychiatrists to recognize the importance of psychoanalysis.

I sincerely hope that a copy of this address this morning will be sent to Professor Freud, for I know that it would please him.

Dr. Hutchings has always made education his keynote. Few living men have had buildings dedicated to them, named for them, devoted to the thing that they believed in and worked for—the education of psychiatry. I think the State of New York is to be congratulated on having done this to Dr. Hutchings, for the beautiful building at the institutions at which Dr. Hutchings has given so many years of his life.

Dr. Hutchings is always interested in psychiatry and everything that pertains to psychiatry. He has always encouraged the young men, himself always studying, thinking, working, to make progress in our branch of science. He has done this work bravely, persistently, conscientiously, even when the clouds of personal tragedy rested upon his shoulders.

The ideas advanced by President Hutchings, I think, were very important, the function of psychiatry in education and the psychiatric education in particular, need of more home provision, the increasing number of patients, the need of more trained supervision for the care of them, the importance of new viewpoints in our scientific work. I think we should take these ideas seriously. I know our Association would represent his actual work, the fruits of his work, the new suggestions our President has made this morning.

Dr. Hutchings, I think it should be a great meeting, one of the finest meetings yet, with such a leader who has spoken so characteristically this morning, with dignity and charm, with intelligence, and above all with a youthful spirit.

Thank you!

CHAIRMAN SANDY.—Before we adjourn, the Secretary has an announcement to make.

Announcement by Secretary Ruggles.

CHAIRMAN SANDY.—The morning session is now adjourned.

The meeting adjourned at twelve thirty-five o'clock.

WEDNESDAY MORNING SESSION.

MAY 10, 1939.

The meeting convened at nine-forty o'clock, President Hutchings presiding.

PRESIDENT HUTCHINGS.—The session will please come to order.

We have some business to be transacted before the regular program begins. First is the report of the Council. The Secretary will please read it.

Secretary Ruggles read the report of the Council.

SECRETARY RUGGLES.—Meeting of the Council was held at the Palmer House on May 9th at 4 p. m. with President Hutchings in the Chair and fourteen members present.

Dr. Arthur P. Noyes made an additional report for the Committee on Standards and Policies reading a letter from Dr. William Menninger for the Central Neuropsychiatric Association dealing with a proposed study of private mental hospitals. This matter was referred to the Committee on Standards and Policies.

Dr. S. W. Hartwell, Chairman of the Committee on Psychiatric Social Service, submitted his report which was accepted for publication and also gave a report from a sub-committee, which had drawn up minimum standard curriculum for schools on psychiatric social work. The Council authorized his committee to send out letters to the various schools on psychiatric social work announcing these standards and he was instructed to report back to the Council results of such letters.

Dr. George H. Stevenson, Chairman of the Committee on Psychiatric Nursing, presented the report of his committee, which was discussed and adopted for publication with commendation for the work of the committee.

The Council approved a certificate that may be issued to approved nursing schools for undergraduate, postgraduate and affiliated courses, such certificate to be furnished by the Committee and the Council also approved the standard set up by the Committee for undergraduate, graduate and affiliated courses recommended by the Committee.

Council also requested that these standards be printed, if possible, in the JOURNAL and, if not, to be independently printed.

Dr. Neil A. Dayton reported for the Committee on Nomenclature and Statistics. The report was adopted for printing and it was approved that the present nomenclature be preserved intact for the 1940 revision of the standard classified nomenclature of diseases.

Dr. Winfred Overholser reported for the Committee on Legal Aspects of Psychiatry, this report was discussed and adopted by the Council for publication.

Dr. Clarence B. Farrar, Editor of the AMERICAN JOURNAL OF PSYCHIATRY, reported and announced that a vacancy existed on the Editorial Board due to the resignation of Dr. Harry Stack Sullivan. He recommended Dr. William C. Sandy for this vacancy on the Editorial Board and Dr. Sandy was duly elected by the Council.

The Council voted to Dr. Farrar authority to obtain necessary editorial assistance and to expend for such assistance up to $2000 for the coming fiscal year.

Meeting adjourned 5.20 p. m.

SECRETARY RUGGLES.—I would like to announce the registration of last night. There are 530 Members and Fellows registered and 619 non-members, a total registration of 1149.

PRESIDENT HUTCHINGS.—The next order of business is the report of the Nominating Committee, Dr. C. Macfie Campbell, Chairman.

DR. CAMPBELL.—Mr. President, I beg to submit the report of the Nominating Committee for officers of the American Psychiatric Association to be elected at this meeting:

For President, Dr. William C. Sandy, Harrisburg, Pa.

For President-Elect, Dr. George H. Stevenson, Ontario Hospital, London, Ontario.

For Secretary-Treasurer, Dr. Arthur H. Ruggles, Providence, R. I.

For Members of the Council:

Dr. Richard H. Hutchings, Utica, N. Y.
Dr. Lawrence Kolb, Washington, D. C.
Dr. William J. Tiffany, Albany, N. Y.
Dr. E. Armitage Baber, Cincinnati, Ohio.

For Auditor, Dr. Charles W. Castner, Austin, Texas.

The report is signed by the members of the Committee:

C. MACFIE CAMPBELL, *Chairman,*
ROSS MCC. CHAPMAN,
CLARENCE O. CHENEY,
WILLIAM L. RUSSELL,
C. FRED WILLIAMS.

DR. FREDERICK W. PARSONS.—Mr. Chairman, I move the Secretary be instructed to cast one ballot for the report of the Nominating Committee.

The motion was seconded by Dr. Thom.

PRESIDENT HUTCHINGS.—It is moved and seconded the Secretary be instructed to cast one ballot for the slate of officers nominated for the coming year.

There is an opportunity for other nominations from the floor. Does the Chair hear any other nominations? There seem to be none. I will then put the motion.

The motion was put to a vote and carried.

PRESIDENT HUTCHINGS.—Is there any unfinished business that anyone has to offer?

Is there any new business?

At this time I take great pleasure in presenting to you one of our esteemed Fellows who is the President-Elect of the American Medical Association, Dr. Rock Sleyster.

DR. ROCK SLEYSTER.—*Mr. President, Fellows of the Association, My Friends:* You see I am acquiring presidential technique. We are living in a very muddled time and a very muddled world and I think probably this accounts for the American Medical Association trying out psychiatric leadership this coming year.

It has been a pleasure indeed to be here this morning and to greet you. I hope that during the year that I occupy this office, the members of this Association will feel very free to call on me for any help I can be in advancing our specialty. Thank you.

PRESIDENT HUTCHINGS.—Thank you, Dr. Sleyster! We, on our hand, wish you success and satisfaction in the administration of your office for the coming year.

The meeting adjourned at nine-fifty o'clock.

ANNUAL BANQUET.

At 7 p. m. on Wednesday, May 10, 1939, the Annual Banquet occurred in the Grand Ballroom of the Palmer House at which 500 members and guests had the privilege and pleasure of hearing Dixon Ryan Fox, LL. D., Litt. D., present his most stimulating address.

Following the Annual Address, there was dancing until 1.30 a. m. with music by the Lew Diamond Orchestra.

THURSDAY MORNING SESSION.

MAY 11, 1939.

The meeting convened at nine thirty-five o'clock, President Hutchings presiding.

PRESIDENT HUTCHINGS.—The Secretary has an important announcement to make.

Secretary Ruggles announced a meeting of the Council and several changes in the program.

PRESIDENT HUTCHINGS.—The next order of business is election of members. I would like to call on Dr. Cameron, of the Board of Examiners, to come forward and make a statement.

DR. CAMERON.—*Mr. President, Members of the Association:* You will find on the chairs lists of members who have been recommended for your consideration to various positions within the Association, those recommended as Associates and those for Membership and those for Fellowship. I would like to emphasize to you that these names have been recommended by the Board of Examiners to the Council and by the Council to you for final action. Considerable care is taken in collecting data in regard to the various applicants throughout the year. The Executive Secretary of the Association has been in correspondence with many of these people in regard to their various qualifications.

This year, as those of you who attended the dinner last night heard, particular care has been directed towards selecting for fellowship those people who make particularly outstanding contribution to our Association in the field of psychiatry. The Board hopes that you will give these recommendations careful scrutiny as the final decision as to who is elected rests with you.

PRESIDENT HUTCHINGS.—You have before you the complete list of Associate members, Members and Fellows, whose qualifications have been investigated by the Board of Examiners and who were approved by the Council and whose election is recommended to the Association, also those recommended for transfer.

DR. FREDERICK W. PARSONS.—Mr. Chairman, I move the Secretary be instructed to cast one ballot for the election of the ladies and gentlemen on the attached list as members and for the election as Fellows of those on that list, including the entire list, to the classes in which they properly belong.

The motion was seconded by Dr. Stevens, was put to a vote and carried.

MEMBERS ELECTED MAY 11, 1939.

ASSOCIATE MEMBERS.

1. Abrahamer, Hyman W., Buffalo, N. Y.
2. Alderman, Jerome E., Syracuse, N. Y.
3. Alexander, Howard G., Marion, Ind.
4. Anderson, Glenn C., Los Angeles, Calif.
5. Beanstock, Sam, Danville, Ill.
6. Berlien, Ivan Clark, Detroit, Mich.
7. Bernstein, Eric L., Arlington Ht., Mass.
8. Boldt, Waldemar H., New York, N. Y.
9. Bramkamp, Robert G., Talmage, Calif.
10. Brill, Norman Q., New York, N. Y.
11. Caldwell, John M., Jr. (Captain), Canal Zone.
12. Card, John P., San Francisco, Calif.
13. Chase, Louis S., Boston, Mass.
14. Cottington, Frances, Worcester, Mass.
15. Cromwell, James Oliver, Blackfoot, Idaho.
16. Cushing, Jean G. N., Baltimore, Md.
17. Deronde, Margaret, Norristown, Pa.
18. Des Rochers, Jean B., St. Cloud, Minn.
19. Diamond, Murray A., Lexington, Ky.
20. Dub, Leonard M., Columbus, Ohio.
21. Easton, Donald Robertson, Toronto, Ont., Canada.
22. Eisenbud, Jule, New York, N. Y.
23. Eisner, Eugene, Osawatomie, Kan.
24. Felsher, Zachary, Knoxville, Iowa.

25. Fineman, Leon E., New York, N. Y.
26. Grand, Henry O., Ward's Island, N. Y.
27. Grossman, Maurice, Palo Alto, Calif.
28. Heksh, Bela H., New York, N. Y.
29. Hieronymus, Ethel E., Lakeland, Ky.
30. Inburgia, Frank J., Marion, Ind.
31. Isenberg, Morris, East Moline, Ill.
32. Jenkins, Richard L., Orange County, N. J.
33. Kaplan, Maurice, Chicago, Ill.
34. Klotz, Maurice, St. Cloud, Minn.
35. Kupper, William H., Miami Springs, Fla.
36. Lang, Leonard C., Buffalo, N. Y.
37. Lau, Joseph Fook Chan, Kaneohe, Oahu, Hawaii.
38. Lester, David Washington, Compton, Calif.
39. McClintock, Edward A., Palo Alto, Calif.
40. Madden, Arthur B., N. Chicago, Ill.
41. Majka, Frank A., Ft. Lyons, Colo.
42. Mason, Aaron S., Knoxville, Iowa.
43. Mershon, Ronald B., Canandaigua, N. Y.
44. Moore, Walter La Mar, St. Louis, Mo.
45. Morse, Robert T., Rockville, Md.
46. Pessin, Joseph, Madison. Wis.
47. Peterson, Elmer, American Lake, Seattle, Wash.
48. Pignataro, Frank P., Marlboro, N. J.
49. Pleasure, Hyman, Dannemora, N. Y.
50. Plesset, Marvin R., Norristown, Pa.
51. Rendall, Margaret G., Toronto, Ont., Canada.
52. Robinson, Joseph Franklin, Iowa City, Iowa.
53. Ruskin, Samuel H., Eloise, Mich.
54. Sandy, William Allee, Indianapolis, Ind.
55. Shawver, John R., Dayton, Ohio.
56. Stein, Sam I., Chicago, Ill.
57. Stellner, Howard A., Chillicothe, Ohio.
58. Stevens, Ralph E., Chattahoochee, Fla.
59. Sutton, Isaac, Toronto, Ont., Canada.
60. Turner, William J., North Little Rock, Ark.
61. Vicary, William H., Bedford, Mass.
62. Wellman, Marvin, Toronto, Ont., Canada.
63. Wintrob, Benjamin, Toronto, Ont., Canada.
64. Yee, Cyrus William, Kaneohe, Oahu, Hawaii.

MEMBERS.

1. Alcorn, Douglas E., Victoria, B. C., Canada.
2. Allen, Austen G., Cobourg, Ont., Canada.
3. Anderson, John B., Ft. Monroe, Va.
4. Atkinson, Velma H., Hathorne, Mass.

5. Barbato, Lewis, Galveston, Texas.
6. Bartle, Harvey, Jr., Philadelphia, Pa.
7. Barton, John C., Independence, Iowa.
8. Berger, Benjamin, Mayview, Pa.
9. Beyer, Margaret V., Sykesville, Md.
10. Bigelow, Newton J. T., Utica, N. Y.
11. Bingham, Roy E., Louisville, Ky.
12. Binzley, Richard Francis, Brentwood, L. I., N. Y.
13. Blankinship, Rex, Richmond, Va.
14. Boyd, David A., Jr., Ann Arbor, Mich.
15. Brennam, Edward L., Hartford, Conn.
16. Bridgeman, Richard B., Jr., Marion, Ind.
17. Brockman, Helen, Independence, Iowa.
18. Brosin, Henry W., Chicago, Ill.
19. Brown, Ralph W., Northampton, Mass.
20. Cameron, Eugenia Sue, Baltimore, Md.
21. Campbell, Howard R., Dayton, Ohio.
22. Chappell, Sidney L. (Lt. Col. M. C.), San Francisco, Calif.
23. Chor, Herman, Chicago, Ill.
24. Clinco, Arthur A., Brooklyn, N. Y.
25. Clouse, Everett F., Columbus, Ohio.
26. Corwin, William, Waltham, Mass.
27. Coyne, Anna R., Washington, D. C.
28. Craig, Henry Roosevelt, Eloise, Mich.
29. Crane, Albert L., Greenwich, Conn.
30. Davis, John E., Jr., Trenton, N. J.
31. Drysdale, William T., Wood, Wis.
32. Evans, John Westley, Portland, Ore.
33. Ewalt, Jack R., Denver, Colo.
34. Fair, Baxter B., Ellis Island, N. Y.
35. Fialko, Nathan, Wards Island, N. Y.
36. Fingert, Hyman H., New York, N. Y.
37. Ford, Hamilton Faith, Galveston, Texas.
38. Foster, Ruth, New York, N. Y.
39. Foster, Thomas L., Larned, Kans.
40. Glotfelty, James S., Sheridan, Wyo.
41. Gosline, Anna J., Utica, N. Y.
42. Griffin, Edgar D., Washington, D. C.
43. Grotjahn, Martin, Chicago, Ill.
44. Hamburger, Werner, Utica, N. Y.
45. Hannett, Frances, Chicago, Ill.
46. Harkavy, Edward E., New York, N. Y.
47. Hatfield, George C., Fairville, N. B. C.
48. Hauk, Oscar S., Knoxville, Tenn.
49. Hearn, Harry E., Governors Island, N. Y.
50. Heim, Herbert E., Harrisburg, Pa.
51. Hirrschoff, Ernest A. F., Canandaigua, N. Y.

52. Holmes, Charles M., Orangeburg, N. Y.
53. Holt, William L., Jr., Worcester, Mass.
54. Holtz, Edward, Amityville, L. I., N. Y.
55. Inman, William C., Danvers, Mass.
56. Jarvis, Marjorie M., Rockville, Md.
57. Jensen, Reynold A., Minneapolis, Minn.
58. Jensen, Walter S. (Major, M. C.), Hot Springs, Ark.
59. Johnson, George D., Spencer, W. Va.
60. Kerschbaumer, Louise, St. Peter, Minn.
61. Kozol, Harry L., Boston, Mass.
62. Kwiterovich, Peter O., Danville, Pa.
63. La Mar, Norvell C., New York, N. Y.
64. Landis, Edward E., Louisville, Ky.
65. Levy, Norman A., Chicago, Ill.
66. Lipetz, Basilia, Albany, N. Y.
67. Long, Roy Hamilton, Morganton, N. C.
68. McCarty, William H., Marion, Va.
69. McDowell, James R. (Major), Washington, D. C.
70. Marcovitch, Joseph, Dwight, Ill.
71. Markey, James B., Hopkinsville, Ky.
72. Marmor, Judah, New York, N. Y.
73. Marsh, Ina A., Buffalo, N. Y.
74. Meehan, Marjorie C., Princeton, N. J.
75. Meister, Franklin O., Madison, Wis.
76. Merillat, Herbert C., Sedro Woolley, Wash.
77. Meyerson, Solomon B., Menard, Ill.
78. Miller, Horace Gilmore, Salem, Ore.
79. Mitchell, Henry S., Ft. McHenry, Baltimore, Md.
80. Moulton, Lillian G., Indianapolis, Ind.
81. Mueller, William D., Denver, Colo.
82. Nielsen, Johannes M., Los Angeles, Calif.
83. Osgood, Carroll Wilcox, Wauwatosa, Wis.
84. Owen, Joseph Walker, New York, N. Y.
85. Peffer, Peter A., Mendota, Wis.
86. Perry, Herbert A., Medical Lake, Wash.
87. Petty, Alonzo A., Dayton, Ohio.
88. Phillips, Charles G., Lexington, Ky.
89. Phillips, Paul L., Hartford Conn.
90. Pinney, Ione, Stockton, Calif.
91. Poliak, Percy P., San Francisco, Calif.
92. Porter, William C. (Lt. Col.), Washington, D. C.
93. Pouppirt, Pearl S., San Francisco, Calif.
94. Preu, Paul W., New Haven, Conn.
95. Purmort, William R., Jr., Newberry, Mich.
96. Ransone, John Taylor, Kecoughtan, Va.
97. Regan, John Francis, Howard, R. I.
98. Rehn, Adolph T., Lapeer, Mich.

99. Rennell, Edwin J., Traverse City, Mich.
100. Reynolds, Chester L., Chicago, Ill.
101. Rheingold, Joseph C., Chicago, Ill.
102. Robinson, G. Wilse, Jr., Kansas City, Mo.
103. Roeling, George F., New Orleans, La.
104. Rogers, William D., Chattahoochee, Fla.
105. Romano, John, Boston, Mass.
106. Rose, John Andrew, Ithaca, N. Y.
107. Ross, Mabel, Baltimore, Md.
108. Rubin, Emanuel, Canandaigua, N. Y.
109. Rubinstein, Hyman S., Baltimore, Md.
110. Rudnick, Julius, New York, N. Y.
111. Sauriol, Louis E., N. Westminster, B. C., Canada.
112. Savitt, Robert A., Queens Valley, N. Y.
113. Sawyer, George J., Jr., Baltimore, Md.
114. Saxe, Louis J., Phœnix, Ariz.
115. Schiller, Maurice A., Chicago, Ill.
116. Semrad, Elvin V., Waltham, Mass.
117. Serrurier, Laurence R., Portland, Ore.
118. Shapiro, Louis B., Elgin, Ill.
119. Shirley, Hale Foreman, Iowa City, Iowa.
120. Simon, Alexander, Washington, D. C.
121. Smith, Beverley E., Pinebluff, N. C.
122. Smith, Herbert P., St. Louis, Mo.
123. Solomon, Joseph C., Baltimore, Md.
124. Stadtherr, Edward F., San Francisco, Calif.
125. Steele, Ivan Woodward, Lexington, Ky.
126. Steer, John C. D., New Toronto, Ont., Canada.
127. Steinberg, D. Louis, Elgin, Ill.
128. Stone, Leo, New York, N. Y.
129. Stricklet, Edward J., Manila, P. I. (Lt. Col.).
130. Summers, William R., Lakeland, Ky.
131. Talkington, Perry C., Dallas, Texas.
132. Tillim, Sidney J., Amityville, L. I., N. Y.
133. Tisdale, Clifford V., Woodstock, Ont., Canada.
134. Trefousse, George, Staten Island, N. Y.
135. Turner, John W., Sunmount, N. Y.
136. Tutunjian, Khacher H., Manteno, Ill.
137. Wall, Conrad, Worcester, Mass.
138. Warren, Victor C., Washington, D. C.
139. Werner, Henry C., Fond du Lac, Wisc.
140. Wharton, Russell S., N. Little Rock, Ark.
141. Wheeler, Morris B., Rusk, Texas.
142. Whitehead, Duncan, Utica, N. Y.
143. Williams, James Newton, Richmond, Va.
144. Wilson, Jewell R., Bolivar, Tenn.

145. Wolfe, Paul S., Pueblo, Colo.
146. Wolfsohn, Leo, Poughkeepsie, N. Y.
147. Wortis, Joseph, New York, N. Y.
148. Young, Bascom B., Wingdale, N. Y.
149. Young, William G., Hartford, Conn.
150. Zeifert, Mark, Brooklyn, N. Y.

TRANSFER FROM ASSOCIATE TO MEMBER.

1. Barnes, Janet S., E. Gardner, Mass.
2. Barrett, William G., Boston, Mass.
3. Brown, Robert W., Rochester, N. Y.
4. Cameron, Norman, New York, N. Y.
5. Carlson, Carroll C., Tacoma, Wash.
6. Carter, Harold R., Washington, D. C.
7. Duffy, Raymond J., Boston, Mass.
8. Duvall, Albert Ing, Marlboro, N. J.
9. Fellows, Ralph M., Osawatomie, Kans.
10. Glauber, Israel P., New York, N. Y.
11. Guiglia, Alphonso, Louisville, Ky.
12. Hecker, Arthur O., Woodville, Pa.
13. Keiser, Sylvan, New York, N. Y.
14. Lewis, H. Ryle, Hartford, Conn.
15. Matthaei, Pearl V., San Antonio, Texas.
16. Miller, Charles, Jr., Medical Lake, Wash.
17. Orenstein, Leo L., New York, N. Y.
18. Pearson, Grosvenor B., Foxborough, Mass.
19. Phillips, Stonewall Jackson, Pineville, La.
20. Porter, Marshall E., Mendocino, Calif.
21. Reider, Norman, Topeka, Kans.
22. Roback, Harry N., Medical Lake, Wash.
23. Rogers, Lieuen M., Lexington, Ky.
24. Rosenberg, Seymour J., Perry Point, Md.
25. Sugar, Carl, Welfare Island, N. Y.
26. Thorner, Melvin W., Philadelphia, Pa.
27. Titley, William B., New York, N. Y.
28. Wadsworth, Morton L., Brooklyn, N. Y.
29. Watters, Theodore A., New Orleans, La.
30. Webb, Robert W., Dallas, Tex.
31. Wolff, Harold G., New York, N. Y.
32. Wortis, Herman, New York, N. Y.
33. Yost, Orin R., Hartford, Conn.

TRANSFER FROM MEMBER TO FELLOW.

1. Bloomberg, Wilfred, Boston, Mass.
2. Braceland, Francis J., Philadelphia, Pa.

3. Chassell, Joseph Olin, Towson, Md.
4. Davidoff, Eugene, Syracuse, N. Y.
5. Fitzpatrick, Charles P., Providence, R. I.
6. Hartwell, Samuel W., Buffalo, N. Y.
7. Mc Cuaig, Charles H., Toronto, Ont., Canada.
8. Mc Niel, Edwin E., Honolulu, T. H.
9. Mac Kay, Robert W. M., Dartmouth, N. S., Canada.
10. Milici, Pompeo S., Kings Park, L. I., N. Y.
11. Murdock, Harry M., Towson, Md.
12. O'Brien, Frank J., New York, N. Y.
13. Saul, Leon J., Chicago, Ill.
14. Van Ophuijsen, Johan H. W., New York, N. Y.

REINSTATEMENTS.

1. Alpers, Jacob J., Spencer, W. Va.
2. Freeman, Walter, Washington, D. C.
3. Fuller, Justin K., Washington, D. C.
4. Odom, Cleve C. (Lt. Col.), Corozal, Canal Zone.
5. Peddicord, Frank L., Lakeland, Ky.

PRESIDENT HUTCHINGS.—The Secretary has cast the ballot, and I declare these members elected.

The meeting adjourned at nine-forty o'clock.

FRIDAY MORNING SESSION.

MAY 12, 1939.

The meeting convened at nine-forty o'clock, President Hutchings presiding.

PRESIDENT HUTCHINGS.—The first order of business is a short report by the Secretary.

Secretary Ruggles read the report of the Council.

SECRETARY RUGGLES.—The Council met at the Palmer House at 4:30 p. m., Thursday, May 11, 1939. Dr. Hutchings, the President, presided. There were nine members present.

Dr. Adolf Meyer has requested that he not be renominated as a Member of the American Board of Psychiatry and Neurology from the American Psychiatric Association. It was moved, seconded and voted to nominate Dr. Nolan D. C. Lewis in Dr. Meyer's place and to renominate Dr. C. Macfie Campbell, Dr. Franklin G. Ebaugh and Dr. Edward A. Strecker.

The nomination of Dr. Ross McC. Chapman and Dr. Arthur P. Noyes as members of the Executive Committee for the ensuing year to serve together with the President, President-Elect and Secretary-Treasurer. Moved, seconded and voted.

A communication by Dr. John N. Frederick read suggesting that the meeting might be improved by the use of lapel microphones or flash light pointers or both. This matter was referred to the Executive Assistant for his disposition.

The name of Dr. Arthur P. Noyes was placed for nomination to succeed Dr. Edward A. Strecker on the Board of Examiners for five years. This nomination was moved, seconded and voted.

The Secretary reported the election of the following Section officers for the ensuing year:

Section on Convulsive Disorders:
David C. Wilson, M. D., Chairman, Medical School, Va.
Albert W. Pigott, M. D., Secretary, Skillman, N. J.

Section on Forensic Psychiatry:
James L. McCartney, M. D., Chairman, Philadelphia, Pa.
Lowell S. Selling, M. D., Secretary, Detroit, Mich.

Section on Mental Deficiency:
E. P. Lewis, M. D., Chairman, Toronto, Canada.
Oscar J. Raeder, M. D., Secretary, Boston, Mass.

Section on Psychoanalysis:
Clarence P. Oberndorf, M. D., Chairman, New York, N. Y.
Flanders Dunbar, M. D., Secretary, New York, N. Y.

The retiring President, Dr. Richard H. Hutchings, paid tribute to the work during this meeting of the Executive Assistant, Austin M. Davies. It was then moved by Dr. Charles H. Dolloff and seconded by Dr. George S. Johnson that the salary of the Executive Assistant be increased from $3600 to $4000 beginning May 1st. This was voted.

The meeting adjourned at 5.15 p. m.

PRESIDENT HUTCHINGS.—You have heard the report of the Secretary. What is your wish? Shall it be received and placed on file?

DR. KLOPP.—I so move.

The motion was seconded, was put to a vote and carried.

The business session adjourned at nine forty-five o'clock, and reconvened immediately following the scientific session at twelve-ten o'clock.

PRESIDENT HUTCHINGS.—We have a pleasant ceremony before us, that of hearing the report of the Committee on Resolutions. I believe Dr. Myers is to present it.

Dr. Glenn Myers read the report of the Resolutions Committee.

PRESIDENT HUTCHINGS.—What is your pleasure with reference to the report of the Committee on Resolutions?

DR. CHENEY.—I move the adoption of the report.

The motion was seconded, was put to a vote and carried.

PRESIDENT HUTCHINGS.—I wish to express my thanks to the Committee for its care in preparing this report.

The Chair is looking over the room trying to see if he can see two muscular and able-bodied Pennsylvanians to call into service. I think I see Dr. Maeder and Dr. Petry of that state, and I would ask them to locate the President-Elect and accompany him to the front of the room.

Drs. Maeder and Petry escorted President-Elect Sandy to the platform.

PRESIDENT HUTCHINGS.—I have yet another task to be performed by a Pennsylvanian. If Dr. Klopp is present, will he kindly introduce his colleague, Dr. Sandy?

DR. KLOPP.—We consider it not only a personal honor but an extreme pleasure to present for induction into office as president, a man whom we have known in his official capacity since December, 1921 when he was appointed the first Director of the Bureau of Mental Health of the Pennsylvania Department of Welfare. It is our opinion that the incoming president has all the background and experience which a man occupying the said office should have; not only from the academic and medical standpoint but also that of the specialty of psychiatry, he having given his best years to this branch of medicine, dating from 1902, this covering institutional, administrative, and field survey activities as well as building programs.

He started at the foot of the ladder as assistant physician and has held advancing positions in state institutions in New Jersey, New York, South Carolina, Connecticut and lastly Pennsylvania. During the World War he was commissioned in the Medical Corps of the U. S. Army, office of the Surgeon General of the Army, Division of Neurology and Psychiatry. He now holds the rank of Lieutenant Colonel, Medical Reserve Corps, U. S. Army.

Following the World War he became the psychiatrist of the New York State Commission for Mental Defectives and in this capacity organized the first system of clinics for mental defectives in New York State. He continued in this position until his appointment as Director of the Bureau of Mental Health, Pennsylvania Department of Welfare, in 1921. In this capacity he has been a factor in the advancement of psychiatry in Pennsylvania and the development of the standards of all its mental hospitals. In addition to promoting the formulation of laws in behalf of said mental hospitals, he initiated interest in and the planning of the institution for Epileptics, the Selinsgrove State Colony and that of the Western State Psychiatric Hospital at Pittsburgh. He also initiated interest in the Penn-

sylvania Institution for Male Defective Delinquents. He participated in the drive for complete State care and abolishing of County care. He has promoted and participated in State-wide mental clinics, that of occupational and music therapy in our state and county institutions. He has assisted in establishing and maintaining high standards for professional personnel for the various institutions for mental patients. He is a member of County, State and National Medical Societies, especially those of Psychiatry and Mental Hygiene. He is the author of numerous articles dealing with mental diseases and the different phases of mental hygiene, published in various medical and other journals.

You have known the man who is about to occupy the chair of President in the capacity of Secretary-Treasurer of this Association from 1933 to 1938 and also as President-Elect. You know his capacity for work and his ability as an executive. We have every reason to anticipate and know that he will fill the office of President with credit to himself and above all, this association. We, therefore, take great pleasure in presenting to you, Mr. President, Dr. William C. Sandy for induction into office of President which you have so ably and creditably filled and are about to relinquish.

PRESIDENT HUTCHINGS.—Dr. Klopp, we thank you for the fine account you have given us of our new President. We agree heartily with all you have said.

Dr. Sandy, the Association congratulates you upon the splendid record which you have achieved as a member and Fellow of this Association. It has watched your career with great interest. Five years ago you were elevated to this important office of Secretary, which you have fulfilled with the highest satisfaction of the membership. So that greater rewards were deemed earned by you and you have now reached a point where it is my pleasure, and in obedience to the mandates of The American Psychiatric Association, to induct you into this office of President. So with great satisfaction and pleasure, it is my privilege to hand you this gavel as a token of your high office.

President Sandy assumed the chair.

PRESIDENT SANDY.—*Dr. Hutchings, Dr. Klopp, Members of the Association, Ladies and Gentlemen:* At this time it is quite natural that there is an emotional reaction which makes it really difficult to express adequately my appreciation of being elevated to this high office at your command. My activities with the Association have been among the most satisfactory of my professional experiences. It has been stimulating and interesting to pass through the various chairs, so to speak. As Dr. Klopp and Dr. Hutchings have pointed out, I was a member of the Committee and Committee Chairman, and it was in 1933 when you saw fit to entrust me with the responsibilities of the office of Secretary and Treasurer, which I had the honor of holding until the end of the 1938 session in San Francisco. The past year as President-Elect has been one of lessened responsibility and a time for con-

templation. This occasion has really seemed quite remote but now there is no question as to the reality. I must confess that I have no false impression that it will be easy to reach the level of efficiency of my distinguished predecessors in conducting the affairs of this Association. I shall, however, give my very best efforts to carry on and will lean heavily on the officers, the chairmen of the committees, Mr. Davies, and I hope also to have the stimulating cooperation of the membership of the Association.

We shall adopt as an objective the further growth and progressive development of the Association as a professional group, with the ultimate good for all phases of psychiatry I hope. I thank you very deeply for the honor you have conferred upon me.

I now have a very pleasant duty to perform. As you all know, we cannot really add to the laurels of Dr. Hutchings; in behalf of the Association, we do wish to provide him with a medal, a honor mark of distinction, of service, so to speak, the past president's badge. With Dr. Hutchings' permission, I would like to have the honor of pinning this on his lapel.

RETIRING PRESIDENT HUTCHINGS.—Thank you, Dr. Sandy, and thank you, Fellows and Members of The American Psychiatric Association. I shall treasure this memento of my term as President and cherish the highest and best memories of the year. It has been successful because of the fine cooperation which I received from all who have been in any way associated with its management. I wish to thank one and all for the cooperation and help which I have had. Thank you very much!

PRESIDENT SANDY.—Is there any further business, Mr. Secretary?

SECRETARY RUGGLES.—There is none.

PRESIDENT SANDY.—I wish to remind those present that the ninety-sixth annual meeting will be held in Quebec, Canada, June 10 to 14, 1940. I hope that all those who can be present will come. It is a most romantic spot. It makes it international, and we hope we will have a large attendance.

There being no further business, it becomes my duty to hereby declare the ninety-fifth annual meeting of The American Psychiatric Association adjourned.

The meeting adjourned at twelve-thirty o'clock.

The total registration was 1402, of which 574 were members.

ARTHUR H. RUGGLES, M. D., *Secretary-Treasurer.*

PROCEEDINGS SCIENTIFIC SESSIONS

OF THE

NINETY-FIFTH ANNUAL MEETING

OF THE

AMERICAN PSYCHIATRIC ASSOCIATION.

MAY 8-12, 1939, CHICAGO, ILLINOIS.

JOINT SESSION OF THE SECTION ON CONVULSIVE DISORDERS AND THE AMERICAN CHAPTER OF THE INTERNATIONAL LEAGUE AGAINST EPILEPSY.

MONDAY MORNING SESSION, MAY 8, 1939.

Dr. Douglas A. Thom presiding.

The Control of Normal and "Convulsive" Brain Potentials, by Dr. Ralph W. Gerard.

Experimental Studies on the Convulsive Mechanisms in Hypoparathyroidism and Hyperinsulinism, by Drs. Irvine McQuarrie and Mildred R. Ziegler.

Carbon Dioxide and Epilepsy, by Drs. E. L. Gibbs, W. G. Lennox and F. A. Gibbs. Discussion by Drs. E. Gellhorn, Irvine McQuarrie, W. G. Lennox, Temple Fay, Wilder Penfield and Ralph W. Gerard (closing).

The Spread of the Epileptic Discharge, by Dr. Theodore C. Erickson. Discussion by Drs. Stanley Cobb, Ralph W. Gerard and Theodore C. Erickson (closing).

Constitutional Differences between Deteriorated and Non-Deteriorated Patients with Epilepsy, with Special Reference to Friction Skin Patterns and Nail Fold Capillaries, by Drs. Meyer Brown and Harry A. Paskind. Discussion by Drs. W. G. Lennox and Meyer Brown.

MONDAY AFTERNOON SESSION, MAY 8, 1939.

Dr. Irvine McQuarrie presiding.

Further Experiences with the Use of Sodium Diphenyl Hydantoinate in the Treatment of Convulsive Disorders, by Drs. H. Houston Merritt and Tracy J. Putnam.

Preliminary Report on the Comparative Effects of Phenobarbital and Dilantin in the Treatment of Epilepsy, by Drs. Leon J. Robinson and Rudolf Osgood. Discussion by Drs. Joseph L. Fetterman, W. G. Lennox, L. Erwin Wexberg, J. H. Conn, Jack Weinberg, Charles V. Taylor, H. Houston Merritt (closing), and Leon J. Robinson (closing).

Psychiatric Approach to the Treatment of Epilepsy, by Drs. Stanley Cobb. Discussion by Dr. Adolf Meyer.

Important Structural Inadequacies in the "Idiopathic" Epileptic, with the Results of Treatment of Certain Types, by Drs. Temple Fay and Michael Scott. Discussion by Dr. W. G. Lennox.

Institutional Epileptics—Neurological and Roentgenological Studies, by Drs. S. M. Weingrow, T. S. P. Fitch and A. W. Pigott.

Metrazol as a Diagnostic Agent in Epilepsy, by Professor Laszlo von Meduna.

Election of Section Officers.

Resolution re Research on Epilepsy.

MONDAY EVENING SESSION, MAY 8, 1939.

Dr. David C. Wilson presiding.

Panel Discussion—Round Table.

The evening was devoted to the many leading questions obtained through a questionnaire.

SECTION ON FORENSIC PSYCHIATRY, MONDAY MORNING SESSION, MAY 8, 1939.

Dr. Frank J. O'Brien and Dr. LeRoy M. A. Maeder, presiding.

The threat of Institutionalization, by Dr. Harold B. Hanson. Discussion by Drs. Hyman S. Lippman, Conrad S. Commer, R. L. Jenkins, Clarence P. Oberndorf, and Harold B. Hanson (closing).

The Truancy School, by Dr. Sara G. Geiger. Discussion by Drs. Frank J. O'Brien, Louis A. Lurie, Frank Curran and Sara G. Geiger (closing).

The Juvenile Court Detention Home, by Dr. George M. Lott. Discussion by Drs. Milton E. Kirkpatrick, Louis A. Lurie, Florence Clothier, George M. Lott (closing), Robert H. Haskell and Oscar J. Raeder.

The Training School, by Dr. Manly B. Root. Discussion by Drs. S. W. Hartwell, R. L. Jenkins, C. B. Horton.

MONDAY AFTERNOON SESSION, MAY 8, 1939.

Dr. Paul L. Schroeder presiding.

The Period of Observation in the Psychiatric Court Clinic, by Dr. Charles B. Thompson. Discussion by Drs. Paul L. Schroeder and Dr. Charles B. Thompson (closing).

The Short Term Prisoner, by Dr. Lowell S. Selling. Discussion by Drs. Clarke W. Magun, E. J. Carroll and Lowell S. Selling (closing).

The Incarcerated Psychopath, by Dr. Marion R. King. Discussion by Drs. Paul L. Schroeder, Royal G. Grossman, J. H. Cowan and Marion R. King (closing).

Psychiatry in Correctional Institutions, by Dr. V. C. Branham. Discussion by Dr. David Philips.

The Long Term Prisoner, by Dr. Marvin Sukov. Discussion by Drs. Karl Bowman, B. R. Webster, Andrew H. Woods, E. Wexberg and Marvin Sukov (closing).

SECTION I, TUESDAY AFTERNOON SESSION, MAY 9, 1939.

President Hutchings presiding.

The Desiderata of Central Administrative Control of State Mental Hospitals, by Dr. Winfred Overholser.

The Teaching and Research Functions of State Hospitals, by Drs. Franklin G. Ebaugh and Charles A. Rymer.

Trends in the Activities of Mental Hospitals, by Drs. Samuel W. Hamilton and Grover A. Kempf.

Discussion by Drs. Ross McC. Chapman, Neil A. Dayton, Roy D. Halloran, C. M. Hincks, Nolan D. C. Lewis, Hugo Mella, L. M. Rogers and M. A. Tarumianz.

JOINT SESSION WITH THE SECTION ON PSYCHOANALYSIS AND THE AMERICAN PSYCHOANALYTIC ASSOCIATION, WEDNESDAY MORNING SESSION, MAY 10, 1939.

President Hutchings at first presiding, later Dr. Nolan D. C. Lewis.

Appointment of Nominating Committee for Section on Psychoanalysis.

Results of Psychoanalytic Therapy in a Mental Hospital, by Dr. Joseph Chassell. Discussion by Drs. Dexter M. Bullard, Robert P. Knight and Ernst Simmel.

Somatic Suicide, Total and Partial, by Dr. Karl A. Menninger. Discussion by Drs. Nolan D. C. Lewis, Franz Alexander, Karl A. Menninger (closing).

Analytical Study of a "Cure" at Lourdes, by Dr. Smiley Blanton. Discussion by Drs. Franz Alexander, Karl A. Menninger, Nolan D. C. Lewis, David M. Levy, Smiley Blanton (closing).

Election of Section Officers.

Business Meeting.

WEDNESDAY AFTERNOON SESSION, MAY 10, 1939.

President-Elect Sandy presiding.

Historical Reports of the Mass Psychoses. by Drs. E. Messinger and M. K. Amdur. Discussion by Dr. Gregory Zilboorg.

Social Psychiatry—Our Task or a New Profession, by Dr. Samuel W. Hartwell. Discussion by Miss Katherine Moore Wickman.

Psychiatric Needs in Industry, by Dr. William A. Sawyer. Discussion by Drs. Lydia G. Giberson and C. Charles Burlingame.

The Sociological Implications of Neuroses, by Dr. Paul Schilder. Discussion by Drs. Sandor Rado and Paul Schilder (closing).

The Psychiatric Findings in the Cases of Five Hundred Traffic Offenders and Accident-Prone Drivers, by Dr. Lowell S. Selling. Discussion by Joseph L. Gilbert and Lowell S. Selling (closing).

THURSDAY MORNING SESSION, MAY 11, 1939.

Dr. John R. Ross presiding.

Results and Observations on the Insulin-Shock Treatment of Schizophrenia, by Drs. Earl D. Bond, Joseph Hughes and James A. Flaherty.

An Objective Evaluation of Metrazol Therapy: A Rorschach Study," by Drs. Eugene Eisner and William D. Orbison.

A Fatal Emotional Delirium Induced by Convulsive Therapy, with Neuropathologic Study, by Drs. A. W. Hackfield and Gale Wilson.

Fatal Complication Following Metrazol Treatment for Schizophrenia, by Drs. J. Pessin and H. H. Reese.

The Histopathology of the Brain Following Insulin and Metrazol Injections into Rabbits—A Comparative Study, by Drs. Arthur Weil and Erich Liebert.

The Effect of Insulin on the Central Nervous System in Experimental Animals, by Drs. B. J. Alpers, K. E. Appel, Joseph Hughes and D. W. Hastings.

A Comparative Study of the Development of the Clinical, Electroencephalographic and Biochemical Changes During Insulin Hypoglycemia, by Drs. H. E. Himwich, J. P. Frostig and J. F. Fazekas.

Psychological Performance Tests as Prognostic Agents for Insulin and Metrazol Therapy in Schizophrenia, by Drs. Carney Landis, Joseph Zubin and M. Majorie Bolles.

Electrocardiographic Observations in Convulsive and Nitrogen Therapies, by Drs. Edward A. Strecker, James A. Flaherty, Howard Rome and William Zintl.

Evaluation of Therapeutic Factors in Pharmacologic Shock, by Drs. L. S. Lipschutz, R. W. Cavell, R. Leiser, E. N. Hinko and S. H. Ruskin.

Metrazol Treatment of Dementia Præcox—Preliminary Report, by Drs. Henry O. Colomb and George L. Wadsworth.

The Results of Shock Therapy in the Treatment of Affective Disorders, by Dr. David C. Wilson.

THURSDAY AFTERNOON SESSION, MAY 11, 1939.

Dr. Samuel W. Hamilton presiding.

A Comparative Study of Hypoglycemic Shock Treatment and Control Observation in Schizophrenia, by Drs. J. Notkin, C. E. Niles, F. J. DeNatale and G. Wittman.

Use of Metrazol in the Treatment of Acute Alcoholism, by Drs. Leo L. Orenstein, Karl M. Bowman, Julia R. Kagan and Walter Goldfarb.

Regarding Sodium Amytal as a Prognostic Aid in Insulin and Metrazol Shock Therapy of Mental Patients, by Drs. M. M. Harris, William A. Horwitz and E. A. Milch.

Investigations and Hypotheses Regarding Pharmacotherapy in Schizophrenia, by Drs. Ladislas Meduna and Bela Rohny.

A Review of the Results of the Pharmacological Shock Therapy and the Metrazol Convulsive Therapy in New York State, by Drs. John R. Ross and Benjamin Malzberg.

Vertebral Fractures as a Complication of Convulsions in Hypoglycemic Shock and Metrazol Therapy in Psychiatry Disorders, by Drs. Phillip Polatin, Murray M. Friedman, Meyer M. Harris and William A. Horwitz.

Discussion by Drs. Ralph Fellows, Charles P. Fitzpatrick, James P. Case, Manfred Sakel, Clarence O. Cheney, Joseph Epstein, Eugene Ziskind, E. Gellhorn and Julius Steinfeld.

FRIDAY MORNING SESSION, MAY 12, 1939.

President Hutchings presiding.

Anorexia Nervosa, by Drs. H. W. Brosin and Walter L. Palmer. Discussion by Drs. Burtrum C. Schiele, W. W. Young, Benjamin Erps, H. S. Ripley, Jr. and H. W. Brosin (closing).

Prognostic Criteria in Dementia Paralytica, by Drs. Maurice H. Greenhill and Morris Yorshis. Discussion by Drs. Joseph R. Blalock and Will Rebec.

The Non-Criminal Neurotic Character, by Dr. Gregory Zilboorg—read by title.

The Total Push Method of Treatment of Chronic Schizophrenia, by Drs. Abraham Myerson and Kenneth J. Tillotson. Discussion by Drs. Clarence O. Cheney, Harry Stack Sullivan, Ira M. Altshuler, Richard H. Hutchings and Kenneth J. Tillotson (closing).

Psychoneurotic Manifestations Associated with Somatic Dysfunction, by Drs. William E. Gardner and William K. Keller. Discussion by Drs. William S. Sadler and Solomon Katzenelbogen.

SECTION II. CLINICAL STUDIES, TUESDAY AFTERNOON SESSION, MAY 9, 1939.

Dr. William C. Sandy, President-Elect, presiding.

A Study of Drugs as a Factor in the Etiology of Mental Disease, by Dr. Merrill Moore and M. G. Gray, Ph.D. Discussion by Drs. Frank J. Curran, Glenn E. Myers and Merrill Moore (closing).

Psychoses with Myxedema, by Drs. Ralph M. Crowley. Discussion by Drs. C. Macfie Campbell and Ralph M. Crowley (closing).

A survey of Mental Illness Associated with Pregnancy and Childbirth, by Dr. John L. Smalldon. Discussion by Drs. Grace Baker, Gregory Zilboorg, Jacob Kasanin and John L. Smalldon (closing).

Aggression in Schizophrenia, by Drs. Hugh M. Galbraith and Gregory Zilboorg. Discussion by Drs. Leo H. Bartemeier, A. W. Hackfield and Gregory Zilboorg (closing).

The Language of Phantasy, by Eva Ruth Balken, Ph.D. and Dr. Jules H. Masserman. Discussion by Drs. Friz Kollenhoff, Harry Stack Sullivan and Jules H. Masserman (closing).

WEDNESDAY MORNING SESSION, MAY 10, 1939.

Dr. William C. Sandy, President-Elect, presiding.

Sensitivity, by Drs. Eugen Kahn and Helen G. Richter. Discussion by Drs. John C. Whitehorn and Eugen Kahn (closing).

Early Schizophrenia and its Relationship to the Neuroses, by Dr. W. R. Miller. Discussion by Drs. D. Ewen Cameron and W. R. Miller (closing).

Affective States and Gastric Function: Experimental Studies on Patients with Peptic Ulcer, by Drs. Bela Mittelmann and Harold G. Wolff. Discussion by Drs. George S. Johnson and Roy G. Hoskins, William Nelson and Bela Mittelman (closing).

Psychopathological Disorders in the Mother: A Problem in Child Psychiatry, by Dr. Mabel Huschka. Discussion by Drs. Hyman S. Lippman, Thomas M. French and Mabel Huschka (closing).

Dementia Paralytica Accompanied by Manic-Depressive and Schizophrenic Psychoses: The Significance of their Co-Existence, by Dr. David Rothschild. Discussion by Drs. Clarence A. Neymann and David Rothschild (closing).

JOINT SESSION OF THE SECTION ON PSYCHOANALYSIS AND THE AMERICAN PSYCHOANALYTIC ASSOCIATION, WEDNESDAY AFTERNOON SESSION, MAY 10, 1939.

Dr. Samuel W. Hamilton presiding.

Developments in the Psychoanalytic Conception and Treatment of the Neuroses, by Dr. Sandor Rado. Discussion by Drs. John A. P. Millet, Bela Mittelmann, Thomas M. French, M. Ralph Kaufman and Sandor Rado.

Utilization of Early Current Dreams in Formulating Psychoanalytic Cases, by Dr. Leon J. Saul. Discussion by Drs. George E. Daniels, Bela Mittelmann, Sandor Lorand and Leon J. Saul (closing).

The Counter-Transference, by Dr. Robert Fliess. Discussion by Drs. Otto Fenichel, Thomas M. French and Robert Fleiss (closing).

Psychoanalytic Interpretation of a Court Case of Incendiarism, by Dr. Ernst Simmel. Discussion by Drs. Robert Fliess, George M. Lott and Ernst Simmel (closing).

ON EXTRA MURAL STUDIES. THURSDAY MORNING SESSION, MAY 11, 1939.

Dr. S. W. Hartwell presiding.

Association of the Internist and Psychiatrist in Private Practice, by Drs. Clarke H. Barnacle and Ward Darley. Discussion by Drs. Flanders Dunbar, A. E. Bennett, Franklin G. Ebaugh and Ward Darley (closing).

The Associations of Former Patients in the Mental Hospitals of Illinois: A New Approach in After Care, by Drs. Abraham A. Low. Discussion by Drs. Louis Wender, Paul Schilder and Abraham A. Low (closing).

The Post-Hospital Adaptation of a Group of Selected Cases of Dementia Præcox: A Preliminary Report, by Drs. Jay L. Hoffman, Ernest H. Parsons and Margaret Hagan, B. A. Discussion by Drs. Arthur P. Noyes and Jay L. Hoffman (closing).

An Evaluation of the First Year of a Parole Clinic, by Dr. Martin H. Hoffmann. Discussion by Drs. Ira A. Darling and Martin H. Hoffman (closing).

Treatment in the Psychiatric Outpatient Department of a General Hospital, by Dr. O. Spurgeon English. Discussion by Drs. Edward G. Billings, Lauren H. Smith, O. Spurgeon English (closing), Temple Fay, S. W. Hartwell.

JOINT SESSION WITH THE SECTION ON MENTAL DEFICIENCY, THURSDAY AFTERNOON, MAY 11, 1939.

Dr. C. C. Kirk presiding.

Appointment of Nominating Committee.

Psychiatric Problems in Diabetic Children, by Drs. Hale F. Shirley and Ina M. Greer. Discussion by Drs. Priscilla White, William Keller, Elizabeth Kane and John D. Griffin.

Behavior Differences in Mentally Retarded Children Measured by a New Behavior Rating Scale, by Dr. Alfred A. Strauss and Newell C. Kephart, Ph.D. Discussion by Dr. Jacob Kasanin.

Psychotic Behavior in Children, by Dr. Martha W. MacDonald. Discussion by Drs. William Malamud, Frank J. O'Brien, Louis A. Lurie, Jacob Kasanin, J. M. Murdock, Charles S. Raymond, C. C. Kirk and Martha W. MacDonald (closing).

Prognosis in Post Encephalitic Behavior Disorder; A Preliminary Report, by Dr. Philip H. Heersema. Discussion by Drs. Leslie B. Hohman, Lauren Smith, William Holt and Lloyd J. Thompson.

SECTION ON MENTAL DEFICIENCY. FRIDAY MORNING, MAY 12, 1939.

Dr. C. C. Kirk presiding.

Syphilis in Serum Negative Feebleminded Children, by Dr. Clemens E. Benda. Discussion by Drs. Adolf Meyer, Samuel Epstein, Oscar J. Raeder, Benjamin Boshes, Clemens E. Benda.

Differentiation of Symptoms from the Point of View of Therapy in Mental Defect and Other Organic Diseases of the Central Nervous System, by Dr. Hedda Bolgar. Discussion by Drs. A. W. Hackfield, Alfred A. Strauss, Paul L. Schroeder, Benjamin Boshes, C. C. Kirk and Hedda Bolgar (closing).

Additional papers read by title:

The Predelinquent Child and His Contribution to Crime, by Dr. Douglas A. Thom.

Vagotonic Hypostasis in the Neuroses, by Dr. Baldwin L. Keyes.

SECTION III. STUDIES FROM THE LABORATORY. TUESDAY AFTERNOON SESSION, MAY 9, 1939.

Dr. Clarence A. Neymann presiding.

Detoxication in Some Neuropsychiatric Disorders, by Drs. Finkelman, Hora, Sherman and Horwitt. Discussion by Drs. Edwin F. Gildea, Arthur Weil, Roy G. Hoskins, M. K. Horwitt, Edwin F. Gildea, and Isidore Finkelman (closing).

Electro-encephalography in Schizophrenia: Localization of Atrophy in the Cerebral Cortex, by Dr. Morton A. Rubin. Discussion by Drs. William G. Lennox, Oskar Diethelm, Ira C. Nichols and Morton A. Rubin (closing).

Long Term Studies of the Variations in Serum Proteins, Non-Protein Nitrogen and Lipoids in Patients with Schizophrenic and Manic Depressive Psychoses and in Normal People, by Dr. Edwin F. Gildea. Discussion by Seymour D. Ludlum, Roy G. Hoskins and Edwin F. Gildea (closing).

Mutism and Resistance in Psychotic Patients: A Physiologic Study, by Drs. Alfred P. Solomon and Chester W. Darrow. Discussion by Drs. Oskar Diethelm, Seymour D. Ludlum, Alfred P. Solomon (closing) and Chester W. Darrow (closing).

The Pharmacology of the Analeptic Drugs with Especial Reference to Their Effect in Hibernating Ground Squirrels, by Drs. Carl C. Pfeiffer and David Slight. Discussion by Drs. Eugene Davidoff, Temple Fay, L. Maude Warren and David Slight (closing).

The Illuminable Brain Model, by Dr. Edith Klemperer. Discussion by Drs. Walter L. Bruetsch and Edith Klemperer (closing).

ORGANIC BRAIN PATHOLOGY. WEDNESDAY MORNING SESSION, MAY 10, 1939.

Dr. Lloyd H. Ziegler presiding.

The Mental State of Aphasia. (The phenomena of aphasia as the reactions of an unstable mentality to difficult tasks), by Dr. Leland B. Alford. Discussion by Dr. Kurt Goldstein.

Category Behavior and Lesions of the Frontal Lobe in Man, by Dr. Ward C. Halstead. Discussion by Drs. Roy R. Grinker, Spafford Ackerly and Ward C. Halstead (closing).

A Case of Partial Bilateral Frontal Lobectomy—A Psychopathological Report, by Drs. Ira C. Nichols and J. McVicker Hunt. Discussion by Drs. Adolf Meyer, Raymond W. Waggoner, Frederick J. Krueger, Kurt Goldstein and Ira C. Nichols (closing).

Intracranial Tumors in Mental Hospital Population (A Statistical Study), by Dr. Charles P. Larson. Discussion by Drs. Andrew J. Akelaitis, Walter L. Bruetsch, Clemens E. Benda, Arnold W. Hackfield and Charles P. Larson (closing).

Pick's Disease with Temporal Lobe Atrophy, by Drs. N. Malamud and D. A. Boyd, Jr. Discussion by Drs. Naomi Raskin, Eugen Kahn and N. Malamud (closing).

THERAPEUTIC AND CLINICAL STUDIES. WEDNESDAY AFTERNOON SESSION, MAY 10, 1939.

Dr. Franklin G. Ebaugh presiding.

Hypnotic Suggestion: Its Dynamics, Indications, and Limitations in the Therapy of Neuroses, by Dr. Sandor Lorand. Discussion by Drs. Paul Schilder and Sandor Lorand (closing).

Reactive Depression and Involutional Melancholia, by Drs. Henry H. Dixon and G. B. Haugen. Discussion by Drs. Harold F. Corson, Jacob H. Conn and Henry H. Dixon (closing).

The Effect of Treatment of Depression with Estrogenic Hormone, by Dr. Herbert S. Ripley. Discussion by Drs. John Y. Notkin, Titus H. Harris, Henry G. Smith and Herbert S. Ripley (closing).

Therapeutic Quartan Malaria in the Treatment of Neurosyphilis Among Negroes—A Progress Report, by Dr. George C. Branche. Discussion by Drs. Walter L. Bruetsch, John E. Lind and George C. Branche (closing).

A Study of Insight of Psychiatric Patients, by Dr. Carroll W. Osgood. Discussion by Drs. Earl D. Bond, Shelton G. Silverberg, W. B. Cline, Jr. and Carroll W. Osgood (closing).

SPECIAL TESTS. THURSDAY MORNING SESSION, MAY 11, 1939.

Dr. H. Douglas Singer presiding.

The Respiratory Responses of Psychoneurotic Patients to Ideational and to Sensory Stimuli, by Drs. Jacob E. Finesinger and Sarah G. Mazick. Discussion by Drs. Foster Kennedy, Andras Angyal, George E. Daniels, Foster Kennedy and Jacob E. Finesinger (closing).

The Syndrome of Fingeragnosis, Disorientation of Right and Left Agraphia, Acalculia, and its Local Diagnostic Value, by Dr. Josef Gerstmann. Discussion by Dr. Kurt Goldstein.

The Significance of Special Mental Tests for Diagnosis and Prognosis in Schizophrenia, by Dr. Kurt Goldstein. Discussion by Drs. C. Macfie Campbell, Arnold Hackfield, and Kurt Goldstein (closing).

Rorschach's Experiment in Traumatic Mental Disorders, by Dr. Emil Oberholzer. Discussion by Drs. Oskar Diethelm, S. J. Beck and M. R. Harrower.

An Experimental Study of Age Regression by Hypnotic Reorientation, by Dr. Milton H. Erickson. Discussion by Drs. Hans Syz, Lewis B. Hill, Foster Kennedy, Sandor Lorand, Alfred Solomon and Milton H. Erickson.

Autonomic and Respiratory Responses of Schizophrenic and Normal Subjects to Changes of Intra-Pulmonary Atmosphere, by Drs. H. Freemand and E. H. Rodnick. Discussion by Drs. Erich Lindemann, S. Bernard Wortis, Alfred Solomon, Lee W. Darrah and H. Freeman (closing).

MISCELLANEOUS TOPICS. THURSDAY AFTERNOON SESSION, MAY 11, 1939.

Dr. Karl Menninger presiding

Hereditary and Environmental Factors in the Causation of Manic-Depressive Psychoses and Dementia Præcox, by Drs. Horatio M. Pollock and Benjamin Malzberg. Discussion by Drs. William C. Sandy, James K. Hall, Kenneth J. Tillotson and Horatio M. Pollock (closing).

A Critique of Some Trends in Medical Education, by Dr. Foster Kennedy. Discussion by Drs. C. Macfie Campbell, Franklin G. Ebaugh, George McC. Lott and Foster Kennedy (closing).

The Educational and Administrative Problem of the Affiliate Nurse, by Dr. Charles P. Fitzpatrick. Discussion by Drs. George H. Stevenson, John L. Van de Mark, Winfred Overholser, Miss May Kennedy, Miss Marion Faber, Drs. Charles Wordell and Charles P. Fitzpatrick (closing).

Tuberculosis Prevention and Treatment in Ontario Mental Hospitals, by Dr. Clarence A. Wicks. Discussion by Drs. Samuel W. Hamilton, Henry I. Klopp, Joseph R. Blalock, Walter A. Thompson and Clarence A. Wicks (closing).

Children's Reactions to the Discovery of Genital Differences, by Dr. Jacob H. Conn. Discussion by Drs. David Levy, Mabel Huschka, Jules H. Masserman, Hyman S. Lippman, Goldberg and Jacob H. Conn (closing).

ROUND TABLE DISCUSSIONS.

THURSDAY EVENING, MAY 11, 1939.

ADVISORY PSYCHIATRIC SERVICE IN COMMUNITY SOCIAL AGENCIES.

Moderator: Dr. Lloyd J. Thompson.

CHILD PSYCHIATRY—CONCEPTS AND TREATMENT OF JUVENILE DELINQUENCY.

Moderator: Dr. Paul L. Schroeder.

COLLEGE MENTAL HYGIENE.

Moderator: Dr. Theophile Raphael.—Dr. Helen Langner presiding in Dr. Raphael's absence.

FORENSIC PSYCHIATRY: "AFTER PRISON, WHAT?"

Moderator: Dr. R. G. Barrick.

FORMER PATIENT ASSOCIATIONS.

Moderator: Dr. Appleton H. Pierce.

LANGUAGE AND THOUGHT IN SCHIZOPHRENIA.

Moderator: Dr. Jacob Kasanin.

OCCUPATIONAL THERAPY.*

Moderator: Dr. John A. Pritchard.

PHARMACOLOGICAL SHOCK THERAPY.

Moderator: Dr. Solomon Katzenelbogen.

PSYCHIATRIC EDUCATION.

Moderator: Dr. Franklin G. Ebaugh.

PSYCHIATRIC NURSING.

Moderator: Dr. George H. Stevenson.

PSYCHIATRIC SOCIAL SERVICE.

THE TRAINING AND EMPLOYMENT OF PSYCHIATRIC SOCIAL WORKERS IN MENTAL HOSPITALS.

Moderator: Dr. William A. Bryan.—Dr. S. W. Hartwell presiding in Dr. Bryan's absence.

PSYCHIATRY IN INDUSTRY—LEARNING TO LIVE WITH THE JOB.

Moderator: Dr. Lydia G. Giberson.

PSYCHOSOMATIC RELATIONSHIPS.

Moderator: Dr. Flanders Dunbar.

RESEARCH ORGANIZATION.

Moderator: Dr. John C. Whitehorn.

THE INTER-RELATION OF SCHIZOPHRENIA AND MANIC DEPRESSIVE PSYCHOSES: THE SIGNIFICANCE OF THEIR CO-EXISTENCE.

Moderator: Dr. Clarence A. Neymann.

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* A report of this round table will appear in the October, 1939, number of Occupational Therapy and Rehabilitation.

THE RÔLE OF THE PSYCHOANALYST AS AN ADVISER IN SEXUAL AND MARITAL ADJUSTMENT.

Moderator: Dr. Clarence P. Oberndorf.

UNSUCCESSFUL SEX ADJUSTMENT IN MARRIAGE.

Moderator: Dr. S. Bernard Wortis.

VETERANS ADMINISTRATION.

Moderator: Dr. Appleton H. Pierce.

ARTHUR H. RUGGLES, M. D., *Secretary-Treasurer.*

REPORTS OF COMMITTEES.

The following reports of Committees and of the Secretary-Treasurer were presented to the Association and approved by it during the convention sessions in Chicago, Illinois, May 8-12, 1939.

Report of the Executive Committee.

Following the personal investigation of hotel facilities in Chicago by the Executive Committee and the Executive Assistant and upon their recommendation, the Palmer House was selected for the 95th annual meeting and the time May 8-12, 1939.

On December 28, 1938, the Council met at 7.30 p. m., at the Hotel Jefferson, Richmond, Va. Twelve members of the Council were present, the Chairman of the various committees, and the Executive Assistant, Mr. Austin M. Davies, was also in attendance.

Richard H. Hutchings, President, in the chair.

The Executive Committee reported to the Council its approval of the retirement and life insurance plan for the employees of the Association.

Mr. Austin M. Davies, Executive Assistant, gave a report of the activities of his office since the last meeting.

Dr. William E. Gardner, Chairman, reported for the Membership Committee that he felt the Fellows should bring to the attention of the Association the names of promising members who might properly be considered for promotion to fellowship. It was voted that the Secretary send a letter to all fellows asking them to bring to the attention of the Association such possible candidates. The matter of a proper ceremonial for the inauguration of fellows was discussed and it was voted to refer this to the Membership Committee with power to act.

Dr. Overholser, Chairman of the Committee on Legal Aspects of Psychiatry, gave his report and stated some money may be forthcoming for the survey of criminal courts in connection with the American Bar Association. Report accepted.

Dr. C. C. Burlingame, Chairman of the Committee on Public Education, reported and made the suggestion that year round publicity in various regions of the country through qualified members should be considered. Doctor Burlingame also recommended the consideration of proper ceremonial at the time of the inauguration of fellows at the annual meeting. The report was received and approved as a preliminary report.

Dr. Roscoe W. Hall, Chairman of the Program Committee, gave an outline of the work already done and stated this was a report of progress. It was voted that the Program Committee be empowered to decide about the reading of papers by non-members and that they should definitely state on

the program that non-members were reading by courtesy, unless such papers were read upon actual invitation of the Program Committee.

Mr. Davies reported for the Arrangements Committee that the annual meeting was now self-supporting and provides a small balance for the Association.

Dr. Whitehorn reported for the Committee on Research and stated that arrangements had been made for a round table conference on research at the annual meeting.

Dr. Richard H. Hutchings next appointed a committee to recommend a successor to Dr. Allen Jackson on the Membership Committee. The Committee consisted of Dr. C. M. Hincks, Dr. Partlow and Doctor Ruggles, who later recommended the appointment of Dr. D. Ewen Cameron. The recommendation was approved and Doctor Cameron was appointed as a member of the Board of Examiners to succeed Dr. Jackson.

Next followed the discussion of the appointment of Dr. Harry Stack Sullivan to represent the American Psychiatric Association at the meeting of the National Research Council. In the discussion it was determined that Doctor Whitehorn's appointment had not terminated and, therefore, it was moved, seconded and voted that the action of the Council at the San Francisco meeting in May be declared null and void. This was voted and Dr. John C. Whitehorn continued as the representative of the Association at the National Research Council.

Dr. George H. Stevenson, Chairman of the Committee on Psychiatric Nursing presented a preliminary report. A letter from the Secretary of the New England Psychiatric Association asking the American Psychiatric Association to state its position regarding various types of training for the psychiatric nurse was discussed at length. Following the discussion it was voted that the Council recommend to the New England Society of Psychiatry that it does not sanction a standard of psychiatric nursing lower than the standard of the respective states for the R. N. It was also voted that authorization for varying present standards of psychiatric nursing be postponed until further study by the Committee.

The report of the Committee on Psychiatric Social Service was presented by the Chairman, Dr. Samuel Hartwell. Dr. Hartwell requested that additional members should be appointed to this committee and that a sub-committee be authorized to draw up, with representatives of the American Psychiatric Social Workers Association minimum standards for the training of Psychiatric social workers, and be given authority to act for the Association and report back their action at a subsequent meeting of the Council. Dr. Karl Bowman was added to this Committee and the President appointed Doctor Hartwell, Dr. George S. Stevenson and Doctor Bowman to act as such a sub-committee.

Doctor Hutchings presented a letter from the Surgeon-General of the United States Public Health Service suggesting the appointment of a member of the American Psychiatric Association as an official delegate to the annual meeting of the Pan-American Conference to be held at Lima, Peru,

March 25, 1939. It was voted that the President be authorized to appoint any member of the American Psychiatric Association who is going to this meeting.

Meeting adjourned at 11.45 p. m.

On December 28, 1938, at 5.30 p. m., the Executive Committee met at the Hotel Jefferson, Richmond, Va. Richard H. Hutchings, M. D., President, presided. There were present Dr. Ross McC. Chapman, Dr. William C. Sandy and Dr. A. H. Ruggles, Secretary-Treas.

A plan for the proposed retirement and life insurance plan for the employees of the Association in the New York office was presented by Mr. Davies. This was approved by the Executive Committee to be effective immediately, in order that the advantage of a lower rate could be obtained.

The report of the proposed changes in the format of the AMERICAN JOURNAL OF PSYCHIATRY was presented and discussed at some length. Because of the amount of detailed study needed in this matter, it was moved, seconded and voted that this matter be left to a committee consisting of Dr. Farrar, Dr. Sandy, Dr. Ruggles and Mr. Davis, with power to act.

Mr. Davies next presented the question of Miss Rubenstein's salary and recommended an increase of $150, in order that this salary might be in line with the work she is doing and with other salaries of the medical organizations in the New York Academy of Medicine. This was voted.

Next, the matter of one member and two Fellows of the Association whose dues had been remitted by the Action of the Council, was taken up. It was voted that the Secretary should write to these members and state that unless the 1939 dues can be paid the Council must consider dropping their names from the rolls.

Authorization was given the Secretary-Treasurer to have available reporting facilities at future meetings of the Executive Committee and Council. This was approved.

A report was made on the Symposium on Mental Health which was then taking place at the Annual meeting of the American Association for the Advancement of Science. This report was accepted and ordered referred to the Council.

Meeting adjourned at 6.30 p. m., for dinner of the Council.

Meeting of the Council held at 2 p. m., Thursday, December 29.

Report of the Secretary-Treasurer was presented and accepted.

It was voted that the 1940 meeting be held at the Chateau Frontenac, Quebec; the exact date to be decided by the Executive Committee.

Resignation of five members with one year of unpaid dues was received. It was voted that these five members be dropped from membership.

A letter was read from Dr. Rosanoff, who has recently been appointed Director of Institutions in California, asking the American Psychiatric Association to endorse plans submitted by Dr. Rosanoff for the erection of an acute psychiatric unit to be established in San Francisco on the grounds of the University of California Hospital; (2) that a sum be added to the maintenance budget for the next biennium for salaries and traveling expenses

of one hundred field workers. This was discussed at some length and it was voted that the Secretary write a letter expressing the Council's general approval of Doctor Rosanoff's plan for a hospital for early treatment and the development of adequate facilities for community care. The following letter was sent to Governor-Elect Olson of California.

December 30, 1938.

"*Hon. Culbert L. Olson,*
Executive Chambers, State House,
Sacramento, California.

"My Dear Governor:

"The Council of The American Psychiatric Association in executive session on December 29, in Richmond, Va., requested me to congratulate you on the progressive program you plan to develop for the mentally sick in California. They wish me to say that they approve of the establishment of an acute psychiatric unit in San Francisco for the accepted early treatments of beginning mental illness. They also approve of your proposed extension of adequate community care of patients outside of the institutions. The Council of the American Psychiatric Association will be very glad indeed to give you and those entrusted with these responsibilities, any professional advice that may be desired.

"Very truly yours,

"Arthur H. Ruggles, M. D.,
Secretary-Treasurer."

A letter from Mrs. Romaine presented a request that two delegates from The American Psychiatric Association be asked to attend the meeting of the Federal Art Project of the W. P. A. to be held in Washington in February. This matter was discussed and a motion to appoint two members resident in Washington was voted down.

The report of Dr. Franklin G. Ebaugh, Chairman of the Committee on Psychiatry in Medical Education was presented. This included a request for $1200 for the establishment of an institute to be conducted early in 1939 at one of the Western or Southern mental hospital centers. It was voted that general approval be given to this plan but that Doctor Ebaugh be asked to submit further details of estimated cost and choice of location to the Executive Committee, and that the Council give to the Executive Committee authority to act on this matter.

Meeting adjourned.

On April 21, 1939, the Executive Committee met at the office of the Association at 2 East 103rd Street, New York City, all members being present.

Following a discussion of a number of special applications received after March first and presenting certain special reasons why they would like to be admitted to membership or fellowship, it was moved, seconded and voted that a letter be sent to all applicants whose applications were received after

March first stating that the Executive Committee regrets that action cannot take place on their applications until the annual meeting in 1940.

It was next moved and voted that Mr. Austin M. Davies be authorized to pay back assessment of Social Security and also to pay future increases.

It was next moved, seconded and voted that at the 1939 meeting in Chicago, certificates be presented to Fellows elected in 1938 and to those reinstated who are present; and that the President be requested to make it clear at this meeting that in the future certificates are to be presented only to Fellows attending in person, unless exempted from personal attendance by action of the Executive Committee.

After some discussion concerning a proposed Fellowship oath, it was moved, seconded and voted that two copies of the Fellowship oath be sent to applicants for Fellowship; that one of these be signed by the applicant and returned with their signed application and payment for the Fellowship certificate to the office of the A. P. A. and there retained; that the other copy be held by the applicant.

Meeting adjourned at 3.45 p. m.

A meeting was then held of Dr. Chapman's Committee at which the members of the Executive Committee were present.

ARTHUR H. RUGGLES, M. D., *Secretary-Treasurer.*

REPORT OF THE SECRETARY, 1938-39.

The following is a statement of the membership of the American Psychiatric Association as of April 15, 1939:

HONORARY MEMBERS.

Present number		19

LIFE MEMBERS.

Former number	59	
Fellows to Life Members	5	
Total		64
Died		4
Present number		60

CORRESPONDING MEMBERS.

Present number		4

FELLOWS.

Former number	872	
Members to Fellows	11	
Reinstatements	6	
Total		889

Fellows to Life Members	5	
Resigned	4	
Dropped	4	
Died	16	
Total		29
Present number		860

MEMBERS.

Former number	828	
Associate Members to Members	24	
Elected	163	
Reinstatements	1	
Total		1016
Members to Fellows	11	
Resigned	9	
Dropped	4	
Died	7	
Total		31
Present number		985

ASSOCIATE MEMBERS.

Former number	98	
Elected	34	
Total		132
Associate Members to Members	24	
Dropped	3	
Total		27
Present number		105

TOTAL MEMBERSHIP.

Honorary	19	
Life Members	60	
Corresponding Members	4	
Fellows	860	
Members	985	
Associate Members	105	
Total membership April 15, 1939		2,033
Total membership May 15, 1939		1,880

ARTHUR H. RUGGLES, M. D., *Secretary.*

Report of Treasurer.

STATEMENT OF CASH RECEIPTS AND CASH DISBURSEMENTS FOR PERIOD FROM MAY 15, 1938 TO APRIL 14, 1939.

(For Eleven Months.)

Cash Balance—May 15, 1938		$38,404.19
Cash Receipts.		
Membership Dues	$15,243.17	
Fellowship Certificates	660.00	
Exhibits—1938 Convention	135.00	
Exhibits—1939 Convention	1,377.00	
Committee on Arrangements—1938 Convention	172.92	
Interest on Savings Account	506.47	
Total Cash Receipts		18,094.56
		$56,498.75
Cash Disbursements.		
Executive Assistant	$3,300.00	
Clerical Salaries	1,371.25	
San Francisco Special Train Account	$17,132.30	
Less Receipts	7,380.95	
	9,751.35	
John and Mary R. Markle Foundation	2,483.47	
Symposium on Mental Hygiene	$4,941.19	
Less Receipts	4,583.32	
	357.87	
Rent	825.00	
Mental Hospital Survey Committee	363.07	
American Journal of Psychiatry—Subsidy	4,600.00	
Committee Expenses	1,574.96	
Convention Expenses—1938	81.69	
Convention Expenses—1939	389.73	
Reporting—1938 Convention	856.67	
Programs—1938 Convention	546.32	
Directory of Members	710.94	
Insurance and Annuities	$600.19	
Less Deductions	38.25	
	561.94	
Printing	276.67	
Postage	491.17	
Bond—A. M. Davies	50.00	
Accounting	40.00	
General Expense	345.67	
Total Cash Disbursements		28,977.77
Cash Balance, April 14, 1939		$27,520.98

AMERICAN JOURNAL OF PSYCHIATRY.

Statement of Cash Receipts and Cash Disbursements for Period From May 15, 1938 to April 14, 1939.

(For Eleven Months.)

Cash Balance—May 15, 1938		$370.76
Cash Receipts.		
Subscriptions	$8,454.28	
Reprints	1,262.04	
Back Numbers	204.33	
Advertising	1,753.69	
Total Cash Receipts		11,674.34
		$12,045.10
Cash Disbursements		
Printing (Volume 94, No. 4 through Volume 95, No. 3)	$10,687.32	
Editorial Assistance	757.38	
Telephone and Telegraph	137.73	
Postage	94.43	
Mailing Back Numbers	97.16	
Office Supplies	26.65	
Miscellaneous Expenses	54.50	
Total Cash Disbursements		11,855.17
Cash Balance—April 14, 1939		$189.93

SCHEDULE OF CASH BALANCES, APRIL 14, 1939.

	Book Number.	Balance.
Public National Bank		$1,877.44
Bank for Savings	1,324,532	3,635.33
Union Dime Savings Bank	1,115,778	6,621.70
Emigrant Savings Bank	137,048	8,973.53
Bowery Savings Bank	258,266	6,412.98
Total		$27,520.98

TOTAL CASH RESOURCES.

American Psychiatric Association (as above)	$27,520.98
AMERICAN JOURNAL OF PSYCHIATRY	180.93
Total	$27,701.91

ARTHUR H. RUGGLES, M. D., *Treasurer.*

Mr. Austin M. Davies, Executive Assistant, American Psychiatric Association, 2 East 103rd Street, New York, N. Y.

DEAR SIR:

As per your request, I have made an audit of the books and records of The American Psychiatric Association and of the AMERICAN JOURNAL OF PSYCHIATRY for the period of May 15, 1938 to April 14, 1939.

Attached herewith you will find a report containing the following:

1. Statement of Cash Receipts and Cash Disbursements of The American Psychiatric Association for Period from May 15, 1938, to April 14, 1939.
2. Statement of Cash Receipts and Cash Disbursements of the AMERICAN JOURNAL OF PSYCHIATRY for Period from May 15, 1938 to April 14, 1939.
3. Schedule of Cash Balances of The American Psychiatric Association as of April 14, 1939.

Please note that these statements represent the cash transactions only for the period under review. No consideration has been taken for accrued expenses.

In connection with the audit, I added all cash books, reconciled your bank statements, examined all vouchers and analyzed all items of receipts and disbursements.

Should you desire any further information upon any item in this report, please do not hesitate to call upon me.

Respectfully submitted,
A. A. TUROFF,
Certified Public Accountant.

REPORT OF THE EXECUTIVE ASSISTANT.

Your Executive Assistant herewith submits his annual report:

Among the outstanding new features during the past year, was the adoption of a compensation plan for the employees of the Association, which became effective January 1, 1939. The total cost of the plan, which will provide $100.00 monthly for the Executive Assistant at the age of 65 and $50.00 monthly for the Secretary at the age of 60, is $600.19. However, the Executive Assistant is to contribute $108.00 and his Secretary $45.00, which will make the total net cost to the Association of $447.19. The refunds from employees are being deducted from monthly pay checks.

The funds from the John and Mary R. Markle Foundation, the Symposium on Mental Hygiene, were completely disbursed and the balance paid to the Mental Survey Committee, completing the payment over a three-year period.

I am glad to report that the financial condition of the JOURNAL has been considerably improved and, at the end of the fiscal year, only two issues are owed to The Lord Baltimore Press. In order to bring about this improvement, it was necessary to take a subsidy of $4600 from the membership fund.

There has been an increase in JOURNAL subscriptions and we now have a total of 666. Advertising revenues are running about the same as previous years.

Your Executive Assistant has continued to assist the Secretary-Treasurer, the Board of Examiners, the Committee on Program, the Committee on Public Education, the Committee on Arrangements and other committees where possible in making effective the work of the Association.

REPORT.

May 15, 1938–April 15, 1939.

INCOME:

New Business and Renewals

New Subscriptions ($60.00 still due)	$780.00	
Renewal of Subscriptions ($48.00 still due)	3,182.28	
New Advertising ($254.89 still due)	283.03	
Renewal of Advertising ($592.99 still due)	1,762.12	
Commercial Exhibits—May 1938	135.00	
May 1939	1,377.00	
Sale of Back Numbers	204.33	
Total		$7,723.76

DISBURSEMENTS:

Office Expense

Rent of Office	$900.00	
Telephone and Telegraph	137.73	
Office Supplies	26.65	
Postage	94.43	
Miscellaneous Expense	54.50	
Printing (Letterheads, envelopes, etc.)	56.61	
Dorothy Rubenstein—Salary	1,412.50	
Austin M. Davies—Salary	3,600.00	
Total		$6,282.42

Very truly yours,

AUSTIN M. DAVIES, *Executive Assistant.*

REPORT OF COMMITTEE ON PUBLIC EDUCATION.

A review of the work of this committee for the past five years indicates clearly a growing appreciation on the part of the press and the public that psychiatry is no longer a field in which spectacular, effervescent material is produced regularly for the amusement and amazement of the reading public, and to the dismay of our professional colleagues. Instead, there is a clear understanding by all news gathering agencies, that The American Psychiatric Association is a professional organization, with high professional standards of ethics, and functioning in a field where definite, unmistakable scientific progress is being made.

While this committee would not have you believe that this gradual shifting of emphasis from the bizarre to the scientific came about solely as a result of its efforts, we would have you know that the change has actually come

about. What we have tried to contribute in this direction has been based on three basic ideas:

1. The minimizing of personalities and the emphasizing of the scientific work being done.
2. Stressing the standards of the organization and its professional requirements for membership.
3. Creation of an orderly and dignified method of news distribution, by requiring all material to be canalized through this committee and by discouraging personal interviews, except in rare instances.

To be sure, the efforts of this committee would have been fruitless if the membership of the Association had not cooperated with it as heartily as they have, both in furnishing copies of their manuscripts to the committee and in the observance of the Association's rules regarding personal interviews and informal dissertations. An infraction of these rules is now a rarity and when it does occur, investigation usually discloses misunderstanding rather than specific intention to break the rules.

Another contributing factor to progress along these lines has been the increasingly fine work of the Program Committee in formulating programs which have consistently stressed the sound investigative aspects and has eliminated the unsound. A perusal of the program of the Association for the past five years will show an increasing emphasis on laboratory and clinical work and a steady diminution of the metaphysical, the unscientific or the purely philosophical.

In any event, the work of the Program Committee has made the work of the Committee on Public Education much easier, and with little necessity for anything that even borders on censorship.

The five-year period just ending has been one full of satisfying progress by the Association, both in growth of its membership and in the quality of members. As it has grown in size, and as the work of its members has become more widely known, we have come to be regarded more and more as the official organization to speak on matters psychiatric. The time must come when, if we are to assume our proper rôle in the field of medicine, and exercise the leadership which our membership strength and representation could earn for us, we must give attention to a more regular program of public education. In our last report we pointed out this need for revamping the public education work of the Association on a wider base, and with much closer rapport with local and regional events. At that time, we recommended the creation of regional sub-committees to cooperate actively in the work of public education, both as an outlet for psychiatric information, and as a source of information concerning local conditions throughout the country.

We realize that the matter is receiving consideration, together with other suggestions that have been made pointing toward a more cohesive organization, and have no desire to be importunate in pressing the point. However, it is suggested that such a plan could easily be put into effect in a semi-

official way, if this committee were empowered to go ahead with the appointment of such regional groups to cooperate with it, pending further consideration by the Council.

There has never been a time when the public has been more avidly and sincerely interested in scientific progress. It would seem that this interest has increased as the state of disruption and chaos has grown stronger throughout the world, almost as though the average citizen was turning to science hopefully, as an antidote to the spirit of barbarism and ruthlessness which threatens to engulf him. As we sense this interest, it is not merely the casual interest in the new and unsual, but a genuine hopefulness that through science a better order of things may come about.

This growth of interest is especially noticeable in the field of medicine and particularly in psychiatry. How else can be explain the phenomenon of ex-mental patients banding themselves together in voluntary organizations to act as agencies of public education. This has happened not in one instance, but in several instances, most notably in Illinois, but this spontaneous movement by former patients has also started in the East.

Under the proper guidance and leadership there is every reason to believe that this could grow into a national movement of great importance; and there is no reason why those who have been mentally ill and who have recovered, should not be the greatest and most successful workers we have in the field of psychiatric public education. In a sense, it is a reflection on our own lack of drive, that one of the first things they are seeking to do is to revise state commitment laws to eliminate onerous court proceedings.

We mention this, merely as a trend—but a significant one, and we believe it possible for this Association by proper organizational methods, to help harness some of this interest and enthusiasm, so that the cause of public education in mental and nervous illnesses may be better served.

We have considered it advisable to touch briefly on these general trends rather than to submit statistics showing the quantity of material which has been released and printed by the press in practically every state in the Union. Sufficient to say that practically every national news gathering agency and most of the metropolitan newspapers have come to look to us for authentic news of the latest developments in our field. We have tried to keep them fully informed and in return, we have received their hearty cooperation.

C. Charles Burlingame, M. D., *Chairman,*
William C. Menninger, M. D.,
Karl M. Bowman, M. D.,
William W. Young, M. D.,
Thomas A. Ratliff, M. D.

The Committee on Research has no regular report to make, and as Chairman of the Committee on Research, Dr. Whitehorn represents the Committee on the National Research Council.

Report of the American Psychiatric Association Representative in the National Research Council.

The annual meeting of the Division of Medical Sciences of the National Research Council was held April 22, 1939, for the discussion of activities, projects and budgets. As your representative is this year an executive member of the division, the expense of attending the meeting was defrayed from Council funds.

The most notable activity of the National Research Council concerned with psychiatry this year has been the launching of the Journal of Psychosomatic Medicine, made possible through the activity of the committee on problems of neurotic behavior. The first number of this journal has been issued and has probably come to the attention of all members of our Association. It may be expected to accelerate the growth and diffusion of knowledge in one of the fields of psychiatry of great importance to medical education. The joint committee on problems of neurotic behavior, in the report to the National Research Council, also emphasized the significant appreciation of such studies by the American Association for the Advancement of Science which at its Richmond meeting not only sponsored a symposium on mental hygiene but also gave its annual scientific award for work on the experimental production of neurotic behavior in rats.

The committee on problems of sex continues to emphasize the psychobiological aspects, as indicated perhaps most notably by the publication of Terman's study on marital adjustment.

The committee on drug addiction has had the satisfaction of seeing a still larger portion of its program, and important personnel, taken over by the government in the Public Health Service. This National Research Council committee, it is planned, will continue, in an advisory relationship, its important correlating functions—integrating the chemical, pharmacological and clinical studies on morphine derivatives.

One field of potentially great psychiatric significance under consideration by a committee of the National Research Council is concerned with the biological processes of aging. A program for the support of research in this field is now being formulated.

Respectfully submitted,
John C. Whitehorn.

Report of the Committee on Nomenclature and Statistics.

To the Council of The American Psychiatric Association:

In the 1938 Report this Committee recommended (1) that the preliminary work in preparation for the 1940 Revision of the Standard Classified Nomenclature of Disease be done during 1938, and (2) that a report of recommended changes be made to the Council at the 1939 meeting. This report also requested that members of the Association send into the Committee suggestions for changes. Notification of the new revisions was printed again in a later

number of the JOURNAL. In addition, members of the Committee were requested to survey their own areas, talk this matter over with other psychiatrists, and send in the accumulated suggestions. Only 16 suggestions by 12 persons were made from the entire membership and the committee combined. No agreement was observed, as no two persons made the same suggestions. After review of the suggestions the committee came to the general conclusion that the scattered changes proposed by single individuals did not justify the changing of nomenclature which was already being used by hundreds of hospitals and thousands of workers. The Committee, therefore, recommends that the present psychiatric nomenclature be preserved intact in the 1940 revision of the Standard Classified Nomenclature of Diseases.

NEIL A. DAYTON, M. D., *Chairman*,
FRED P. MOERSCH, M. D.,
ALVIN T. MATHERS, M. D.
JOHN M. DORSEY, M. D.,
GEORGE H. PRESTON, M. D.,
LAWRENCE KOLB, M. D.,
JAMES V. MAY, M. D.,
ROBERT WOODMAN, M. D.,
GERALD R. JAMEISON, M. D.,
FRANK H. LUTON, M. D.

COMMITTEE ON PSYCHIATRIC NURSING.

The work of the Committee on Psychiatric Nursing during the year 1938-1939 has centered largely on the preparation of standards and curriculum outlines for affiliate and post-graduate courses in psychiatric nursing, and of revising the standards and curriculum outlines for undergraduate schools of nursing in mental hospitals for accrediting by The American Psychiatric Association. Tentative outlines were presented to Council at its meeting in Richmond, Virginia, in December 1938 and the completed outlines are submitted with this report for your approval.

The committee is indebted particularly to Dr. Tillotson and Dr. Fitzpatrick for special help in their preparation although valuable suggestions were received from all members of the committee and from members of Council whose advice was also sought.

The usual questionnaire has been sent to all hospitals whose undergraduate schools of nursing are accredited by this association. There has been no change in the number of accredited schools during the year, 48 admitting students. One school which had accepted no students for two years again admitted students but this was offset by another school which admitted no students.

These 48 schools accepted 196 men and 788 women, a total of 984 students. This is an increase compared with the preceding year, of 29 men and 94 women, or a total increase of 123 students.

There were graduated 475 students, 93 men and 382 women, an increase, as compared with the preceding year, of 38 (30 men and 8 women).

With one exception, all accredited schools are observing satisfactory standards and practically all graduates become registered. There is a growing tendency for the general hospital affiliation to be taken in the second year, more than two thirds of the schools having this arrangement.

Greater emphasis is being placed on good academic and health environment for student nurses, with a steady reduction in the number of hours of duty, restriction or elimination of night duty, adequate vacations and satisfactory recreation facilities. Although the majority of schools have suitable residences for students, it would appear that further improvement should be made by some of the schools in their residence and dining accommodation. Very few schools appear to be having difficulty securing suitably qualified applicants. Practically all give candidates physical and psychological examinations. Nursing school committees have been organized in the majority of schools.

Although many hospitals report difficulty in securing graduates for staff needs, nevertheless there is a steady improvement in the increasing numbers of graduate registered nurses employed. In our report last year, the ratio on the women's wards was 1 to 35.6 patients. This year the ratio is 1 to 33.6 patients. There are marked variations among the hospitals in the ratio of nurses to patients, however, and many hospitals should add to the numbers of graduates employed.

Although no general survey has been made of the number of affiliate and post-graduate courses in psychiatric nursing, as was done in each of the two preceding years, nevertheless your committee is aware more affiliate courses are being organized and more general hospital students are being given the opportunity of psychiatric experience as a part of their basic training. Twenty-six of the hospitals which operate undergraduate nursing schools accredited by our association, also operate affiliate courses in psychiatric nursing and report an ever increasing demand for psychiatric nursing experience from general hospital schools.

Our committee held only one meeting during the year, at the San Francisco Convention, although the chairman met informally with several members of the committee later in the year, and all have been assisting with the work of the committee through correspondence. A successful round table on psychiatric nursing was held at the San Francisco meeting and a similar round table is being planned for the Chicago meeting. The chairman contributed a paper to the symposium on mental health arranged by the American Association for the Advancement of Science at Richmond, Virginia in December 1938, entitled, "The Present Status of Psychiatric Nursing in the United States and Canada." Dr. Fitzpatrick is to present a paper at the Chicago meeting on the subject of psychiatric affiliations.

There have been a number of requests for information as to organization, content, and certificates of approval for affiliate and post-graduate courses. If the outlines of standards we have prepared for these courses are accepted by Council, it is planned to issue certificates of approval at an early date to

schools of nursing whose courses equal or exceed these standards, as authorized by Council last year.

All of which is respectfully submitted,

G. H. STEVENSON, M. D., *Chairman,*
GARLAND H. PACE, M. D.,
PAUL G. TADDIKEN, M. D.,
CHARLES P. FITZPATRICK, M. D.,
HENRY I. KLOPP, M. D.,
EMMETT F. HOCTOR, M. D.,
KENNETH J. TILLOTSON, M. D.,
BENJAMIN W. BLACK, M. D.,
IRA A. DARLING, M. D.,
O. O. FORDYCE, M. D.

COMMITTEE ON PSYCHIATRIC STANDARDS AND POLICIES.

For various reasons the interests of the Committee on Psychiatric Standards and Policies have during the past year been particularly concerned with the Mental Hospital Survey Committee, its activities and its future. At a meeting of the Committee on Psychiatric Standards and Policies held at Richmond in December discussion centered largely around the work of the latter committee. The purpose for which the Survey Committee was created was the carrying on of a work which so far as it could be done without special funds was first undertaken in 1923 by the newly created Committee on Standards and Policies composed of Dr. William L. Russell, the late Dr. W. M. English and the late Dr. George H. Kirby. It was as a result of the survey carried on by it during the following two years that Dr. Russell's committee formulated the Minimum Standards which have for 14 years exerted a most important influence on hospital organization and management. The American Psychiatric Association was one of the most active agencies in organizing the Survey Committee and was the only medical association which contributed funds toward its operation. Through the efforts of Assistant Surgeon General W. L. Treadway, Chairman of the Survey Committee, the three-year annual grant of $16,000 from the Rockefeller Foundation was supplemented by the generous assignment of psychiatric, statistical and clerical personnel by the United States Public Health Service together with a limited travel allowance for service personnel. In September, 1938, Dr. Treadway felt that the pressure of official duties in the Public Health Service made it advisable for him to resign from the Survey Committee. Notwithstanding his request that no mention of the matter be made in this report the other members of the Committee on Standards and Policies wish to express their regret that Dr. Treadway's resignation was necessary and desire to record their appreciation of the valuable services he rendered as Chairman.

In September, 1938, the Survey Committee entered upon the third year of active work. On September 22, therefore, a meeting of the committee was held for the purpose of considering future plans. It was the unanimous opinion

of the representatives of the eight participating agencies that the work of the Survey Committee should be continued. On January 18, 1939, the Board of Directors and the Scientific Administration Committee of the National Committee for Mental Hygiene held a conjoint meeting in New York to which the National Committee invited such members of the Mental Hospital Survey Committee as reside within convenient distance of that city. Inasmuch as several members of the Mental Hospital Survey Committee have also official connection with the National Committee organization eight of the thirteen members of the Mental Hospital Survey Committee were present at this meeting. The committee was informed that the grant by the Rockefeller Foundation would not be renewed and would therefore terminate June 30, 1939. It transpired, however, that the United States Public Health Service was sympathetically disposed toward the work of the Mental Hospital Survey Committee and might entertain a request to take over its activities. Since it seemed improbable that further large sums would be otherwise available for continuing the work of the Survey Committee it was unanimously agreed to request the United States Public Health Service to take over and continue its work. The following resolutions were therefore adopted and subsequently forwarded to the Surgeon General of the United States Public Health Service:

WHEREAS, In 1936 under the leadership of Dr. Walter L. Treadway, Assistant Surgeon General of the United States Public Health Service, there was organized a Mental Hospital Survey Committee, consisting of representatives of: The United States Public Health Service, The National Committee for Mental Hygiene, The American Psychiatric Association, the American Medical Association, the American Neurological Association and the American Board of Psychiatry and Neurology, and

WHEREAS, Since July 1, 1936, the mental hospital survey work of this Committee has been made possible through a three-year annual grant of $16,000 from the Rockefeller Foundation to The National Committee for Mental Hygiene, a one-time grant of $5,000 from The American Psychiatric Association and the assignment of psychiatric, statistical and clerical personnel by the United States Public Health Service to the work of the Mental Hospital Survey Committee, and

WHEREAS, The Rockefeller Foundation has informed The National Committee for Mental Hygiene that its three-year grant of $16,000 a year, which terminates on June 30, 1939, will not be renewed because it is the belief of the Rockefeller Foundation that the value of the work has been demonstrated and that its continuation should be the responsibility of the United States Public Health Service, and

WHEREAS, During the past two and one-half years the Mental Hospital Survey Committee has conducted surveys in 169 mental hospitals in 40 states, and

WHEREAS, The Governors and Legislatures of a number of states, notably Kentucky, Michigan and Virginia, have been so responsive as to put into effect many of the recommendations in our survey reports, thus materially improving the care and treatment of the mentally ill in their states, and

WHEREAS, It is the sense of this meeting that the survey work of the Mental Hospital Survey Committee is so vital to the maintenance and raising of the standards of care and treatment of the mentally ill in the various states as to make its continuance imperative, and

WHEREAS, The United States Public Health Service has already proven its interest in this mental hospital survey work by carrying a large part of the burden and is unanimously regarded as the logical agency of the Federal Government to undertake responsibility for the continuance of this work, particularly in the light of the possibility that the projected National Health Program will be undertaken in the near future, therefore be it

Resolved, That the Mental Hospital Survey Committee recommends that the United States Public Health Service consider undertaking the responsibility for carrying on surveys of mental hospitals after July 1, 1939; with the understanding that the Mental Hospital Survey Committee will, if requested, be glad to continue its cooperation in any way possible.

The following is quoted from the Surgeon General's acknowledgment of these resolutions:

"This Committee has done a splendid piece of work, and the Public Health Service is glad to have been able to assist in it. The Service feels with the Committee that this work is not completed and that it should go on, with the end in view of discovering the defects as well as the good points in the various hospital systems, so that by the application of the information gained the standards of care and treatment of the mentally ill, as well as the studies of mental diseases in various jurisdictions may be raised to a level commensurate with the importance of this problem.

"Sensing the need, the Public Health Service will take this work over and carry it on to the extent made possible by appropriations, and in doing so we will be pleased to have the continued cooperation and assistance of the groups and individuals who have done so much to make the work of the Committee a success in the past."

It is gratifying not only that this survey so long advocated by the Association will continue but also that Dr. Samuel W. Hamilton who has served as Director since its inception will continue to be associated with it.

That the work of the Mental Hospital Survey Committee should be recognized in relation to psychiatric education is perhaps as is to be expected. This promises to constitute one of its many valuable results. On January 12, 1939, at an informal conference attended by Drs. William D. Cutter and F. H. Arestad of the Council on Medical Education and Hospitals of the American Medical Association, Drs. S. W. Hamilton and Grover A. Kempf, Director and Associate Director respectively, of the Mental Hospital Survey Committee, officials of the National Committee for Mental Hygiene and the Chairman of the Committee on Standards and Policies, the subject of the Survey Committee's contribution to the promotion of educational standards for residencies and fellowships in psychiatry was discussed. During the Chicago session of the Association representatives of several bodies, including the Survey Committee, will, under the auspices of the Council on Medical

Education and Hospitals, consider opportunities for graduate training in psychiatry with special reference to such standards.

Of interest to the medical profession was the National Health Conference held July 18-20, 1938, by the Interdepartmental Committee to Coordinate Health and Welfare Activities. On December 15, 1938, the Technical Committee on Medical Care requested representatives of various psychiatric organizations and committees including the Committee on Psychiatric Standards and Policies, to submit suggestions for a ten year program for the prevention and treatment of mental disease. Among the suggestions submitted at that meeting was one recommending that an institute for research in mental disease comparable to the National Institute of Health of the United States Public Health Service be organized by that Bureau. With the great need for research in the subject of mental disease such a national research institute under proper control with the resources of the United States Public Health Service available could not only focus on specific lines of research but cooperate with or otherwise promote research elsewhere. Such an institute could also serve as a central bureau for the collection of data dealing with past and present studies in psychiatric and allied fields as recommended by Dr. Chapman in his Presidential Address. The committee recommends that the policy of the Association relative to the establishment of properly controlled research institutes be one of sympathetic support.

The committee feels that the present annual meeting should not pass without special reference to the Symposium on Mental Health presented by Section on Medical Sciences of the American Association for the Advancement of Science, Richmond, Virginia, December 28-30, 1938, with The American Psychiatric Association, United States Public Health Service, Mental Hospital Survey Committee and the National Committee for Mental Hygiene as collaborating agencies. The comprehensive program, very ably prepared by Dr. Walter L. Treadway, included such subjects as Orientation and Methods in Psychiatric Research, Sources of Mental Disease: Their Amelioration and Prevention, The Economic Aspects of Mental Health, Physical and Cultural Environment in Relation to the Conservation of Mental Health, Mental Health Administration and Professional and Technical Education in Relation to Mental Health. In an address, Human Needs and Social Resources, Dr. C. Macfie Campbell presented an excellent summary of the Symposium. The Symposium may well be regarded as the most comprehensive contribution to the subject of mental health yet made in America.

ARTHUR P. NOYES, M. D., *Chairman*,
FREDERICK W. PARSONS, M. D.,
ROCK SLEYSTER, M. D.,
JOSEPH E. BARRETT, M. D.,
EARL D. BOND, M. D.,
WILLIAM C. GARVIN, M. D.,
CLARENCE B. FARRAR, M. D.,
MORGAN B. HODSKINS, M. D.,
WALTER L. TREADWAY, M. D.,
APPLETON H. PIERCE, M. D.

Committee on the Legal Aspects of Psychiatry.

The Committee on the Legal Aspects of Psychiatry herewith submits its annual report for the year 1938-39.

During the year no meetings of the committee have been held, but a very considerable amount of correspondence has been carried on among the members of the committee.

The committee regrets to report that during the year the cooperative activities between it and the Criminal Law Section of the American Bar Association have been in an inactive state, on account of the reorganization of the Criminal Law Section. The interest of the officials of the Section in a possible closer rapport between psychiatry and the criminal law is well known, and it is expected and sincerely hoped that the relations which have existed so closely and profitably since 1927 may shortly be resumed. A project which has been of considerable interest both to the Bar Association and to this committee has been a close survey of the present actual status of psychiatry in the criminal courts of the country. Requests for subsidies have been made to two large foundations but the necessary funds have not been secured. Negotiations are now under way with a prospective private donor, and the committee hopes that in the near future some factual data may be obtained at first hand in the manner originally contemplated.

Cases continue to arise in which the public becomes much stirred over the introduction of insanity as a defense in criminal cases. Such instances inevitably do harm to the cause of psychiatry and at the same time the popular reaction is often such that serious injustice is done to the defendant. One such case recently arose in Colorado, a state which over ten years ago hoped that it partly solved the problem of expert testimony by requiring that where insanity is pleaded as a defense in a criminal case the defendant shall be committed to the state mental hospital for observation and report. In this particular instance the defendant, one Joe Arridy, a former inmate of the state school for mental defectives, a boy of extremely low intelligence, was charged with rape and murder. Three of the physicians of the state hospital after a period of observation reported on the facts of the case and gave an unqualified opinion to the effect that by all the legal standards he should be considered "insane." Certain police officers testified that to them he appeared "normal" mentally, although the physicians found that he had a great deal of difficulty in expressing himself, had an extremely limited vocabulary and usually answered in monosyllables. The jury chose to accept the testimony of the police as against that of the disinterested and qualified psychiatrists. This procedure was sustained by the Supreme Court of Colorado, and in spite of nation-wide publicity concerning the obvious imbecility of the defendant his execution was permitted to take place. The case illustrates forcibly the public demand for a scape-goat that is frequently met in such cases, and the readiness on the part of some juries to overlook any evidence, no matter how competent, which might stand in the way of satisfaction of the public demand for revenge.

In the American Journal of Psychiatry for July, 1938, there was published without comment the report of the Commissioners in Lunacy in

the case of The People *versus* Robert Irwin, a New York case which had attracted national attention. The report of the Commissioners, which was couched entirely in legal language, was a most unjudicial document and went so far as to express an opinion concerning the insanity of the defendant in spite of the fact that the defendant had not conversed with the Commission at all. Furthermore, any evidence, of which there was in fact an ample amount, which would tend to indicate mental derangement on the part of the defendant was glossed over by the Commission in such a way as to appear to justify their report. The report seemed to call for comment, and the Chairman of this committee, after consultation with the other members of the committee, drafted a letter to the Editor of the JOURNAL, which was published in the November, 1938, issue, pointing out the serious defects of the Commission's report. An interesting sequel of the case is that the District Attorney, apparently convinced that the Commission's report would not be sustained as a result of a trial, accepted a plea of guilty in the second degree, and that within about ten days of the prisoner's reception at Sing Sing he was found by the prison physicians to be psychotic and was transferred to the Dannemora State Hospital. An attempt was made last year to secure the legal abolition of these lunacy commissions, which have served no useful purpose but have been a source of great expense to the taxpayers, particularly in New York City. A bill which passed both houses of the Assembly was vetoed by the Governor on technical grounds, as recorded in this report last year. Three bill are now pending before the Assembly with a similar purpose of abolishing these cumbersome and inefficient Commissions.

The work of the Committee of the American Law Institute appointed to consider the possibility of a change of procedure dealing with young criminals continues. No report has yet been published, but the findings of the committee will undoubtedly be of great interest to psychiatrists as well as to students of the law and all of those interested in the improvement of criminal procedure.

In the midst of popular clamor, which sometimes tends to discouragement, it is promising to note that the Commissioners on Uniform State Laws have drafted a Uniform Expert Testimony Act which is now pending before a number of state legislatures and which if adopted will do much to overcome the objections frequently heard in opposition to psychiatric expert testimony. It is of interest likewise to notice that the Rules of Civil Procedure for the District Courts of the United States which became effective September 16, 1938, authorize the court on its own motion to direct the attorneys for the parties to confer with it, among other things, on the limitation of the number of expert witnesses and on the reference of issues to a master for findings. Such a rule is an encouraging recognition of the court's right to step in in matters involving expert testimony and to avoid some of the unsavory features which now obtain too often and too flagrantly.

The committee repeats its recommendation of last year that no psychiatrist enter or appear in a criminal case for one party or the other unless as a condition precedent it is agreed that he may and shall examine the defendant

with the experts employed by the opposing party; and further that he shall be allowed to give his testimony regardless of which side such testimony would favor. It seems reasonably obvious that psychiatrists must at least do their share if they expect public confidence in the psychiatric expert witness to be restored.

WINFRED OVERHOLSER, M. D., *Chairman*,
RAYMOND F. C. KIEB, M. D.,
BERNARD GLUECK, M. D.,
WILLIAM HEALY, M. D.,
JAMES G. MCKAY, M. D.,
GREGORY ZILBOORG, M. D.,
LEROY M. A. MAEDER, M. D.,
R. E. BUSHONG, M. D.,
PAUL L. SCHROEDER, M. D.,
SYDNEY K. SMITH, M. D.

REPORT OF COMMITTEE ON PSYCHIATRIC SOCIAL SERVICE.

The Committee on Psychiatric Social Service begs to submit the following report:

It is the opinion of the committee that the topics reported on last year and in 1934 and 1936, concerning the problem of adequate training and certification for psychiatric social workers and the development of a co-operative alliance between psychiatry and psychiatric social work, are still the most important issues needing discussion and clarification.

The committee is again grateful for the attention given in The American Psychiatric Association program to the relations of psychiatry and social service, and can report that this attention has further stimulated the members of both groups concerned to look toward more advances in clarifying their problems of relationship. The request is made that the Program Committee will again arrange for a section dealing with these relationships. We would also emphasize again the need for individual members of this Association to actively concern themselves, by being informed of the type of training and experience of social workers who are employed or are being trained in their various communities, and by insisting in so far as possible on well qualified workers and teachers being employed in these positions.

The committee reports that it is taking active steps whenever possible, in accordance with the degree of authority delegated to it, to speak for the Association in the matter of standards of training being maintained in schools of psychiatric social work. We call to the attention of the members of the Association that social psychiatry and mental hygiene are being taught in all schools of social work, in most normal schools, in many colleges, and in university departments of education and social sciences. Often this teaching is being done by unqualified people. The committee feels that this situation creates an important major problem to be faced by the Association as a whole, and by individual members when the situation pertains to their communities.

The committee reports that the American Association of Psychiatric Social Workers and the American Association of Schools of Psychiatric Social Work are definitely requesting help from The American Psychiatric Association in three definite directions:

1. These groups wish suggestions and counsel in setting up and improving the formal curricula in schools of training, and a more positive interest on the part of psychiatrists in acting as teachers or advisors on course content.
2. These groups wish a more general interest and drive on the part of the profession of psychiatry toward encouragement of the employment of psychiatric social workers at adequate salaries for trained workers, especially in mental hospitals.
3. They wish the development of an increased number and more adequate training centers, especially in mental hospitals, where the students may get a thorough and vital field training under staffs who are experienced in the training of, as well as working with, psychiatric social workers.

The committee wishes to express its gratitude for the increased interest of the Council and individual members of the Association in its focal problem —of whether or not a definite profession of psychiatric social work, closely knit to the profession of psychiatry, will develop out of the present confused situation, which has arisen largely because of the demand for psychiatric social workers in widely diversified fields. The committee feels that there is a definite danger that the psychiatric profession will surrender in some measure its leadership in the broad field of mental hygiene, unless the integration between psychiatry and psychiatric social work progressively materializes.

S. W. Hartwell, M. D., *Chairman*,
Earl W. Fuller, M. D.,
Mesrop A. Tarumianz, M. D.,
George S. Stevenson, M. D.,
Paul E. Kubitschek, M. D.

Committee on Psychiatry in Medical Education.

The Committee on Psychiatry in Medical Education has engaged largely in generalities in the past years. This year it is unanimous in the recommendation that post graduate institutes be created and offered to state hospitals in appropriate geographical areas where such work is not now available.

In January of this year a questionnaire was sent to superintendents of 178 state hospitals of whom 157 replied. This questionnaire with its finding is as follows:

1. Of the 157 questionnaires returned 155 or 98.7 per cent were in favor of the Institute; while 2 or 1.3 per cent had no interest in the plan.

   The communities selected: Chicago or Elgin 21 (in addition Chicago, Elgin or Colorado 5); Colorado 17 (in addition Colorado or Elgin 5); Baltimore 17; Worcester 11; California 9; Louisville 7; Galveston 8; other areas 22.

2. Approximately 310 men would attend.
3. Subjects for discussion:
   Clinical Psychiatry (69).
   Hospital Administration (46).
   New Forms of Therapy (79).
   Psychopathology (55).
   Legal Aspects of Psychiatry (30).
   Miscellaneous: Psychobiology, neuroanatomy (4).
   Clinical Neurology (62).
   Out Patient Department (26).
   Community Organization (33).
   Organization for Research (44).
   Neuropathology (37).
4. Two weeks was the favored length of time specified for the institutes.
5. Time of year preferred:
   January, February or March (13).
   April, May or June (38).
   July, August or September (32).
   October, November or December (17).
   1 stated time depended on location.

Since only two state hospitals were not in accord with such an organization, and encouraging letters were received from those states carrying out this work it may be generally agreed that the majority of state hospital superintendents are anxious to strengthen the post-graduate teaching within their own staff, but on the whole they are confronted with the difficulty of supplying this instruction. With this in mind, the establishment of institutes organized through the Committee on Psychiatry in Medical Education of The American Psychiatric Association is suggested as a means of bringing to each state hospital staff some type of instruction which would enhance its position as a teaching center, establish a better relation with medical schools, improve research, develop out-patient departments and community organization and improve all forms of therapy. It is suggested that these institutes be composed of four qualified men, representing various branches of psychiatry. Certain strategic areas could be designated from which both the teachers and the attending staff men would be drawn. It is anticipated that such an institute would stimulate younger staff members in the various hospitals to meet the requirements of the American Board of Psychiatry and Neurology. It might well be expected to stimulate subsequent development of residencies in many state hospitals, and encourage more extensive teaching relationships with medical schools, local and state societies, general hospitals, and out-patient clinics.

Your committee realizes that the creation of the proposed institute presents many difficulties in organization, finances and administration. However, it is felt that these difficulties are not insurmountable. We desire benefit of discussion and advice of the Council concerning this work and the approval of seeking financial assistance from one of the interested foundations for the

inauguration of this work. A budget estimate covering traveling expenses, maintenance and a honorarium for a group of four teachers will amount to the sum of $8385 per year based on the organization and operation of four two week institutes in four geographical areas each year.

It is the belief of the committee that the organization of these institutes would pave the way to other educational procedures which would integrate further the teaching and research of state hospitals.

Respectfully submitted,

DR. FRANKLIN G. EBAUGH, *Chairman,*
DR. EDWARD A. STRECKER,
DR. HARRY A. STECKEL,
DR. TITUS HARRIS,
DR. ADOLF MEYER.

REPORT OF THE COMMITTEE ON RESOLUTIONS.

The Committee on Resolutions have considered various matters with reference to resolutions to be submitted to the Association and beg to present the following, the appropriateness of which is sustained by long established custom:

1. *Be it Resolved,* That the Association express its warm appreciation of the superior character of the leadership and administration, during the past year and through the remarkably successful annual meeting that is about to close, of the President, Dr. Richard H. Hutchings; and of the able collaboration of the President-Elect, Dr. William C. Sandy, of the Secretary-Treasurer Dr. Arthur H. Ruggles, of the members of the Council and of the Chairmen and members of the various committees.

2. *Be it Resolved,* That the Association acknowledge with deep appreciation the indispensable contribution to the success of the annual meeting of Dr. H. Douglas Singer, Chairman, Dr. David Slight, Vice-Chairman, and the members of the Committee on Arrangements. They extend this appreciation to Mrs. Leon J. Saul, Chairman, and to the members of the Ladies Committee for their friendly attention to the ladies attending the meeting, and for the attractive entertainment provided for them. The entertainment provided for the members of the Association by the Illinois Psychiatric Society must also be mentioned with thanks and appreciation.

3. *Be it Resolved,* That special commendations be extended to Mr. Austin M. Davies, Executive Assistant of the Association, and to his Secretary, Miss Dorothy Rubenstein, not only for their indefatigable, patient and remarkably efficient service in preparation for and during the annual meeting, but for their more enduring, faithful, and successful attention to the business affairs of the Association throughout the past year.

4. *Be it Resolved,* That the Association express its appreciation of the interest, skill and labor of Dr. Walter L. Bruetsch in preparing and in the administration of the Scientific Exhibit, and of the service of Dr. John D. Reichard in the management and display of the interesting moving pictures

and slides. This appreciation is extended to the staff of the Illinois Neuropsychiatric Institute and the Illinois Former Patient Association for the part they took in the exhibit.

5. *Be it Resolved,* That the Association feels much indebted to Dr. Dixon Ryan Fox, LL. D., President of the Union College, for his informing, inspiring and eloquent address at the Annual Dinner.

6. *Be it Resolved,* That the courteous and remarkably serviceable attention rendered by the manager, executive staff and employees of the Palmer House, especially that of Mr. M. P. Mathewson, Convention Manager, is hereby acknowledged with thanks.

WILLIAM L. RUSSELL, *Chairman,*
H. DAWSON ALLEN, JR.,
GLENN MYERS.

Correspondence.

LEBANON HOSPITAL FOR MENTAL DISEASES.

ASFURIYEH, NEAR BEYROUT, SYRIA.

August 12, 1939.

The Editor, American Journal of Psychiatry.

SIR,

In THE AMERICAN JOURNAL OF PSYCHIATRY, Vol. 95, No. 6 of May 1939, there is an article entitled "Psychiatry in Syria" by E. Lennard Bernstein, M.D. This article professes to speak of the position of "Psychiatry in Syria" without making any mention of the Psychiatric Hospital at Damascus or of its head—my friend and colleague, Dr. Assad El Hakim. Secondly, it is not correct to state that there is no practising psychiatrist and neurologist in Aleppo. My esteemed colleague, Dr. Boghossian, has been engaged in this particular branch of medicine for many years, and is the specialist to the Aleppo Courts of Justice.

The Lebanon Hospital for Mental Diseases, of which I am the medical superintendent, situated at Asfuriyeh, Beyrout, in the Lebanon Republic is not in any way affiliated to the American University of Beyrout. It is a British institution, controlled by a General Committee sitting in London, with such distinguished psychiatrists as Sir Hubert Bond, Senior Medical Commissioner on the Board of Control, Dr. Percy Smith, late of Bethlem Hospital, Dr. J. Whitwell, Hon. Librarian to the Royal Medico-Psychological Association, etc., on its Board. The hospital under my charge is a 450-bed hospital, and it is not supported mainly by private endowments but by fee-paying patients who are drawn chiefly, of course, from the Lebanon Republic, but also from Syria, Irak, Palestine, Cyprus, Transjordan, Egypt, etc. The staff here consists of two assistant medical officers and the senior holds the Diploma of Psychological Medicine of the University of Edinburgh. The matron has two assistants by day and one by night,

all, like herself, doubly qualified and the occupational therapist was formerly of the Maudsley Hospital in London. Male and female nurses number one hundred and are in equal proportions.

It is incorrect to say that formerly "all medical students in the American University put in their time at Asfuriyeh." Only a very limited number did so. The patronising statement "unfortunately, the treatment facilities have not yet been fully exploited, due to lack of funds" gives a wrong impression of a thoroughly modern institution where insulin and cardiazol therapy have been carried on for the past two years in addition to the more usual therapeutic procedures. It is wrong to state that there is any lack of funds, this hospital being particularly well-off in that respect; but owing to the rapid increase in the number of patients during the past five years, all available money has been required, in the first instance, for extension of accommodation.

Correct information is surely one of the aims of such a high-standing JOURNAL as yours. This must be my excuse for writing you at once.

This hospital is so well established (1900) that it can very well look after itself, but what grieves me is any misleading article about my esteemed Syrian and Lebanese confrères, who have been honourably engaged in neurological and psychiatric work in the countries indicated.

Faithfully yours,

R. STEWART MILLER, *Director.*

Comment.

A WORD FROM THE PRESIDENT.

The Editor has afforded this opportunity to bring to the attention of the membership several matters of interest and importance.

The place of meeting for the 96th annual meeting of the Association originally scheduled for Quebec, Canada, in 1940, has had to be changed owing to disturbed conditions occasioned by the war. Please note that The American Psychiatric Association will meet in Cincinnati, Ohio, May 20-24, 1940, with headquarters at the Netherlands Plaza Hotel.

In the current and subsequent numbers of the JOURNAL, there will be comments, reports and papers from the 1939 meeting in Chicago. Members are urged to review the proceedings, especially the reports of committees. The reports contain important statements of accomplishments, proposed plans and suggested policies, indicating trends in various psychiatric fields.

The membership is indebted especially to the Arrangements and Program Committees, and also to the management of the Palmer House Hotel for the success of the Chicago meeting. At the meeting, however, some of the readers of papers were criticized by the members because they could not be understood or heard. It will add to the enjoyment and comfort of the listeners if readers will make a special effort to speak the English language distinctly, carefully utilizing the amplifying facilities always available. Electric pointers have been purchased which will enable readers to direct attention to charts without leaving the microphone.

Those who wish to present papers should observe the regulations and suggestions of the Program Committee, especially the following:

1. Brief, informal abstracts (a letter paragraph is usually sufficient) should be sent to the Chairman of the Committee on Program before December 15, 1939. At its semi-annual meeting during Christmas week, the Committee will consider these contributions and then notify the sender.

2. The time allowed by the rules of the Association for the reading of a paper is 20 MINUTES. Contributors are encouraged, instead of reading the

entire manuscript, to present the essentials of their papers, either reading an abstract or giving the conclusions extemporaneously.

3. While all papers accepted become the property of the Association, publication in the JOURNAL cannot be promised, and by arrangement with the Editor, papers may appear elsewhere. The average number of printed pages for articles in THE AMERICAN JOURNAL OF PSYCHIATRY does not exceed 20 pages.

4. Papers will not be accepted for the program if they have either been previously published or presented at other meetings.

WILLIAM C. SANDY.

News and Notes.

HOSPITAL ACCOMMODATION IN CALIFORNIA.—In his monthly report for August, 1939, Dr. Aaron J. Rosanoff, director of institutions, comments on the construction program for state institutions for the biennium 1939-1941. With a present hospital population of 22,608 it is estimated that the population at the end of the biennium will be 24,294. With a present bed capacity in the seven state hospitals of 17,870, the degree of overcrowding which now exists is 23.49 per cent.

Despite a cut of $3,143,733 by the legislature from a total of $4,500,000 in the Governor's budget for the biennium, allotted for new construction, the building program already under way is expected to provide during the two year period a net increase of bed capacity of 3800. While, therefore, the problem of overcrowding will not be solved during the biennium its gravity will be considerably alleviated, with an estimated reduction from the present 23.49 per cent overcrowding to 11.26 per cent in 1941.

MEETING OF THE NATIONAL ASSOCIATION OF PRIVATE PSYCHIATRIC HOSPITALS.—At the annual meeting of the National Association of Private Psychiatric Hospitals held in conjunction with The American Psychiatric Association in Chicago, May, 1939, the president, Dr. Clifford W. Mack, reported good progress in organization. The Association wishes more members. There are 250 private hospitals caring for over 11,000 patients. This makes one private hospital patient to every 36 in public hospitals. First admissions to private hospitals numbered 18,212 in 1936, which was one to every 5.5 admissions to public hospitals.

A very high plane of medical practice is urged. A certificate of membership in this organization should be of great value to any institution because it signifies that standards have been adhered to. A registration board is desired. Endowment of these institutions should be encouraged. There should be cooperation with the

central Neuropsychiatric Hospital Association. Minimum standards should be formulated. The speaker hoped that the association would become an integral part of The American Psychiatric Association.

The possibilities of a survey were discussed. Since then, we hear, arrangements for such a survey have been made with the American College of Surgeons.

Mount Zion Hospital (San Francisco) Psychiatric Service.—With the increased recognition of the importance of psychiatry in general medicine and with the growing demand for more psychiatric service, a separate psychiatric department was organized at the Mount Zion Hospital in San Francisco, California, by reorganizing the department of neuropsychiatry into two independent services—the neurological service and the psychiatric service. Dr. J. Kasanin, director of the psychiatric department of the Michael Reese Hospital, Chicago, Illinois, and assistant professor of psychiatry at the Rush Medical College, University of Chicago, has been appointed as chief of the service. Associated with him will be Dr. Mervyn Hirschfeld, assistant professor of neurology at the University of California Medical School, who for many years has been interested in the development of such a service. Special attention will be paid to the development of child psychiatry which will be under the direction of Dr. Joseph C. Solomon, formerly on the staff of the Baltimore Child Guidance Clinic.

In its research program the clinic will attempt to perfect more effective methods of therapy of the various neuroses both in adults and children which are commonly found in medical practice. Especial attention will be paid to the field of psychosomatic medicine which is coming into prominence at the present time.

The Journal of Endocrinology.—The first issue of this new British quarterly bears the date of June 1, 1939. *The Journal of Endocrinology* has been founded to bring together the work of British investigators in this field which hitherto has been scattered among other scientific publications. The first issue contains 110 pages and includes 11 contributions, several of which are illustrated.

The editorial staff consists of Professor E. C. Dodds, editor, Dr. R. L. Noble, assistant editor, and an editorial board which includes Dr. P. M. F. Bishop, Professor C. R. Harington, Professor G. F. Marrian, Dr. A. S. Parkes, Dr. F. G. Young and Dr. S. Zuckerman.

Contributions from all countries will be received.

This Journal is a publication of the Oxford University Press.

THE CATHOLIC UNIVERSITY OF AMERICA, WASHINGTON, D. C.—The Catholic University of America in its 1939-40 prospectus announces a new combined course of instruction in the department of psychology and psychiatry. The experiment of this union into one department has been made possible by a grant from the Rockefeller Foundation for further teaching and research in psychiatry; and it is undertaken in the hope that two sciences which have so much in common will attain a more normal and homogeneous development by being closely associated. Undergraduate and graduate courses are included in this program.

The department of psychology has been in existence at the University since its foundation in 1889. In January 1916 a psychiatric clinic was opened at Providence Hospital in which the mental problems of children and adults were dealt with until September 1937, at which time the clinic was transferred to St. Thomas Hall on the University campus.

Rev. Thomas V. Moore, O. S. B., Ph. D., M. D., is head of the department, and professor of psychology and psychiatry.

CHILD PSYCHIATRY AT MAUDSLEY HOSPITAL.—In the new building program at Maudsley Hospital provision has been made for both in- and out-patient children's services. Hitherto no in-patient facilities have been available in Great Britain for the treatment of the psychological and psychiatric disorders of childhood not coming under the head of mental deficiency. The new children's block at Maudsley is therefore an entirely new departure designed to meet this very palpable need, and is significant of the modern trend of psychological medicine. The new building is quite detached from the rest of the hospital.

FELLOWSHIP OPPORTUNITIES IN EXTRAMURAL AND CHILD PSYCHIATRY.—A limited number of fellowships has been provided by The Commonwealth Fund and other sources of training in extramural, especially child, psychiatry. These fellowships are to be administered by The National Committee for Mental Hygiene, through whom fellows are to be assigned for one or two years to a selected child guidance clinic, the term and plan of the fellowship to be determined by the peculiar needs of the fellows. Candidates for fellowship award should have had at least two years of psychiatry in an approved mental hospital, in addition to other qualities fitting them for extramural service. These fellowships come in response to a definite paucity of personnel in this field. Accordingly, other conditions as to age, sex, marital status, etc., must be governed by individual cases and by the nature of current demand. Requests for further information about these fellowships, and applications therefor, should be addressed to Dr. George S. Stevenson, The National Committee for Mental Hygiene, Room 822, 50 West 50th Street, New York, N. Y.

FELLOWSHIP AWARDS IN EXTRAMURAL PSYCHIATRY.—The National Committee for Mental Hygiene announces the following appointments of one year's training in extramural psychiatry under a grant from the Commonwealth Fund: Dr. George E. Gardner of the Massachusetts General Hospital to the Judge Baker Guidance Center; Dr. John H. Waterman of the Hastings (Nebraska) State Hospital to the Philadelphia Child Guidance Clinic; Dr. J. Franklin Robinson of the Iowa State Psychopathic Hospital to the Los Angeles Child Guidance Clinic; Dr. Abraham Z. Barhash of the Essex County (New Jersey) Mental Hygiene Clinics to the Mental Hygiene Clinic at Louisville. Under local provision, Dr. Astor R. Schrag of the Provincial Mental Hospital at Ponoka, Alberta, to the Detroit Children's Center; Dr. Benjamin F. Vogel of the Psychiatric Division of Bellevue Hospital to the Baltimore Mental Hygiene Clinic; Dr. Phyllis D. Schaefer of Greystone Park State Hospital to the Worcester Child Guidance Clinic.

ANNUAL MEETING, AMERICAN ASSOCIATION OF PSYCHIATRIC SOCIAL WORKERS.—The American Association of Psychiatric Social Workers reports a year of progressive activity for 1938-1939. Concentration of effort has centered upon the subject of professional training in psychiatric social work and the issuance of a statement entitled, "Curriculum for Professional Education in Psychiatric Social Work," outlining minimum standards for training in this specialized area to combine with the usual related courses of an acceptable graduate school curriculum. The curriculum, as presented, is the result of several years of study by successive Professional Education Committees of the American Association of Psychiatric Social Workers and the Committee on Psychiatric Social Work of The American Psychiatric Association. It has been offered in response to requests from the American Association of Schools of Social Work, professional schools of social work and others, for a statement of standards on this subject. During the year the Committee on Psychiatric Social Work of The American Psychiatric Association has convened jointly with the Committee on Relationship with Psychiatry of the American Association of Psychiatric Social Workers to discuss problems of mutual interest to the two Associations, especially those concerned with the training of psychiatric social workers. Both Committees are acting together in the process of distributing the "Curriculum for Professional Education in Psychiatric Social Work" to professional schools of social work in an effort to gain their reactions and suggestions concerning its practicability as applied to the current curricula of schools. The round table on psychiatric social work conducted at the annual meeting of The American Psychiatric Association May 1939, was also chiefly devoted to a discussion of this subject. Other subjects of mutual interest to the two Associations include the consideration of the organization and personnel of psychiatric social service departments in mental hospitals and clinics, standards for civil service requirements in psychiatric social work, and ways and means for extending facilities for field training in psychiatric hospitals and clinics to students of psychiatric social work. Committees and studies have been fostered by the American Association of Psychiatric Social Workers to gather information and consider problems related to these subjects.

The membership of the American Association of Psychiatric Social Workers has steadily increased during the past few years, its membership committee having reported on June 15, 1939, a total membership of 535, of whom 402 were active and 133 junior members.

The National Conference of Social Work, held in Buffalo, June 18-24, 1939, indicated a persistent interest in the subject matter of psychiatric social work when the Tuesday afternoon program of the American Association of Psychiatric Social Workers drew an attendance of nearly 1200 persons. The Association's program covered two regular open sessions and included a closed session for members, the latter to consider special committee reports and to offer opportunity for members' expression of interests and ideas regarding future Association activities. The papers * of the regular program were concerned with psychiatric social work as influenced by agency function, drawn from experience represented by a children's clinic and a mental hospital; and case work process considered from two viewpoints—the developing experience of the worker-client relationship and the clarification and meeting of the client's problem or "need." The topic of the dinner speaker, Robert Waelder, Ph. D. of the Boston Psychoanalytic Institute, was "Some Psychoanalytical Aspects of Education."

Among the amendments to the by-laws passed at the annual business meeting of the Association, at which seventy-two active members were present, is the provision permitting the establishment of district branches, their incorporation dependent upon their conforming in membership requirements to the by-laws of the National Association. A vigorous negative vote on a proposed by-law concerned with membership requirements, which substituted experience in community social agencies for that gained from a psychiatric clinic or hospital, left no question that in the minds of the membership psychiatric clinic and hospital experience is regarded as essential and basic to membership requirements.

The American Association of Psychiatric Social Workers welcomed the invitation of The American Psychiatric Association to provide a representative to discuss Dr. Samuel W. Hartwell's chal-

* "Function and Process in Psychiatric Social Work," *The News-Letter*, Summer 1939, may be obtained from The National Committee for Mental Hygiene, 50 W. 50th Street, New York City.

lenging paper "Social Psychiatry—Our Task or a New Profession," presented by him at the annual meeting of The American Psychiatric Association in Chicago, May 1939. In response Katharine Moore Wickman acted as guest discussant.

The newly elected officers 1939-1941, and the executive committee of the American Association of Psychiatric Social Workers are as follows: president, Leona M. Hambrecht; vice-president, Glee Hastings Dervend; secretary, Amelia Igel; treasurer, Mary B. Laughead; executive committee members: Clara Bassett, Hester B. Crutcher, Christine Robb Thompson, Anna Belle Tracy, Katherine Moore Wickman, *ex-officio*.

LEONA M. HAMBRECHT.

Book Reviews.

THE GENETICS OF SCHIZOPHRENIA. By *Franz J. Kallmann, M.D.* (New York: J. J. Augustin, 1938.)

This study of schizophrenic patients and their families was undertaken by the author in 1929 in collaboration with the German Research Institute for Psychiatry in Munich, and with the support of the former Central Medical Bureau of the city of Berlin. The purpose of the study was to assemble conclusive proof of the inheritance of schizophrenia and thus to aid in establishing a dependable basis for the use of clinical and eugenic psychiatric measures.

Before beginning the study, the author was thoroughly convinced of the correctness of the assumption of Dr. Ernst Rüdin that schizophrenia is inherited as a recessive trait in accordance with Mendelian laws. The probands forming the basis of the study were the patients that had been committed to the Herzberge Hospital of Berlin during the years from 1893 to 1902. They were all supposed to be schizophrenics in which the disease onset had occurred before the age of 40. In describing the selection of the cases, the author states that the hospital records contain entries of 60,000 cases. Of these, he selected about 15,000 cases that were received in the hospital between 1893 and 1902. Taking the latter group and without reference to the diagnoses made by the psychiatrists in the hospital, he examined the case histories with respect to neurological and psychiatric findings together with data concerning the onset and progress of the psychosis. Autopsy findings were also considered whenever they were available. It will be noted that the author made his diagnosis of the cases from 27 to 36 years after the patients had been received in the hospital. He states that "out of conscientious scruples and in order to obtain the highest possible clinical and biological uniformity of our source material we finally ruled out all cases in which exogenous factors might have played a decisive rôle in the origin or development of a psychosis that resembled schizophrenia." He finally assembled a group of 440 male cases and 647 female cases, the smaller number of male cases being due to the fact that exogenous factors appeared more frequently in male cases than in female. Presumably other selective factors were used by the author in choosing his group. Of the 15,000 cases examined, it is probable that about 25 per cent, or 3,750 were schizophrenic. The number chosen by the author was less than one-third of this number. It may reasonably be questioned, therefore, whether the sample chosen by the author for study represented a fair sample of schizophrenics.

After choosing cases to form the basis of his study, the author proceeded to make a thorough-going study of all available relatives of the probands. From the data he was able to collect by interviewing various relatives and other persons, he made diagnoses of all disorders reported. Such diagnoses in some cases were the results of personal interviews with the afflicted persons but in other cases were based on information derived from other persons. All diagnoses were merely the individual determination of the author. Several years were required to amass the data and analyze them for presentation in book form.

The completed study before us is an extremely difficult treatise to read and understand. Likewise the statistical treatment leaves much to be desired. There is also a grave question whether the conclusions arrived at by the author are justifiable by the data presented. However, the author apparently is conscientious and desires to reach conclusions that will be confirmed by the reader. In material of this kind, of course, various interpretations are possible. The following are some of the principal conclusions arrived at by the author:

1. The predisposition to schizophrenia is specific, but leads as in the case of tuberculosis, through the interplay of furthering and resisting somatic processes to various disease forms in the phenotype. These clinical variations seem to depend on qualitative genetic differences and can be classified as a nuclear and a peripheral group of schizophrenia.

2. The pathogenesis of schizophrenia represents a complex hereditary process which cannot yet be described in its detailed ramifications.

3. The mode of manifestation seems to depend on the total constitution, which probably determines the disease-form in the individual and is in turn conditioned by special hereditary factors.

4. These heredito-constitutional elements may also be responsible for the less favorable hereditary prognosis among the blood-relations of the probands in the nuclear group than that among those in the peripheral group.

5. The finding of various taint differences in the descent groups of schizophrenia, according to the original disease-form, by no means disagrees with the assumption that the genotype of schizophrenia is a single-recessive trait, penetrating only with a probable manifestation of approximately 70 per cent.

The book as a whole is worthy of the attention of all persons interested in the genetics of mental disease. One need not agree with the author's point of view or his final conclusions in order to appreciate the tremendous, painstaking work that is represented by the data presented in this volume.

Horatio M. Pollock, Ph. D.,
New York State Dept. of Mental Hygiene, Albany.

The Primate Thalamus. By *A. Earl Walker, M. D.* (Chicago: The University of Chicago Press, 1938.)

This comprehensive work on the structure of the thalamus and on the fibre-tracts connecting this organ with other parts of the central nervous

system is based on the author's detailed anatomical and experimental study of the macaque monkey.

Following preliminary chapters dealing with the history of investigations of the thalamus and with the author's methods of study of the subject, a detailed description of the gross and microscopical anatomy of the macaque thalamus is given. This is illustrated by clear photomicrographs and diagrams. The afferent connections of the thalamus were studied by noting degeneration in the thalamus following experimental lesions of very many sensory pathways.

The relationships of the thalamus and geniculate bodies to the cerebral cortex form the largest section of the work. These relationships were investigated mainly by experimental destruction of different cortical areas, with subsequent study of the retrograde degeneration of nerve-cells in the thalamic and geniculate nuclei.

There is a final chapter on the significance of the thalamus.

Each chapter has a comprehensive bibliography of articles dealing with the various phases of the subject.

The book will be most particularly of value to the experimental physiologist. It is well illustrated throughout by diagrams of the experimental lesions and of the thalamic degeneration which result from them.

Eric A. Linell, M. D.,
University of Toronto.

The 1938 Mental Measurements Yearbook of the School of Education, Rutgers University. By *Oscar Krisen Buros,* Editor. (New Brunswick; Rutgers University Press, 1938.).

The series of annual test bibliographies, published by the School of Education of Rutgers University since 1935, has become well-known and has proved to be of great service to test users. The present volume is the most recent contribution to that series.

The scope of this issue is much enlarged over earlier ones. It includes original reviews of standard tests, a bibliography, and book review-excerpt section of statistical and research methodology books, reports of regional programs, periodical directory and index, publishers' directory and index.

It is not possible for a single reviewer to evaluate such an extensive work in detail. Undoubtedly this issue will be even more helpful than earlier numbers in the series. It is a monumental piece of cooperative effort, and the author and his collaborators naturally welcome suggestions for future issues and knowledge of errors of omission or commission.

The reviewer would like to see a section setting forth the main systematic trends from time to time, and a critical evaluation of the major signs of progress, both in test construction and in researches involving test usage. If possible, some of the more qualitative and highly significant psychological tests might also be included, with particular reference to their research application.

The author and his collaborators are certainly to be congratulated on the excellence of their product, and on the service they are giving to all workers in this field.

W. Line, Ph. D.,
University of Toronto.

Analysis of Parergasia. By *Gladys C. Terry and Thomas A. C. Rennie*, M. D., (New York: Nervous and Mental Disease Monographs, 1938.)

This book is divided into three sections with an introduction by Adolf Meyer. After presenting their material and method of study the authors discuss the clinical types, variety and range of clinical reaction patterns, and the life history of the parergasic patient. There is a final section on outcome, assets and summary. Seventeen individually coded and charted cases are appended.

In this analysis of parergasia, a term originated by Adolf Meyer to replace that of dementia præcox, the psychobiological approach in psychiatry is very clearly outlined. As Meyer points out in the introduction, psychiatry at the end of the nineteenth and beginning of the twentieth century was preoccupied with the concept of dementia and deterioration rather than with the study of the life history of the individual patient in terms of mental health and recoverability. It was because of his great dissatisfaction with the purely descriptive psychiatry of Kraepelin that Meyer introduced his psychobiological concepts.

The authors elaborate these concepts by stating that equal emphasis is placed on the specific development of the case in which all the factors at hand, prognosis and assets, are given equal consideration. With such an emphasis on synthesis and adjustive factors, any fatalistic concept is avoided. That deterioration is not an inevitable end-stage of the psychosis is one of the main theses of the psychobiological approach.

With this as their formulation the authors studied 77 cases of parergasia. The detailed nature of the anamnesis, which is undoubtedly superior to the older methods of history taking, is indicative of the absence of any rigidly preconceived theories as to the mechanisms of the psychosis. Every factor which may have a possible bearing in the development of the abnormal reaction, including heredity, constitution, inferiorities, conditioning environmental influences, family interdependence, difficulties of adaptation, extrafamilial difficulties and personality assets, is evaluated. Although the authors state that the great majority of the cases analyzed fall into several groups, the aggressive reaction, conversion reaction, deliroid reaction, affective reaction, and defeatist reaction, they are quick to point out that no rigid nosological system can do full justice to the ever-changing aspects of the parergasic reactions.

The outstanding data elicited in the histories of the 77 cases were: (1) the great incidence (60 cases) of odd personalities in the relatives; and (2) the presence in early life of personality trends which later caused difficulties. The reviewer feels that control studies would be definitely worth-

while for comparison with the authors' figures, first, in normals and, second, in psychoneurotics. The authors make the observation, which is probably insufficiently stressed in psychiatric literature, that hypochondriasis, hysteria, neurasthenia, and obsessive states may all be seen in relatively pure culture as precursors of the parergasic reaction. In evaluating the authors' material one cannot escape the outstanding conclusion that heredity or constitutional factors play the most important rôle in the development of the parergasic psychosis.

A very interesting and usually neglected aspect of the parergasias, namely, the relationship between the assets of the subjects and recoverability, is discussed by the authors. Some of the outstanding assets of value were found to be a sympathetic environment to which the patients can return, onset of the psychoses in a setting of unusual strain possible to modify, evidence of intact thinking and personal appearance, absence of hallucinatory phenomena, affective admixtures in the psychoses, temporary infectious processes at the onset or during the psychoses.

Of the 77 patients, 34 recovered sufficiently so that they did not need hospital care. On the basis of these figures, the authors advise against undue enthusiasm in evaluating the "cures" obtained by the newer methods of shock therapy until patients are followed for an adequate period of time.

In discussing the hygienic aspects of the parergasic reactions, the authors avoid a pessimistic attitude by stressing that an attempt should be made to treat the adverse environmental conditions. Thus, the submissive, shy, timid child might need special attention. Protection from undue family security should be avoided. Limited interests need expansion. Very important is the provision of an atmosphere of security and of frankness and tolerance. Finally, the authors reiterate the concept upon which the psychobiological approach is based. A study of every aspect and manifestation that promises results—not to get stuck on undue emphasis of any one item or set of items, whether it be brain histology or Berger's waves, endocrines or infections, but to bear always in mind that no set of symptoms isolated from the setting in which it occurs can have quite the same significance.

Whether through the psychobiological approach psychiatry has gained a better understanding of the mechanism of the parergasic reactions may be open to question. That its liberal and unbiased attitude in psychiatry is healthful cannot, however, be denied.

The reviewer recommends the "Analysis of Parergasia" as one of the clearest expositions of the psychobiological approach in psychiatry that has come to his attention.

JULIUS LOMAN, M. D.,
Boston State Hospital, Boston.

PASTORAL PSYCHIATRY. By *John Sutherland Bonnell.* (New York and London: Harper & Brothers, 1938.)

This is another of those somewhat numerous books written by clerical gentlemen upon various aspects or transformations of faith healing, the

current mode being "pastoral psychiatry." Here we have the phenomenon of a layman writing a treatise on a medical subject, and to add emphasis to his authority the publishers suggest on the cover that within one may learn "How to make psychiatry a servant of religion." That should make the other medical disciplines look up respectfully. With calm disregard of the established scientific use of the term, the author justifies his employment of the word "psychiatry" to signify "spiritual healing to the soul" by citing the several definitions of "psyche" in Liddell and Scotts' Greek Lexicon. The author's position recalls that of Heinroth who wrote about *Seelenstörungen* in 1818.

The publishers state that Dr. Bonnell, who is presently pastor of the Fifth Avenue Presbyterian Church, New York City, brings to the writing of this book "a background of thorough training in a mental hospital." The author himself makes specifically no such claim. His father had been supervisor of attendants at the provincial mental hospital at Charlottetown, Prince Edward Island, and at the age of 17 the son entered upon the duties of an attendant at that institution. The training, as he tells us, consisted of the lectures given by the superintendent during a two years' course for the nurses.

The technique discussed in this book is essentially the technique of the confessional. The author gives as examples the case of a teacher who had had an affair with a married man and whose conscience troubled her (but only after the man's sudden death); the case of a young man with a fear of death who was a victim of shame over "hidden sin"; the case of an unmarried socialite who at the age of 55 began to blame herself for an illegitimate child she had had at 20; the case of the pair of lovers who were in a dither as a result of "petting parties." In all these cases there is the implication of sin from which release must be had through confession and prayer. In some of them the author describes what seems to be a sort of "conversion" experience.

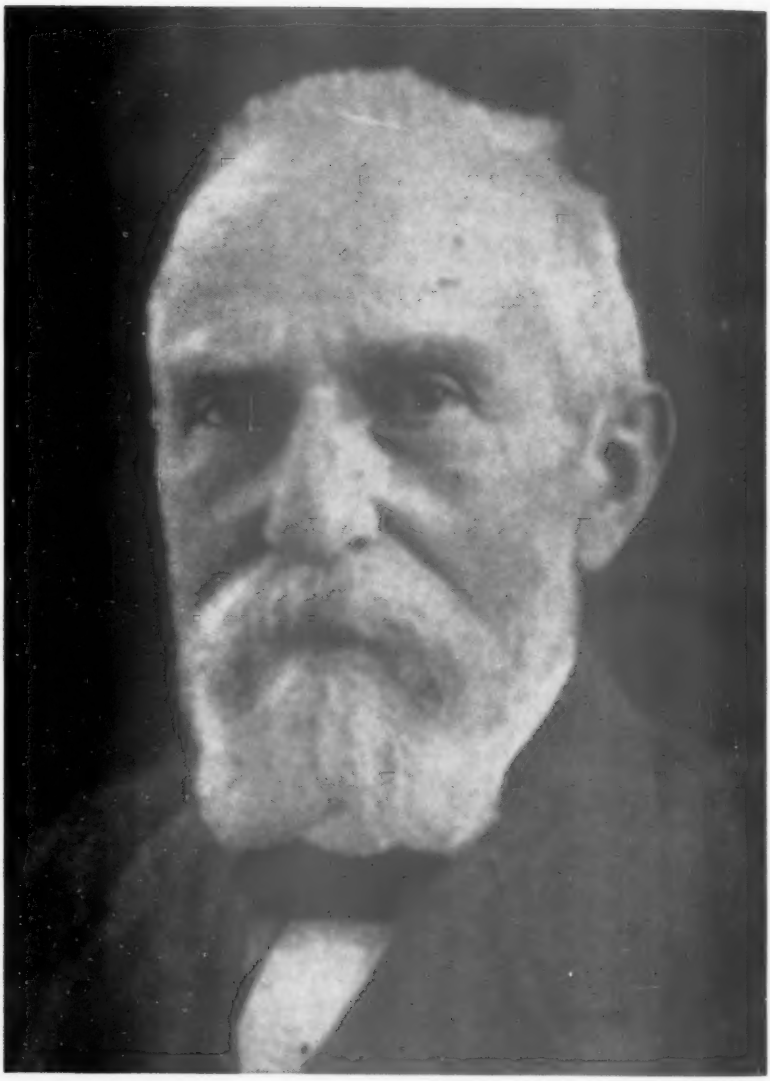
Without doubt there are those who find satisfaction in confessing their "sins" and in the authoritative assurances of priest or clergyman; and the pragmatist may argue that any method that works is a good method. That faith healing is a form of psychotherapy to be recommended is however another question. Problems such as those under discussion can be dealt with in simpler ways and in common physiological, psychological or social terms without the artificial concept of "sin" and the consequent necessity for "forgiveness." Moreover faith healing cannot avoid emphasizing the passivity of the subject, his own inadequacy and dependence; whereas psychotherapy should stress the active rôle of the patient in his own treatment and endeavor to build up his own self-sufficiency. In fact ideas of sin, pangs of conscience, guilt, remorse are often themselves symptoms against which the efforts of the therapist have to be directed.

Medical reports of treatment methods usually have to record failures as well as successes; by the Bonnell method there are apparently no failures. He offers a series of cases with uniformly satisfactory solution of personal problems. Some of these accounts are highly dramatic, they suggest com-

position as in a painting. Whether the emotional season of prayer, with which the pastoral interviews commonly end, are to be regarded as wholesome or good mental hygiene will probably depend upon the religious bias of the reader. To the devout it may represent the working of the spirit at its best; to the less devout the promotion of detailed sex recitals and prayerful emotionalism may not connote the best possible taste.

The author's grasp of the problems of health and ill health and treatment are reflected in the following declaration, with the quotation of which we lay aside this book: "The physician works with the body, the psychiatrist with the mind, the pastor with the soul. . . . . Many disorders of body and mind are due to maladies of the soul with which only a spiritual ministry is equipped to deal."

C. B. F.



DR. EUGEN BLEULER

In Memoriam.

EUGEN BLEULER.

1856-1939.

After a short illness of about three weeks, Eugen Bleuler died at the age of 83 at his home in Zollikon, Zurich, on July 15, 1939. He was seemingly convalescing from grippe when he was overcome by weakness and gradually sank into a comatose state three days before his death.

Through his death The American Psychiatric Association has lost one of its most prominent honorary members and the psychiatric world its most outstanding representative. For Eugen Bleuler was an active and energetic worker and a prolific contributor to his chosen science for over half a century. Shortly before he became comatose he discussed agonal hallucinations with his son, Dr. Manfred Bleuler.

While still a student Bleuler chose psychiatry as his life's work, and in 1883 he became assistant to Schaerer in the psychiatric clinic at Bern. After a year's work in this clinic Bleuler was attracted to Paris, where he attended Charcot's and Magnan's clinics. It was there that Bleuler became impressed with the value of hypnotism and suggestion, not only as a method of therapy, but also as a process of investigating mental mechanisms. His work, *Affectivity, Suggestibility, Paranoia,* was undoubtedly stimulated by Charcot's clinics. Following his sojourn in Paris, Bleuler spent some months in England where he oriented himself on the psychiatric work in English speaking countries, and then took a six months' course in cerebral anatomy with Gudden in Munich.

In 1885 Bleuler became an assistant to August Forel at the clinic of psychiatry at Burghölzli, Zurich. August Forel, the most progressive psychiatrist of his time, sociologist and naturalist of no mean ability, soon realized that he had found a worthy assistant in Eugen Bleuler, who resembled him in many respects. They were both versatile and broad-minded, both dared go beyond the narrow confines of pre-Kraepelinian psychiatry. Just 50 years ago, they

organized the *Trinkerheilstätte* (at Elikon, Zurich). It is still the best institution of its kind for the treatment of alcoholism and an examination of its mode of operation shows that Forel and Bleuler anticipated by half a century what we now consider the most modern way of treating alcoholism. Bleuler was the leading spirit of this institution until he died. On the notice of his death one reads: "The deceased requested that instead of flowers, checks be sent to the secretary of the Swiss Society for Abstinence."

In 1886 Bleuler became the director of the Psychiatric Hospital at Rheinau, a position for which he had been highly recommended by August Forel. He remained there for 12 years until 1898, when he succeeded to the directorship of the Clinic of Burghölzli, Zurich, on the retirement of August Forel. Bleuler worked in Burghölzli for almost 30 years, until his retirement at the age of 70.

That Bleuler made many important and original contributions to psychiatry is known to every student in this field. Throughout the whole scientific world he was known as the great pioneer in modern psychiatry. A glance at his prolific productions shows that Eugen Bleuler anticipated many new problems and had the courage to put them into operation. He was among the first psychiatrists, if not the first, to recognize the importance of occupational therapy, while he was still the director of Rheinau. With Professor von Monakow he founded the Zurich Society for Psychiatry and Neurology, and throughout his life he was one of its most active members. He was one of the first psychiatrists to accept and defend Emil Kraepelin's new formulations. But, as can be seen from his work, *Schizophrenie* (1911), and his *Textbook of Psychiatry,* Bleuler made extensive and important modifications and additions to Kraepelin's original views. He broadened and clarified the concept of dementia præcox when he changed its name to "schizophrenia," which stresses the element of splitting, rather than the elements of dementia and precocity; and he put new life, new interest and new meaning in schizophrenia and into psychopathology in general by applying to it Freud's "depth-psychology"—*Tiefenpsychologie,* as Bleuler often referred to psychonanalysis.

It was a daring undertaking to take Freud seriously at a time when all the leading psychiatrists either ignored or scoffed at everything that came from that investigator. Bleuler was the first

psychiatrist who had the great vision to appreciate the importance of the psychonanalytic concepts, and in his courageous and energetic manner resolved to test them in his hospital. Anyone conversant with the history of the psychoanalytic movement knows the important part played by the Zurich School in the introduction and promulgation of Freud's works.

Although Bleuler played a great part in the beginning of the psychoanalytic movement and accepted Freud's basic principles, he never agreed with everything that came from Freud and his School. He himself clarified his position in this respect in two papers: *Freudian Mechanisms in the Symptomatology of the Psychoses* (1906) and his *Critical Remarks on Freud's Psychoanalysis.* These works are masterpieces of psychoanalytic exposition and clear thinking which could be read by the psychiatrists of today with interest and benefit.

Professor Bleuler published over 150 psychiatric works and many semi-scientific papers dealing with the psychological and social phases of psychiatry. For the last two decades of his life he devoted himself to the relation of biology to psychiatry. He wrote a very profound and interesting book, "The Natural History of the Psyche," (*Die Naturgeschichte der Seele*—2 editions), and numerous papers which embody these views. He was particularly impressed by Richard Semon's *Mnemism,* some of whose views and terminology he incorporated into psychiatry. In my paper, *Determinism in Psychiatry and Psychoanalysis,** I gave a review of this phase of Bleuler's works.

Professor Bleuler was appreciated and honored in his homeland and abroad. He was professor of psychiatry in the University of Zurich until his retirement, when he was appointed honorary professor. He was the "Rector" of the University of Zurich from 1924-26, and held many other scientific and honorary positions. But of all these distinctions I know that he prized most highly the honors that came to him from this country—namely, his honorary membership in The American Psychiatric Association, and his two invitations to the opening ceremonies of the Phipps and New York Psychiatric Institutes. At the opening of the Phipps Institute, to which he was invited in 1913 by his friend and colleague, Dr. Adolf

* American Journal of Psychiatry, Vol. 95, No. 3, November, 1938.

Meyer, he spoke on "Autistic Thinking," and at the New York Psychiatric Institute to which he was invited in 1930 by Dr. George H. Kirby, he spoke on "Psyche and Psychoid: Scientific Conceptions of the Relation between Psyche and Body."

Eugen Bleuler was a genial and good natured husband and father, a friend and ready helper to those of his numerous pupils who were in touch with him. He represents a most important epoch in the history of psychopathology. He is the spiritual father of interpretative psychiatry.

A. A. Brill, M. D.

THE DESIDERATA OF CENTRAL ADMINISTRATIVE CONTROL OF STATE MENTAL HOSPITALS.*

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Among the startling phenomena connected with the growth of this country since the time of the Civil War few are more striking than the tremendous development both in numbers and in size of state institutions for the care of the mentally ill and the mentally defective. Today, indeed, the operation of state mental institutions may well be classed as "big business," with an investment in the physical plants of probably over one-half a billion dollars, and an annual cost for maintenance of over one hundred million dollars, not to mention capital charges, such as interest and depreciation and cost of new construction. Such a growth was entirely unforeseen in the early days of this Association. In 1851 the Association of Medical Superintendents of American Institutions for the Insane (as this Association was then known) set 250 beds as the desirable upper limit of size of mental hospitals, but within 15 years, owing to what was even then considered rapid growth, and after careful deliberation, they raised this limit to the huge number of 600 beds! In 1870 the United States boasted 50 public asylums, as they were then called, and 16 private, with a total number of 17,735 patients and an annual admission rate of slightly over 10,000.† By contrast we find in the year 1936,‡ according to the United States census, 176 state and federal hospitals, together with 25 mental hospitals operated by the Veterans Administration, 67 city and county hospitals, 8 state psychopathic hospitals and 199 private hospitals, or a grand total of 523. At the beginning of the year 1936 there were under care in all of these

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

† See "Report of E. T. Wilkins, Commissioner in Lunacy for California." Sacramento, 1871.

‡ See Patients in Mental Hospitals: 1936. U. S. Bureau of the Census.

hospitals combined 419,832 patients (49,113 more were on parole or otherwise absent), and during that year 146,650 were admitted, making a total of 566,482 under care during 1936 as against approximately 28,000 in 1870.

Side by side with this huge physical and financial expansion there has come about an extraordinary development of the medical specialty of psychiatry, and particularly during the past two decades a great demand for extra-mural services, services which can well be rendered by the states with the state hospitals as nuclei. The need of expert planning on a state-wide basis with regard to the development of existing institutions, the establishment of new hospitals and the furnishing of community services, has been increasingly recognized, and with it has come growing realization of the need of some type of central control under expert supervision.

The purpose of the present paper is to outline something of the evolution and the present state of central administrative control of mental institutions, together with some indication of the purposes served by such control and the ways in which these purposes may be better served.

Mental hospitals, or, as they were then called, asylums, were in the early days small institutions and usually there was only one within a state, under the control of a superintendent and board of trustees. They were self-contained, essentially custodial as the name implied, and presented a very simple administrative problem. Since the underlying concepts were those then prevailing in the field of relief, such supervision as was exercised by any outside source was that of the State Board of Charities or some similar organization. The number of institutions gradually increased, so that when the first State Board of Charities, that of Massachusetts, was organized in 1863 there were in that state nine state institutions, three of which were mental hospitals. During the succeeding decade ten similar state bodies were organized. Their chief interest was at that time financial, and their principal functions were to provide for stricter accounting and to avoid overlapping of function. There was no particular medical supervision, and a rather striking lack of uniformity existed in the methods of care and treatment in the several mental hospitals. Considerable resis-

tance was manifested on the part of the superintendents and trustees to the establishment of any type of control from above, and the proceedings of this Association in 1876 were considerably enlivened by the introduction of some resolutions presented by Dr. Isaac Ray. From the context, and from some of the discussions which followed the introduction of the resolutions, it is apparent that one of the advantages alleged for central control was that by it "railroading," as it was called, would be prevented. Obviously, however, this was not the sole reason, or perhaps even the principal one. Dr. Ray's resolutions recommended as the best means of government of mental hospitals, vesting the control of the whole establishment in an "eminently qualified" physician as superintendent, and a board of directors or trustees. He proceeded with the eloquence of which he was a master, as follows: "Resolved, that any supernumerary functionaries endowed with the privilege of scrutinizing the management of the hospital, even sitting in judgment on the conduct of attendants and the complaints of patients, and controlling the management, directly by the exercise of superior power or indirectly by stringent advice, can scarcely accomplish an amount of good sufficient to compensate for the harm that is sure to follow."* These resolutions were overwhelmingly adopted by the Association; subsequent experience in a number of states, and notably recently in one in which an excellent system of central control has suffered considerable damage, thanks to a similar attitude on the part of some superintendents, leads one to believe that human nature has not changed greatly since the time of the founding fathers! While it is unfortunately true that in a considerable number of instances the stress has been on the material aspect of the operation of the hospitals, and that in some states a premium seems to have been placed, particularly by the lay Boards of Control, upon a low per capita cost, to the detriment of the standards of care of the patient, the development of the state hospitals has demonstrated in general the advantages of central control.

I am indebted to Dr. Grover Kempf, of the Mental Hospital Survey Committee, for data concerning the various types of central control which have developed and which are in force today.

* Am. Journal of Insanity, vol. 32, pp. 346-348, January 1876.

Before taking up the various states, mention may be made of the Federal institutions. There are under the Veterans Administration 25 neuropsychiatric hospitals which are very closely controlled from the central office at Washington, not only as to finances, but as to medical policies. The only other government mental hospital, Saint Elizabeths, is under the control of the Department of the Interior and is responsible to the Secretary of the Interior through a board of visitors appointed by the President of the United States.

Of the states, eight * still operate their hospitals through local boards of trustees appointed by the governor and responsible to him. Several of these states are decidedly small in population and some of them, indeed, have only two institutions, so that for practical purposes a local board of trustees is essentially the central control body. Three other states † have one board of trustees for all of the mental hospitals, the members of the board serving on a part time basis. Here again, in spite of the name, the board of trustees constitutes a central control body and has the advantage of providing uniform administration. One other state (Missouri) has a single board of trustees for all the institutions, one member serving full time on salary. A group of ten states ‡ have developed what are known as boards of control, all the members of which serve full time on salary, but who as a rule are not medically trained. It is interesting to note that only one of these states, namely, West Virginia, is located east of the Mississippi River. Two states, Michigan and Wisconsin, which were formerly in this group, have recently given up this type of administration for a central state department or commission. Of these ten states, one, namely Washington, is unusual in having a board of control made up of one member. These boards are, as has been indicated, usually lay, and sometimes the law requires that they shall be bi-partisan. Frequently the members are appointed for the term of the governor, and the type of administration in general is rather prone to admit politics and to stress economy, perhaps at the expense of

* Arkansas, Connecticut, Delaware, Louisiana, New Hampshire, North Carolina, New Mexico, Utah.

† Alabama, Mississippi, South Carolina. (The Alabama board is self-perpetuating.)

‡ Iowa, Kansas, Minnesota, Nebraska, North Dakota, Oklahoma, South Dakota, Texas, West Virginia, Washington.

the welfare of the patients. A group of six states,* rather widely scattered from Oregon to Florida, control the institutions through a central board made up of the governor and members of his cabinet ex-officiis. Obviously a board made up of elective officials can have no continuity of interest, and the very set-up renders the risk of political considerations high. A department of welfare or a bureau of a department not headed by a psychiatrist is found in nine states.† Some of these have been notoriously political in their operation of the hospitals, but this appears to be a difficulty which may be encountered in any type of central organization, or indeed in solitary institutions! Five more states ‡ have a department of welfare not usually in control of a physician, but with a bureau within the department headed by a psychiatrist which controls the mental hospitals. Two of these, Ohio and Kentucky, have been set up within the past two years. Three states § have a state hospital commission which in turn appoints a director (in one case *not* a psychiatrist), one (Maryland) has a conglomerate and unclassifiable arrangement with a commissioner, board of managers and a board of mental hygiene, and only two states || have a separate state department of mental health or of mental hygiene at the head of which is found a psychiatrist. Obviously this latter type of organization, which in many ways has much to be said for it, is available only for states of a large population, perhaps two and one-half million or over, or states which have a very considerable number of hospitals and patients. In those states which have a department of welfare there is considerable room for expansion and development, and if three hospitals or more exist within the state, a division of mental hygiene headed by a psychiatrist may properly be established within the department. One extremely interesting and not entirely explicable fact is that no state, so far as can be learned, has ever placed its mental hospitals within the department of health, which in most states is the only strictly medical department!

* Arizona, Florida, Montana, Nevada, Oregon, Wyoming.

† California, Colorado, Georgia, Idaho, Illinois, Maine, Rhode Island, Tennessee, Vermont.

‡ Indiana, New Jersey, Pennsylvania, Kentucky, Ohio.

§ Michigan, Virginia, Wisconsin.

|| Massachusetts, New York.

It is perhaps impossible to say for any given state what is the best type of central organization if a central organization is desirable. Obviously, in the smaller and more sparsely settled states a central body is hardly called for. In only the very largest states, probably, is a separate state department imperative, even though, as Noyes * points out, it is probably those states with a coordinate special department that have made the greatest progress in "modernizing their state institutions, in securing more adequate appropriations and in developing needed community services for the prevention and treatment of mental disease." He adds, "so far as practicable, therefore, the state agency dealing with mental health administration should be a separate department." Then, too, local conditions and situations constitute factors which must be taken into consideration and which may alter considerably the optimum type of organization. In general, it may be said that if a central organization is desirable an excellent plan is to have a representative board or commission of five to seven members, with staggered terms, thus avoiding a complete turnover during the incumbency of a single governor. Such an arrangement obviously reduces to a considerable extent the intrusion of political considerations. This board should have the authority to select a qualified director from whatever part of the country the best man may be secured. Frequently in setting up a new organization better results will be found if someone is imported rather than appointing someone who is so close to the situation that his perspective may be warped. The director, or commissioner, or whatever else his title may be, should be a well trained psychiatrist, a diplomate of the American Board of Psychiatry and Neurology, who has had considerable administrative as well as clinical hospital experience; perhaps even more important than his training should be his personality. He should be an effective speaker in presenting modern principles of mental hygiene to the public and to the legislature; he should be a man of vision and progressive ideals; above all, he should be a man who can be trusted by his fellows to do what is best for the hospitals, rather than for himself or for political

* A. P. Noyes: Purposes of a Centralized State Administrative Organization—Symposium on Mental Health, American Association for the Advancement of Science, Richmond 1938.

aggrandizement, and who can deal with his colleagues and the public on a basis of esteem, affection, respect and mutual helpfulness. Those of us who were privileged to know the late Dr. George M. Kline realize full well that he embodied these ideal qualities, which are all too rare. Fortunately for the progress of psychiatry, there are still those who share his ideals of service, progress and square dealing. The board should have the power to formulate general rules and regulations upon the recommendation of the director, which would be binding upon the several institutions. Finally and importantly, a state-wide civil service merit system is desirable if the proper sort of personnel is to be attracted and held.

A word should be said about the place of local boards of trustees in states where some type of central control exists. A number of states in setting up their central board have abolished the local board of trustees. However, I am convinced that the local boards of trustees have an important rôle to play even though a strong central control organization exists. They should be, and frequently are, made up of representative citizens of the community, men and women with a public interest, willing to give of their time and services. In the public mind they represent the public, and being unpaid may properly be looked upon generally as more truly disinterested than any paid public official, no matter how high his ideals may be. The trustees may well be given advisory power and to some little extent may be allowed to share with the central board the responsibility for the government of the local institution. The final decision in matters of policy should rest with the central board, but there is no reason why cordial relations should not exist between the trustees and the central authority. Most importantly, they stand as an added bulwark against political interference with the several hospitals and can, if they have those ideals which the boards of trustees in the time of Isaac Ray obviously had, be extremely helpful in upholding high principles of administration in the event of public criticism or if the inroads of spoilsmen threaten.

It may be helpful to summarize rather briefly the types of activity which may suitably be undertaken by well organized central administrative departments, functions which may be referred to as

supervisory, coordinating and integrating. Although the medical care of the patients naturally comes first in the operation of mental hospitals, the administrative aspect certainly can not be overlooked.* It is only proper to remind this group that this Association started as an organization of state hospital superintendents, men who were deeply interested in the administrative problems as well as in the welfare of their patients. At times in recent years it has seemed to a group of the members of this Association that too little reference has been made to the various administrative functions of mental hospitals, and it is partly for this reason that this symposium has been organized today. Let us consider first, then, some of the administrative activities and functions which may well be exercised through a central department. First of all, perhaps, come the budgetary requirements. Experience has shown that unless some type of central supervision of budgetary requests from a number of mental hospitals is exercised, certain institutions may get more than their share of funds, whereas others represented by less influential trustees or other protagonists will be somewhat slighted. Favoritism is prone to develop without a central check, and standards of care depending in large measure on the sums available, tend to become and remain discrepant in the several hospitals. The budget requests should be prepared by the superintendents of the various hospitals, and due consideration should be given to the special needs of the various institutions, their traditions and their special activities. The central control, however, furnishes at least a rough standardization and tends to promote uniformity. In this manner, furthermore, the budgetary requirements of the hospital may be brought into line with the general fiscal policy of the state.

Of equal importance is the matter of construction. Building plans should be elaborated in cooperation with the hospitals in question and in conformity with the local needs as expressed by those who are most familiar with them and who are, so to speak, on the grounds. A uniform basis for estimating capacity should be set up as a guide to planning. Further than this, long range planning not only as to the several existing hospitals but as to new

* A helpful volume on the general topic is "Administrative Psychiatry," by William A. Bryan, Norton, 1936.

institutions for the mentally ill and for the feebleminded should be undertaken and an attempt made to foresee the development of the needs of the state.

Important corollaries of this function of planning are the collection and analysis of adequate statistics. These statistics should be gathered both as to the operation and finances of the hospital, and as to the patient population, with reference to trends, age distribution, diagnoses, length of stay and similar items of information. Comparative data showing the functioning of the various institutions with regard to such matters as visits and discharge rates and seclusion and restraint, may be utilized for the purpose of stimulating friendly competition among the hospitals. The very valuable statistical work which has been undertaken in New York under the direction of Dr. Horatio M. Pollock, and in Massachusetts under that of Dr. Neil A. Dayton, deserves special mention. Massachusetts, indeed, is unique in preparing at the central office the complete statistical reports of each of the state hospitals. Attention may be called to the detailed study now nearing completion under Dr. Dayton's supervision, subsidized by a large grant from the Rockefeller Foundation. When completed this study will furnish a mass of data regarding the epidemiology of mental disorder such as has never before been accumulated, and will be extremely practical not only in the State of Massachusetts but in other states as a guide to future planning.

In a number of states the central control board has instituted cooperative or central purchasing, and in many other states state purchasing bureaus have been set up. The savings from this type of purchasing are substantial, provided always that the specifications are agreed upon by the institution and that the final acceptance or rejection of the purchased article is dependent upon the approval of the institution. Central purchasing when organized on a strictly price basis may result in the long run in inefficiency; properly handled it can be a great advantage both from the points of view of economy and of efficiency. Uniform systems of accounting are highly important in order that statistics of costs may be comparable and in order that an adequate check on expenditures may be kept. The laws of many states properly forbid exceeding the amount appropriated, and frequently control from a source

outside the hospital is necessary to insure compliance with the spirit of the law. In some of the larger states successful supervision of farm activities has been instituted, with a substantial increase in the productivity of the farms and herds and other activities. This, particularly when coordinated with an adequate inspection and supervision of the food service, can be of great assistance to the institution. A uniform dietary or standard diet is highly desirable, provided it is made sufficiently flexible to meet local conditions. It is important from the budgetary point of view, as well as from the viewpoint of providing adequate and varied diets for the patients. Another activity is that of the supervision of the plant operation, notably the engineering department of the institution; the amount of coal consumed by the larger hospitals is very substantial and it is desirable to see that the proper degree of efficiency is maintained. All of these activities lay stress upon securing the full dollar's worth for the institution.

On the other side is the question of income for hospitals. Experience has shown that the determination of the residence of patients with particular reference to whether or not they should be sent to other states if they belong there, is most efficiently handled by a central organization which can very well combine with this function the collection of money for the support of patients in hospitals. In some states the ability to pay is determined by the court at the time the patient is committed. This is a highly unsatisfactory way of determining the facts, and furthermore is not sufficiently flexible to make provision for collections in the case of bequests or other unexpected sources of income for the estates of patients. A considerable number of states have bilateral agreements regarding the requirements for residents and the inter-state exchange of patients. These can be handled effectively only by state-wide organizations acting on behalf of the state with other similar state agencies. It is only proper that all patients sent to a mental hospital shall receive the best possible care available as early as possible. It is equally fair that where those patients or their families are able to pay, the state should be reimbursed in accordance with the ability of the family. Furthermore, it is right that states should be called upon to take care only of those patients who have an adequate claim upon the institutional

provisions of that state. This question, however, is one which should be determined *after* the patient arrives at the hospital rather than before. In some states, unfortunately, the patient who cannot prove a state residence is cared for in a correctional institution or county infirmary rather than being sent to the hospital. Such an arrangement hardly comports with modern concepts of humane care.

Finally, on the administrative side a central organization would seem to be the logical agency to recommend changes in the laws in accordance with local needs. Certainly no better agency can be conceived that is equally familiar with desirable legislation concerning such matters as the commitment of the mentally ill and defective, and other provisions of law relating to their care. It is to be expected that the legislature would pay considerable attention to the recommendations of such a body.

On the medical side, coördination and integration would seem even more important for the welfare of the patient than the activities which have heretofore been enumerated. All activities of institutions, no matter how remote they seem from the patient, affect his welfare. It is a corollary which follows necessarily that a psychiatrist should be in complete charge of the individual hospital as superintendent, and perhaps *a fortiori* a psychiatrist should be in charge likewise of the state's coordinating activities as director or commissioner. It is an aphorism that bricks and mortar do not make a hospital, but rather the men and women who care for the patients. The most important single problem in mental hospitals today is that of personnel, particularly medical and nursing. A central department can well set up standards for initial appointment and for promotion, preferably under civil service, particularly if the civil service of the state is intelligently and non-politically operated. The personnel of the hospital should be made to realize that they are part of a system and that their eligibility to promotion is not confined to the institution in which they are working. In such a manner the prospects for promotion are considerably increased, likewise serving as an added force for a healthy morale. Uniform and adequate salaries should be established by the central organization, as where institutions set their own salaries one hospital is likely to be put in the position of com-

peting with another in a manner which does not conduce to the best spirit of team-work. Dr. Ebaugh, in the paper which follows, will deal not only with the preliminary training of physicians, but with post-graduate training and the establishment of training courses in state hospital systems. Such courses have been set up in a number of systems with great advantage to the hospital and to the patients cared for by them. The appointments in the various hospitals should be made entirely by the superintendent, with the approval of the board of trustees, if such an organization exists. The practice of assigning the employees to hospitals from a central source such as the governor's office, a state employment bureau or the central control body, is a pernicious one; it tends to break down morale and to decrease the feeling of responsibility of the employee toward the head of the institution, and should accordingly be discouraged. As for the superintendent himself, the qualifications should be high, and set, if not in the law, by the central body. Consideration may well be given, as is already the law in Massachusetts, to a requirement that the superintendent should be a diplomate of the American Board of Psychiatry and Neurology. Certainly anyone who is to have the control of a hospital in which many mental patients are cared for should have met the strict requirements of this board, a very important share in the formation of which was exercised by the American Psychiatric Association. Where a board of trustees exists they should have at least a veto power over the selection of a superintendent. His appointment should be "during good behavior," and not for a term as in certain states. It cannot be repeated too often that the greatest bane in the appointment of state hospital personnel is the intrusion of partisan politics. Unless the central organization is reasonably non-political and secure, and unless security of tenure is provided to the superintendent, it is all too clear that the best type of individual will not be attracted to these important positions. In all too many states it is still the case that a new administration at the State House means a "cleaning out" from superintendent on down in the various hospitals. Clearly, it is the patients who suffer by an arrangement of this sort. Some states in recent years have shown improvement in this regard, but it is to be regretted that certain others have exhibited a tendency to

regress toward a frank spoils system in the mental institutions. The individual initiative of the superintendents should be encouraged—they should not be forced to conform to too rigid repression of individual and progressive ideas. There is a danger of stifling such initiative if too centralized a control is exercised, and such a danger should be strictly avoided.

Standards of medical care should be set up for the purposes of uniformity involving such matters as clinical records and rules regarding the limitation and recording of restraint and seclusion. In some states the matter of family care, something which has recently received considerable attention as a result of economic pressure, is dealt with by the central organization rather than by the various hospitals. It would seem that if family care is a proper method of treating mental patients, it can properly be handled by the separate hospitals which are responsible for the welfare of those patients. It is questionable whether any central organization needs to be called upon to supervise family care.

Again, it is a helpful thing for patients in hospitals to feel that there is a higher authority to whom they may appeal in the event that they consider themselves to be unfairly dealt with. The complaints of patients are not always unfounded and cannot always best be investigated by those involved in the allegation. Investigation not only of alleged abuse, but of alleged wrongful detention, is a matter which is properly handled by a central organization. Furthermore, such an organization can well act as a sort of court of appeal for the release of patients about whose dangerousness there may be some doubt. Any hospital should welcome the opportunity to divide the responsibility of releasing such patients.

Training schools for nurses may be developed by a central organization, since it is in a position to determine which institutions are best equipped to operate such schools. Curricula and standards may be set up, not only for nursing care, but for social service and occupational therapy training, and the work unified and coordinated.

Another function of the central organization consists in transferring patients from one hospital to another, making adjustments of population dependent upon crowding of institutions, and setting up hospital districts in order to provide a more equitable distribution of hospital loads.

The supervision and the licensing of private hospitals is an important activity of a central control body. In altogether too many states private mental hospitals are unsupervised, with the result that some are found to be operated by wholly unqualified persons. Under such circumstances the hapless patient receives a minimum of attention, very little, if any, therapeutic interest is taken in him, and the prices charged may be wholly inordinate. Only private institutions which are licensed and regulated by the state should be permitted to hold themselves out for the care of the mentally ill and the mentally defective. Supervision can thus be maintained not only upon the physical features, such as the fire hazards, but upon the type of care rendered, the existence of undue amounts of seclusion and restraint, and the qualifications of the physicians in charge. From the public point of view it is equally important that there shall be some sort of control in order to avoid the suspicion that patients are committed and detained for ulterior reasons. "Railroading," as it is colloquially termed, is virtually unknown, particularly in public institutions; nevertheless, it is not enough that there shall be no such conspiracies—the public must be satisfied that there are none. Inspection and supervision of private mental hospitals by the state can do much to assure the public on this score.

The maintenance of the standards of care set for the hospitals, public and private, may be assured by reports and by the establishment of an inspection service. The inspectors should be well trained, mature and tactful, and while alert should avoid the tendency to "snoop." Incidentally, such a service, while of value to the central body, is likewise useful for the training of the psychiatrists selected for that assignment.

The importance of extra-mural activities has grown almost beyond belief in the past two decades, and is still growing. Experience has shown, however, that if the organization and maintenance of out-patient clinics of one sort and another is left to the individual hospitals, some institutions will neglect or will not attempt this work, whereas in other instances, unless there is central control and planning, the functions of the several hospitals may overlap. In one or two instances an attempt has been made to organize the state in such a way that the central body would

have control of the clinics only but not of the hospitals, and it is understood that at least one other state is now contemplating such a change. The desire of state hospital superintendents to avoid the supervision of trained central bodies is perhaps understandable, even though the preponderance of evidence seems to point towards the desirability of such oversight and assistance. There are other reasons, however, why the clinic program cannot well be divorced from the administration of the hospital. In the first place, the training in extra-mural psychiatry is extremely valuable for the members of hospital staffs. It directs emphasis upon the earlier and non-institutional types of cases and broadens the perspective of the state hospital physicians. Fully as important is the fact that by using the hospital as the nucleus much valuable educational work can be done in the community which will result in breaking down still further the barriers which exist between the community and the hospital, and toward removing the supposed stigma which attaches to care in a mental hospital. Again, the state hospital may, by organizing clinics outside of the institution, accomplish the added incidental purpose of providing a place where patients on visit may report for examination and consultation. Many community clinics started with this as the primary function, although such limitation is far from desirable. The great importance which has been attached in the public mind to mental hygiene and particularly to the preventive aspects of mental hygiene activities, assures a ready welcome for the establishment of such clinics in almost any community. The hospitals may be assigned districts and serve as focal points for this important service. In this manner too the expense of travel may be considerably curtailed. The clinics fall into two general groups, those for children, that is, in the nature of child guidance or habit clinics, and those for adults. There is no need to expatiate upon the functions of such clinics, for they are well known to this audience. Massachusetts was probably the first state to start such a program on a state-wide basis in 1922 under the commissionership of Dr. George M. Kline and under the immediate supervision of Dr. Douglas A. Thom. Some individual hospitals in other states have been especially active, one of the most effective hospital programs having been that of Danville, Pennsylvania, under the late

Dr. J. Allen Jackson. The Delaware State Hospital, which is unique by having as its jurisdiction an entire state of small area, has done outstanding work in this field. These are mentioned only as examples, for in many states, at least 18, some services of this sort are being given under state auspices; notable examples are New York, Oregon, Massachusetts, Delaware, New Jersey and South Carolina. Similar fairly well organized programs are under way in Pennsylvania, Connecticut and Maryland. A reference to the issue of "Mental Hygiene" for January 1936, containing a list of the known community mental hygiene clinics, will indicate the astonishing spread and development of the clinic movement.* There remains still much work to be done along these lines, particularly in the field of public mental hygiene education, which can be well accomplished by central organizations as the importance of preventive activities becomes increasingly recognized by legislative bodies.

Another type of activity which is somewhat different and yet related, is that of school clinics. Here again Massachusetts led, beginning with the work of Dr. Walter E. Fernald as far back as 1914. Since 1919 a function of the central organization in that state has been the establishment of clinics in the public schools for the examination primarily of retarded children, but more recently for the examination of children not so much retarded as presenting behavior disorders. This is, therefore, a combination of a child guidance clinic with a clinic for suspected mental retardation. The state has been divided into districts, one such district being assigned to each hospital and state school. Up to November 30, 1938, 114,127 school children have been examined in the state of Massachusetts in these clinics. Along with these examinations the mandatory establishment of special classes has been provided by legislation, with the result that many of the school problems brought about by retarded and otherwise disordered children have been very considerably reduced. In addition, early cases of mental disorder have been detected and hospital care advised. Obviously such a program cannot be conducted on a state-wide basis without some form of central administrative body which will assign dis-

* The author is grateful to Dr. George S. Stevenson of the National Committee for Mental Hygiene for information on this topic.

tricts, establish standards of examination, and make the data uniform, comparable and available for study.

The entire problem of mental defect has been largely neglected in many states. There are reported to be about 84 public institutions for the mentally defective, with a population of over 43,000, but this number of inmates represents but a small fraction of all the feeble-minded. No state, indeed, provides institutional facilities for more than 10 per cent of its mentally defective. The majority, of course, do not require institutional care, but probably every state could report a substantial waiting list, if the facts were available. A state program, which can only be carried out effectively by a central administrative body, should include, according to Raymond: (1) early identification; (2) central registration of all known feeble-minded; (3) educational facilities in the public school system and in the institutions; (4) protection, particularly during the formative years; (5) segregation for those who are incapable of living at home; (6) supervision of those in the community (90 per cent or more) and those who are paroled from state institutions; (7) research into the causes and prevention of mental deficiency; and (8) public information. That the problem is primarily a psychiatric one need hardly be argued; that it is large, and capable of efficient handling only on a statewide basis is fairly clear.

As another example of activities whereby state hospital systems may be made available to other agencies of government may be mentioned the services to be rendered to the courts, both civil and criminal. Primarily it is the personnel of the state hospitals which will be used in making examinations and advising the court. Nevertheless, a central administrative body (where there are several hospitals) can mobilize these activities, making the assistance available through existing facilities and exercising a general supervisory control over the standards of examination and reporting. The valuable results of the Briggs Law in Massachusetts may be mentioned as a sample of the sort of service which may be rendered.

In the papers which follow Dr. Ebaugh will present some consideration of the functions of mental hospitals regarding research and the training of students and personnel. Dr. Hamilton, who

for the past three years has served as the director of the Mental Hospital Survey Committee, and who for 20 years has been at the head of the National Committee for Mental Hygiene's Division of Hospital Service, will present with Dr. Kempf some of the findings of the Mental Hospital Survey Committee which will serve to illustrate the very considerable discrepancies which will still obtain in this country in the care of the mentally ill among the several states. The present paper has been an attempt, from a rather partisan point of view, perhaps, to present the advantages to be derived in states the population, size and development of which justify it, of a central administrative organization for the control of mental hospitals, and some reasons for believing that the establishment of such control may result in increased efficiency of the hospitals both from the point of view of the community, and, most important of all, from that of the patient.

TEACHING AND RESEARCH IN STATE HOSPITALS.*

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The problem of mental disorder is so extensive as to give all thoughtful persons pause in its consideration. Only those who are acquainted with large state hospitals can have a grasp of the magnitude of this problem, and we might logically expect that those persons would be the most concerned with its solution. That this is not necessarily true is probably due to the fact that state hospital administrators and physicians are literally swamped by the mere physical needs of their patients. There is little time, energy, or money left for investigation of the fundamental psychiatric problems. However, there is general agreement that more bricks and mortar added to our state institutions are very little safeguard against the ever increasing number of mentally sick seeking admission, and that we must intensify our efforts to solve those problems of mental illness which make custodial hospitals necessary. Psychiatry differs little from the other medical sciences in the basic principle that without teaching and research, progress is limited or even absent; only throcgh advancement in the understanding of mental disease can we alleviate, and eventually even prevent mental illness.

The National Committee for Mental Hygiene [8] has given us a fairly clear picture of the present status of teaching and research in mental hospitals through its questionnaire to all state hospitals and its personal appraisal of some of them.† Of the 127 state hospitals considered in the report, 33 reported no members of their staff had research interests; 11 reported more than 70 per cent of their staff with such interests; the remaining hospitals fell between these

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† This survey was sponsored through a grant to the National Committee for Mental Hygiene by the John & Mary Markle Foundation to present the pertinent facts of the facilities now available in these institutions.

extremes. Even among those staff members who professed an interest in research, only a relatively few showed a *marked* interest in, and ability for, such work. With reference to facilities for research, the hospitals reported 40 per cent with good facilities, 24 per cent with fair facilities, and 24 per cent with no facilities.* The number of publications from any institution may serve as a tentative gauge of research pursued. Using this criterion, we note that not a single publication appeared during the last three years from almost half of the state hospitals, and that the median number of publications was about one per hospital.

Teaching facilities, too, vary markedly in the different institutions. One third of the hospitals reported having close university contacts; 22 per cent reported occasional contacts; 43 per cent reported no contacts. The provisions for undergraduate, interne, postgraduate and staff teaching will be discussed in greater detail later.

Many factors stifle teaching and research activities in state hospitals. Among these are attitudes and misconceptions relative to mental disease, inadequate staff, insufficient financial resources, lack of equipment and research facilities, the basic organization of the state hospital itself and certain important psychological factors.

If we consider these in greater detail, we note that among the attitudes and misconceptions relative to mental disease may be mentioned (1) the pessimistic view taken by the medical profession in relation to nervous and mental disease; (2) the notion that psychiatry can be and is isolated from general medicine; (3) the general stigma associated with mental disease; (4) the lack of any understanding of the mental and emotional life of man; (5) the view that any sum of money granted for the study of the mentally sick is wasted money; (6) the belief that a low per capita cost is the best evidence of proper administration. These attitudes are not necessarily those of persons responsible for the care of the mentally sick; however, such views held both in and out of state hospitals do tend to retard progress.

Inadequate medical, nursing and attendant personnel retards teaching and research programs in mental hospitals. Custody and emergency care have been the primary responsibilities of state hos-

* Twelve per cent of the hospitals did not respond to this question.

pitals; treatment and restoration have often been secondary. Accordingly, the size of the hospital staff has been limited to the custodial function, and most hospitals are understaffed even by this standard. The patient load per assistant physician should not exceed 150 according to the standards of The American Psychiatric Association; yet in 1937 the median state hospital had a load of 293.4 patients; seven hospitals had a load of 100 to 150 patients, while nine hospitals had a load in excess of 500 patients.

The nursing and attendant staff, too, is overloaded. In contrast to the accepted standard of eight patients per nurse and attendant, the patient load in the median state hospital in 1937 was nearly 12; only one fourth of the hospitals maintained the standard, while 18 per cent had patient loads double the standard. Hence, any research undertaken must often be done singlehanded, without the helpful cooperation of a nursing staff.

Aside from the quantitative inadequacy of the staffs of state hospitals, there often exists a qualitative inadequacy which is even more serious. We all recognize that while there are excellent men in state hospital work, in many cases this field is looked upon as simply providing temporary positions for men looking for something better. All too frequently, these men have an interest only in the custodial care of the patients; all too frequently, they have had little or no training in psychiatric work; all too frequently, they care not whether they receive any sort of psychiatric training. It is difficult to conceive of physicians so little challenged by disease that they fail to ask themselves its cause or cure, but if such a sterile condition exists, we need not only a teaching program in our state hospitals, but a radical revision of teaching in our medical schools.

Budgetary deficiency is one of the chief factors which militates against teaching and research. Inadequate funds mean a lack of facilities and a shortage of personnel for therapy, teaching and research. Inadequate funds provide salaries too low to attract competent men. Inadequate funds explain the failure to budget definite funds for research. With state hospitals now on an approximate budget of $140,000,000 a year, scarcely more than 1 per cent is set aside for research, and one half of this comes from foundations and other private sources. No wonder state hospital superintendents are unsympathetic to research projects which might increase

their budgets when they cannot find sufficient funds to care adequately for patient and worker.

A serious handicap which often arises from budgetary deficiency is the lack of adequate facilities for both teaching and research. As we have noted, a rather large percentage of the hospitals surveyed present conditions not favorable for the prosecution of research. However, this absence of adequate facilities cannot be urged as an extenuating circumstance in all cases, since not infrequently this is a result of, rather than the cause of, a lack of interest in both teaching and research. Not infrequently it is the apathetic attitude of the staff itself rather than budgetary deficiency which accounts for the relatively little progress in these hospitals.

The fundamental organization of the state hospital itself militates against teaching and research. State hospitals are organized to care for large groups of patients, many of whom become permanent residents since the yearly turnover is only about 35 per cent. Moreover, state hospitals are organized along the lines of low maintenance cost and the mere physical preservation of the patient. Fundamental psychiatric treatment and research may often be inconsistent with this type of organization. In addition, the interests of state hospital superintendents have often been along quite different lines. State hospitals tend to attract men capable in the field of organization and administration. Although these men originally may have research and teaching ability, little time remains for its development after engaging in the field of administration. In still other cases, men with few teaching and research propensities enter state hospitals in the belief that this field requires little facility in teaching or research.

Finally, certain psychological factors lie behind the apathy toward teaching and research. There is great suspicion on the part not only of lay people but of the medical profession with reference to the care of patients in state hospitals. This has often resulted in external antagonisms, and there is a more or less constant fear of political reprisals because of this persistent suspicion. This has led hospital people to be wary of stirring up more antagonisms through new procedures which might be termed experimental. In addition to this potent factor, there are other psychological influences which play a part. These are the deadening influence a large resident hospital population exerts on the staff, the hopelessness of working in

a place so overcrowded that facilities even for adequate segregation of patients are lacking, and the disheartening effect of large blocks of hopeless organic and functional cases.

However, although all these factors in state hospital organization and administration do militate against teaching and research, they are not necessarily insurmountable, as is shown by the hospitals which do excel in teaching and research. Even with all its shortcomings, the state hospital organization is the principal functioning unit in our attack on mental disease, and this organization has already accomplished much in this field.

Teaching in State Hospitals.

Let us now turn to a more detailed consideration of teaching in state hospitals. This teaching will be discussed under several headings: teaching of the undergraduate; training of the graduate and postgraduate; instruction and review for the hospital staff.

The teaching of undergraduates is often limited to the occasional medical student who finds his way to a state hospital during the summer months, and about 28 hospitals offer this training to medical students. These externeships are often open to criticism on the basis of inadequate supervision of the student, but they have the value that through them not infrequently the student develops such an interest in psychiatry that he later enters this branch of medicine. Some state hospitals, in conjunction with medical schools, offer demonstrations, clinical clerkships or residential clerkships for medical students. In all, 20 medical schools * use state hospitals to varying degrees for their psychiatric teaching; eight † of these offer resident clerkships of different lengths. In these latter hospitals, of which Worcester is an outstanding example, teaching is carried out through case assignments, ward walks and interviews, staff conferences, assigned reading, discussion of treatment procedures, history taking, child guidance, social service procedures, and legal and administrative considerations. However, the state hospitals that

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have either the time or personnel to make such clerkships an attractive part of teaching are entirely too few.

Up to the present time no interneship in a state hospital is approved by the American Medical Association,* but, according to the report of the survey committee, 43 per cent of the hospitals do offer training periods called interneships. The number of men in these so-called interneships is quite variable and may run as high as 75 per cent of the medical staff. However, the interneship should avoid specialization, and it is unwise for state hospitals to offer training designed to take the place of the general interneship, since in all probability the interne finds himself deeply involved in the psychiatric aspects of the patient long before he is adequately grounded in general medicine. This does not preclude the possibility that under the supervision of a strong clinical director, a worthwhile medical service could be organized both for the interne and resident.

One of the most effective means of training and selecting men for state hospital work is through the medium of the residency, and 53 of 180 state hospitals are approved for residency by the American Medical Association.[2] The residency should be considered a graduate discipline and a definite training for a specialty in contradistinction to the interneship which is more closely identified with undergraduate training. To supply this graduate instruction, there are not enough psychopathic nor adequately equipped private hospitals for training men who are needed in state hospitals and other fields of psychiatry. No one type of institution can assume the burden of training the psychiatric educator, child guidance worker, state hospital administrator, psychotherapist, juvenile court counselor, mental hygienist, or allied worker in the field of psychiatry, but the state hospital can and should have a greater part in this training program. The state hospital teaching program must be so developed as to offer to a graduate a progressive educational experience designed to enable him to become proficient in psychiatry and to give him the intellectual training necessary for continued development within this field. The hospital which has pro-

* It should be noted that this statement applies to *state* hospitals; certain mental hospitals financed by the federal government are approved for interneships.

vided such a teaching program will reap a three-fold benefit: (1) the improvement in the care rendered its patients; (2) the increased number of good men desiring staff appointments; and (3) the greater interest of the staff in the patients and the hospital.

The development of this postgraduate training for residents and for permanent staff members is far from being as complete as we desire. However, some progress has been made in this direction. Massachusetts, New York, Illinois and Pennsylvania have provided an excellent type of postgraduate teaching for state hospital staffs. While the instruction and the organization of subject matter vary, these states achieve essentially the same goal of offering to state hospital staffs courses of instruction that create new interest in the field of psychiatry.

The program developed by the Massachusetts Department of Mental Health [5, 14] includes instruction in (1) subjects in which there are adequate facilities for teaching; (2) subjects which have the most practical applications in state hospitals; (3) subjects with which the average state hospital physician is likely to be unfamiliar and with which he has difficulty in acquainting himself without special aid; (4) subjects which are specifically required by the American Board of Psychiatry and Neurology. The first courses were given originally as "refresher" courses for those who anticipated taking the Board examinations; they proved so popular that later seminars were developed to include other subjects.

Under the supervision of the Director of the New York State Psychiatric Institute and Hospital,[7, 12] a well-rounded program has been developed.* This consists of (1) intensive courses covering a great variety of subjects related to psychiatry given at the Psychiatric Institute for ten weeks for state hospital men and other interested physicians; (2) semi-annual interhospital conferences on current problems for state hospital men; (3) quarterly conferences, chiefly on administrative problems, for superintendents and directors.

The Psychiatric Institute at Chicago, under the direction of Singer,[10] offers courses twice a year of two months duration to state hospital men. One or two physicians from each state hospital

* This program had its inception in the postgraduate courses originally organized in 1902 by Adolf Meyer when he was Director of the Pathological (Psychiatric) Institute, New York State Hospitals.

are given the opportunity to attend one of these courses; however, the more recent appointees are favored. In this period of training, the physician is responsible for the work-up of cases and their presentation at staff meetings. Seminars in special clinical methods and problems are held in addition.

The University of Pennsylvania Graduate School of Medicine [6] has conducted postgraduate courses for staff members of the various state hospitals since 1923. The principal course of instruction, covering a number of pertinent subjects, is given for two months; in addition, lectures are conducted at the school throughout the year on neuro-psychiatry and allied subjects.

These training centers in Massachusetts, New York, Illinois and Pennsylvania promote closer cooperation between state hospital staffs, improvement in therapeutic procedures, increased interest in research, higher standards for personnel, better discharge rates and recognition by the rest of the medical profession of the opportunities and responsibilities found in a state hospital. These training centers also serve as models for the establishment of similar units in the rest of the country.

Certain other states have also taken steps towards the organization of training centers. For instance, courses have been organized for the Baltimore district in Maryland. These courses are given for two hours once a week from November through June. In Ohio, a three to four weeks' postgraduate course in psychiatry is given in connection with the Ohio State University and the Columbus State Hospital. One physician from each state hospital is assigned to this course. The University of Minnesota has organized courses in neurology and psychiatry through its Center for Continuation Study. The exact details of these courses are unknown. Postgraduate courses are soon to be offered to the hospital staffs of all California hospitals; however, this organization is as yet incomplete. The Department of Postgraduate Medicine of the University of Michigan is organizing a series of short review courses together with a few short courses in the special fields of practice to be offered to the general practitioners of the state. Even though this work at Maryland, Ohio, Minnesota, California and Michigan is commendable and very worthwhile, it is not yet comparable to that of the better developed programs of Massachusetts,

New York, Illinois and Pennsylvania, and cannot be considered adequate from a postgraduate standpoint.

It is probably true that the majority of state hospital superintendents are anxious to strengthen postgraduate teaching for their own staffs, but on the whole they are confronted with the difficulty of supplying this instruction. With this in mind, the establishment of institutes organized through the Committee on Psychiatry in Medical Education of The American Psychiatric Association was suggested as a means of bringing to each state hospital some type of instruction which would enhance its position as a teaching center, establish a better relation with medical schools, improve research, develop out-patient departments and community organizations and improve all forms of therapy. It was suggested that these institutes be composed of four qualified men representing various aspects of psychiatry. Certain strategic areas could be designated from which the state hospital men and probably the teachers would be drawn.

In order to determine the demand for, and need of, such an institute for postgraduate psychiatric training, we addressed a questionnaire to 180 state hospitals, of which 160 or 89 per cent replied. This questionnaire attempted to ascertain among other things (1) whether state hospital superintendents are interested in such an institute, (2) the subjects to be considered by this institute, and (3) the locations preferred.

Exclusive of New York and Massachusetts, which already have well organized training institutes, all state hospitals answering the questionnaire except two were in favor of the organization of a postgraduate institute as it would correlate with their hospital programs. Even institutions in other states in which some form of postgraduate instruction is now offered were interested in the project. Of the subjects suggested for consideration, the newer forms of therapy were mentioned 79 times; clinical psychiatry, 69; clinical neurology, 62; psychopathology, 55; hospital administration, 46; organization for research, 44; neuropathology, 37; community organization, 33; legal aspects psychiatry, 30; outpatient department, 26; psychobiology, 2; and neuro-anatomy, 1. Most of the superintendents felt that if the institute were held in their respective states, many more men could take advantage of the instruction. However, California, Colorado, Illinois, Maryland,

Massachusetts, New York and Pennsylvania were suggested as strategic locations. This questionnaire was of especial interest and importance in showing that (1) practically all state hospital superintendents are anxious to offer postgraduate training to their staffs; (2) the majority of superintendents are willing to cooperate in any feasible plan whereby this can be accomplished; (3) superintendents are interested in a wide variety of subjects relative to therapy, teaching, research and administration.

Research in State Hospitals.

Singer [11] has said, ". . . . the prime function of psychiatric research is the discovery of the mechanisms that permit the development of an illness, no matter what the precipitating or concomitant circumstances," and Flexner [4] has pointed out that research and the scientific or intelligent practice of medicine employ exactly the same technique. Hence, every physician in a state hospital can and should look upon himself essentially as a research worker who investigates the etiology, pathogenesis and therapy of mental illness and who seeks to improve his own technique with which to accomplish this task. Clinical workers engage in research not only to perfect their diagnostic and therapeutic technique, but even more fundamentally, because of their lively interest in the dynamics of the field in which they are working. They have an intellectual curiosity linked with a driving desire to know more of the how and why of situations. Thus, the real limitations on research are those of the men themselves; the true barriers are often personality determined. At the present time we do not have sufficient information about the various psychiatric conditions to form even a common basis upon which all workers interested in this field may build, and the plea is made that state hospitals seize the opportunity to investigate any phase of behavior which may aid in therapy or in explaining the development of mental illnesses. Certainly the state hospital has unparalleled opportunities with its wealth of clinical material, and it is regrettable that in the day-by-day care and observation of some 600,000 mentally sick patients, such a relatively small amount of research is carried on.

Any paper which attempts to outline the "next" steps in research is undoubtedly bold; yet almost every current discussion on this

subject eventually falls into the trap of saying what should be done and the way in which it should be accomplished. Innumerable suggestions as to method and content of research have been made, and considerable discussion has arisen as to the most vulnerable aspect of mental illness to attack. For example, it is questioned whether we should stress the study of the child or the adult, the normal or the abnormal, therapy or etiology, organic or functional components, to mention only a few. Some authors feel that psychiatric research is conducted best by a number of skilled workers in the laboratories of the more exact sciences; others feel that the only logical laboratory for the study of mental illness is society itself. Some have suggested cooperative research with workers in allied fields as being the most efficient way of studying problems of the maladjusted; others hold that observation and experiment by individual psychiatrists produce the best results. The answers to these questions concerning the method and content of research lie between the various extremes and yet encompass all of them. No one project for research or no one method of attack can meet the multiplicities of the problems of mental illness.

The problems suggested for research are as numerous as the writers themselves. Among these are the part played by genetics and heredity as contrasted with environment, the importance of the chromosomes and cell structure, the nature of the nerve impulse, the relation of hormones and enzymes to emotion, the chemistry of the brain, and the type of experience enjoyed by one individual as contrasted with another. A special problem which deserves attention in any research program of state hospitals is a systematic follow-up of all cases. State hospitals should know more about the type of adjustment former patients make when returned to society. In this way we may learn the factors which permit a person to remain well following his discharge from a state hospital. From studies [9] already conducted on a systematic follow-up method, it appears that satisfactory studies can be made only through a thorough clinical examination by a competent observer. Other methods commonly used in follow-up studies, such as social investigation and the questionnaire, while valuable, have definite limitations. Perhaps the most important problem in the field of psychiatric research concerns the mechanisms that permit the development of an illness irrespective of the precipitating or concomitant cir-

cumstances. The factors that go into the formation and evaluation of behavior before it is transposed into dissolution seem the logical starting point to determine why people become mentally sick. It seems premature to speak of regression until something more is known of progression. This problem is so great as to encompass much of human behavior, and no one profession or group of institutions can do more than simply share in the work on this problem. The answer to this vital question, if found at all, will eventually be found beyond the walls of a state or psychopathic hospital, but these institutions too must participate in the search.

Such studies as have been outlined above often mean the utilization of many disciplines outside the field of psychiatry. They mean the cooperation of all the sciences relating to the welfare of the human being. Because this field of personality adjustment is so broad, research in human behavior becomes the task of each discipline concerned with man. This means more than investigating simply the socio-economic factor, the educative factor, or the mechanisms of repression, insecurity, hostility or anxiety; it means the study of life and all its ramifications. For this work we need trained observers and skilled workers in science. Such research would result in the synthesis and coordination of facts discovered in the different fields of human behavior; it would promote a better understanding of the other sciences and their influence in related fields; and it would result in a more tolerant attitude toward workers in other fields who may make important contributions to the understanding of mental illness. An understanding of the ramifications of the study of mental disease necessitates a broader horizon on the part of state hospital physicians; they must become better acquainted with the value and rôle of their coworkers in medicine, theology, education and the home.

At the present time the whole field of psychiatric research, in common with many other fields, is without any central organization. No one has attempted until this survey by the National Committee for Mental Hygiene to set down the problems being considered. It is unlikely that any one individual or agency could possibly organize and direct all the problems which need investigation; however, because extensive research in personality adjustment often requires the cooperation of more than one discipline, and because the whole program of research in mental hospitals would be guided

and strengthened by some form of central organization, many leaders in this field have suggested the formation of a Council on Research. This is not new to any of us. It has been suggested and recommended by nearly each president of this association; it has been urged by practically every one who has written on psychiatric research; outstanding in this respect are the reports of Chapman,[1] Fitzpatrick,[2] and Whitehorn and Zilboorg.[18]

The value of a central organization for research cannot be denied. The Council would know not only the facilities of each hospital, but also the projects under investigation. Further, it would have as one of its major functions the gathering of data on the true extent of mental diseases. While a fairly clear picture of the etiology and epidemiology of the majority of medical conditions exists at present, this is scarcely true with reference to mental illness. There are few studies available wherein whole communities have been surveyed for the purpose of determining the prevalence of psychotics among the population. The Council could serve logically as an integrating unit between state hospitals for the purposes of solving mutual problems, of coordinating and guiding research and of correlating the teaching in medical schools and state hospitals. Furthermore, it could organize postgraduate teaching for state hospital staffs; help supply this teaching personnel; coordinate the work of the other disciplines to psychiatry; enlist private and public funds for furthering research activities in public institutions; investigate the contributions which psychology, sociology, anthropology and education can make in a frontal attack upon mental illness; encourage any and all types of research; maintain a bibliographical service for the use of any interested group; record all research work done to date and abstract all literature which pertains to psychiatric research; publish the activities of the Council through a quarterly journal as a supplement to the AMERICAN JOURNAL OF PSYCHIATRY.

No discussion of teaching and research in state hospitals would be complete without some mention of psychiatric or psychopathic hospitals. These institutions are state hospitals of a special kind, and they, too, are concerned with many of the problems which face other state hospitals that we have already considered. No one can question the influential part these institutions are playing in teaching, therapy, research and prevention of mental disease. Situated as these units are as a part of medical schools, they play a unique

rôle in present-day psychiatric thought in developing concepts of personality and its deviations. In fact, it is questionable whether psychiatry can be taught adequately without the aid of this or some similar unit in which the clinical material receives careful and complete study. In addition, because of its important relation to medical schools, the psychopathic hospital may prove the best integrating agency between medical schools and state hospitals. The staffs of the psychopathic hospital and state hospital speak the same language and together their needs may be better articulated to outside groups.

Summary.

In this paper we have discussed the present status of teaching and research in state hospitals. We have outlined the teaching programs in various state hospitals and have attempted to evaluate these programs. We have discussed research in state hospitals from the viewpoints of its present status and its rôle in the solution of mental disease, and we have mentioned a few problems of special importance. We have considered the need for, and the advantages of, some central organization for research, and we have listed some of the functions of such a central council. Finally, the importance of the psychopathic hospital as a liaison between state hospitals and medical schools and as an almost indispensable unit in the teaching of psychiatry has been stressed.

We have found that some very worthwhile work in teaching and research is being carried on at the present time; that some hospitals are alert to the need of teaching and research in a progressive program; that such work in state hospitals is often carried on under great handicaps as to personnel, finances and equipment. However, we have noted that in far too many institutions, the physical conditions and the attitudes and interests of the staff are unfavorable to the development of teaching or the prosecution of research. While in many cases this lack of a teaching and research program is due to insufficient funds, in some it is certainly due to a mistaken adherence to the idea of the purely custodial function of the state hospital. We believe that the state hospital has much to gain from, and much to offer to, an extensive organized program of teaching and research.

We are mindful that state hospital administrators may object that our program is too ambitious; that it is beyond the realm of possi-

bility. Also we are mindful that some will emphasize unduly their financial and personnel handicaps. While we are cognizant of these and other criticisms, we abhor taking a temporizing position in this discussion by merely patting everyone on the shoulder and pleading for more and better work. We beg instead for cooperation by all those concerned to develop their teaching and research programs in order that they may make a worthwhile contribution to the solution of mental disease. No one can question that within a short time, if indeed it is not already true, state hospital administrators will become more cognizant of their position as directors of teaching and research institutions. This means changes in personnel and organization, and a shifting of the emphasis on the functions of a state hospital. Naturally the state hospital must take the lead itself in initiating and developing these changes.

BIBLIOGRAPHY.

1. Chapman, Ross McClure: Am. J. Psychiat., 95: 14, 1938.
2. Eighteenth Annual Presentation of Hospital Data by Council on Medical Education and Hospitals, J. A. M. A. 112: 909, 1939.
3. Fitzpatrick, Charles P.: Symposium on Mental Health. Am. Assn. for the Advancement of Science, Dec. 1938. In press.
4. Flexner, Abraham: Medical Education in United States and Canada. A Report to the Carnegie Foundation for the Advancement of Teaching. Bull. No. 4, 1910, p. 59.
5. Halloran, Roy D.: Personal communication.
6. Klopp, Henry I.: Personal communication.
7. Lewis, Nolan D. C.: Personal communication.
8. Research in Mental Hospitals, National Committee for Mental Hygiene, New York, 1938.
9. Romano, John and Ebaugh, Franklin G.: Am. J. Psychiat., 95: 583, 1938.
10. Singer, H. Douglas: Personal communication.
11. Singer, H. Douglas: J. A. M. A., 104: 2223, 1935.
12. Tiffany, William J.: Personal communication.
13. Whitehorn, J. C. and Zilboorg, Gregory: Am. J. Psychiat., 90: 302, 1938.
14. Yakovlev, Paul I.: Personal communication.

TRENDS IN THE ACTIVITIES OF MENTAL HOSPITALS.*

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AND

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In 1936 this Association with others set up a Mental Hospital Survey Committee. Its history is so recent that it hardly needs be repeated. The United States Public Health Service and The National Committee for Mental Hygiene contributed either personnel or money for running expenses. This Association financed additional field work. Other representation on the committee was from the American Medical Association, the American Neurological Association, the American Board of Psychiatry and Neurology and two Canadian organizations—the Canadian Medical Association and The Canadian National Committee for Mental Hygiene. The latter financed two surveys in Canada. Other funds came from communities and individuals. Surveys and shorter visits have been made in 45 states, the District of Columbia, and three provinces of Canada. A grant from the Rockefeller Foundation through which The National Committee for Mental Hygiene was able to participate comes to an end with the current June, and the present organization breaks up. Fortunately, the United States Public Health Service will salvage as much of the project as possible, and will continue the existence of the Survey Committee.

As regards the locale of our surveys, they have been made from Maine to California and from Florida to British Columbia. One hundred fifty-seven hospitals have been studied with care and 69 have been visited once or more and partially studied, making a total of 226.

The appropriation made by this Association financed surveys of 34 institutions in 16 states; 26 hospitals and 8 institutions for

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mental defectives as well. In several instances our official invitation took us to all the psychiatric institutions in a state including hospitals for mentally ill, hospitals for epileptics and schools for defectives. Detailed reports have been filed with the Council.

Some official invitations were routed from governors to the United States Public Health Service. Again appeals for surveys were made to The National Committee for Mental Hygiene or the Canadian National Committee for Mental Hygiene. In laying out our trips to reach all sections of the country we made free use of the fact that we are representatives of this Association, which has members in most mental institutions. Everywhere we were hospitably received, our requests met patient compliance, and when our reports had been prepared and revised along the lines acceptable to the executive group of our Committee, our recommendations were pleasantly received, and in many instances led to early action. The broad base of our organization, all these associations, was very advantageous and our voluntary constitution seems to have protected us from any suspicion of jealousy or self-seeking. Seventeen members or fellows of this Association participated.

It is common knowledge in this Association that the widest variation of standards exists in the mental hospitals of this country. The Association adopted in 1926 some standards, grouped under 19 points. A few of these will be mentioned in order to emphasize the great range of organization and facilities provided for the treatment of the mentally ill. A colleague said of one institution: "Time and seclusion seem to be the therapeutic agents that bring about recovery, although the general physical welfare of the patient receives fair attention." In contrast, many states maintain very fine hospitals.

Tenure of office in most states depends on professional qualifications, but there are several in which not only the political party but also the political faction favored by the appointee is more important than his ability to do good to human beings in his charge. This applies to physicians and to all other employees. In former days it was customary to dismiss the superintendent and depend on the new appointee to choose subordinates that would strengthen the local political machine. Of late a new technique evolved in Illinois has been adopted in other places as well. The medical staff are left in their positions, thus giving the institution a better appearance

before the public, but the appointment of many subordinates is dictated. One need not discuss where the loyalty of such people points, nor how patients are made to suffer from neglect and even brutality that such a superintendent is hardly able to control. There is a considerable number of poor institutions, and a very few of the better ones in which dual control exists, the superintendent and the steward being both directly responsible to the same appointing authority. Pennsylvania has now turned its back on this system.

Medical staffs we have found ranging from 1 :125.2 up to 1 :1011. The nursing personnel ranges from 1 :5.3 up to 1 :23.7. Dentistry is probably the most consistently well-supported specialty; not only is every medical organization interested in having good dental service for public patients but the dental organizations also exercise a weighty influence in this direction. Consulting staffs range from nothing up to an organization patterned on that of the general hospital where the monthly meeting of the attending physicians is a professional function of much consequence.

Many medical staffs are very well organized and the young physician can enter on his work with every confidence that he will not only learn from experience but that his work will also be carefully scrutinized by one or more senior physicians and that he will be guided towards the goal of professional excellence.

There is usually some sort of room on every medical service where patients may be examined but sometimes this room is shared with the attendants and with whoever comes into the ward on any business errand.

Clinical histories are of all grades. In some places very little is available except one statement from the patient himself; in others, documents are very fine bodies of information about the patient on which thorough understanding and adequate treatment can be based. Filing of histories in these days is usually satisfactory. The clerical help necessary in order to prepare them is sometimes more easily obtained than the physicians to make the observations.

Classification of patients is ordinarily fairly good but institutions have been seen where there was so much crowding that two or three transfers were necessary in order to place a single individual in his proper environment. Under such circumstances patients of discordant types get quartered together. Many hospitals have separate reception buildings, others designate special wards, and in a very

few there is no difference in the appearance of either the ward or the patient in the reception service and elsewhere. The adequacy of provision for the physically sick also varies greatly.

Not many hospitals are without clinical laboratories, but a few have been reported. There is a very widespread lack of staff members able to make pathological study. Some hospitals send tissues to other places but this procedure cannot be followed to any great extent for the study of nerve tracts. Radiology is fairly well looked after, but there are many institutions that ought to take more pictures than they can do now.

Medical libraries range from 7000 down to a few old books of only antiquarian interest. A hospital of over 4000 beds was found to be taking but two psychiatric periodicals and in some places it is reported that none are subscribed for. Other libraries are very well stocked.

Out-patient clinics are organized in various ways. Of course there are poor institutions that do no community work; others have an elaborate organization. The same is true of social workers.

Nursing is of course of all grades. It is hard to think of an institution as a hospital when it has not even one graduate nurse but a few such there are. In many places the graduate nurses have no previous psychiatric experience and no definite training in the mental hospital where they are employed. Their intelligence and their general training serve them in good stead; other institutions have a keen appreciation of the implications of the term "psychiatric nurse" and maintain a high grade of service for their patients.

Mechanical restraint and seclusion are very freely used in some places, and very little in others. The amount of such use depends of course on the standards of the physicians and many of our colleagues have never worked in a hospital where these measures were frowned upon.

In view of the considerable number of institutions studied and the mass of information obtained it has been asked that a report be made about current trends. Fifteen observations are offered.

1. The demands for hospitalization have been increasing for many years and the end seems not to be in sight. Several reasons are obvious:

(a) The average duration of human life has been materially increased, and a much larger number of persons live to the period in which they are subject to senile or arteriosclerotic psychoses. Not until the average human

life has been extended as far as possible will this cause of increasing admissions terminate, unless someone discovers how to prevent the psychoses of the senium.

(b) Hospitals are better than they used to be, and therefore more persons are willing to enter them. To some harried administrators it would seem that this is a penalty rather than a reward for bettering the equipment, personnel and administration of their hospitals. This situation is not limited to mental hospitals, for every other type of hospital is busy and often overcrowded.

(c) Urbanization still extends and the person who acts queerly has less opportunity of escaping unfavorable attention from his neighbors.

(d) The economic status of families is changing. Fewer have their own premises, and not infrequently two families live together because employment is low. Under such circumstances a person who cannot contribute to the family life and perhaps to the family income is unwelcome as a member of the household; accordingly patients who might under better circumstances be cared for at home are sent to the hospital.

(e) Great economic depressions increase all the frictions of the family, exaggerate individual tensions, and thus accentuate the trend towards hospitalization.

Until we know where the increasing average of human longevity will end we cannot begin to estimate how many beds we shall need.

2. Overcrowding increases in many places. Institutions that formerly were comfortable residences for their patients have succumbed to the rising tide of admissions. The figures offered in many states are unscientific and quite misleading because wherever a bed is placed that much capacity is assumed. On the other hand the best organized states and the federal government have definite standards of floor measurement on which figures of capacity are calculated. Taking the country-wide figures as given, the overcrowding is said to have amounted in 1936 to 11.4 per cent. Probably this is to some extent an underestimate, for any inaccuracy is on the side of claiming too great a capacity rather than too small.

Overcrowding has cursed many institutions, broken down one function after another and decreased the effectiveness of treatment. Certain commonwealths in spite of the depression have made a serious and apparently successful effort to overcome overcrowding: Delaware, Illinois, New York, Mississippi, Louisiana, Montana.

The following named hospitals recently had empty beds:

Delaware State Hospital, Delaware, Boston State Hospital, Massachusetts, Evansville State Hospital, Indiana, Ypsilanti State Hospital, Michigan, Eastern State Hospital, Washington.

Of course this condition may change from month to month.

It may be remarked that the veterans' hospitals are seldom overcrowded by a single bed. This is because a new patient is not received unless there is a place for him. The same policy is adopted by some of the states, and no admissions are permitted until vacancies have developed. This rule works great hardship in the case of the state hospital, for the patient may have to go to the almshouse, or more likely to the jail to wait; some years ago 88 patients committed to state hospitals of one large state and waiting for admission died in the jails during a biennium. No great hardship is inflicted by the Veterans Administration in deferring the acceptance of a patient, because every prospective patient is a resident of some state and can probably be cared for in a state hospital until the federal government is prepared to receive him.

3. The growth of institutions is rapid, and their size too often becomes huge. This process, which is welcomed in many important parts of America, creates some practical difficulties for the patient. Some part or other of the physical plant is forever becoming inadequate as the institution grows beyond its plan. The kitchen becomes too small, the storeroom and the heating plant; sometimes the water supply threatens exhaustion. Worse than this, many patients come to have less individual attention. It is said that somewhere around 1500 patients the superintendent starts to lose track of those in his charge. When a member of our own family is ill we do not consider it unreasonable to expect that the patient's main symptoms shall be known to or at least reviewed by a physician whose standing in the community rates him an eight or ten thousand dollar practice. So in a mental hospital it is not unreasonable that the head of an institution (whose salary and maintenance may be rated at about ten thousand dollars) shall review the case occasionally, shall be informed of emergencies, and shall keep a check on the type of treatment employed and the type of personnel assigned.

The large hospital is characteristic of the time and patients tend to become cogs in a factory-like machine. One senses a difference in the reception service of the large and the small hospitals. The physician on the smaller service knows his patients as human beings, not as mere items in a schedule, and maintains a personal rapport, so important to the patient.

We have divided the public mental hospitals according to size, using the figure 1500 beds as a measuring rod. We find that hospitals of 1500 or less number 46, of 1500 to 3000 number 90, of 3000 to 4500 are 26, of 4500 to 6000 are 7, and those over 6000 are already three in number, in the states of New York and Georgia. The wealthier states put their salary schedule on a level where very experienced and well trained men are in charge of all important divisions of the hospital. Even such men have been known to get bogged down in a big service with many patients and few assistants.

4. More physicians and more employees of many grades are engaged in the care and treatment of the mentally sick in hospitals. A mere increase in personnel, however, may not signify an increase in the ratio of physicians or other employees to patients. Indeed there are probably some hospitals in which increases have been made in numbers but the ratio has grown poorer. The declarations of this Association, made in 1926, become effective at a very slow pace. We must bring repeatedly to the attention of governors, commissioners and members of appropriations committees that the minimum ratio of assistant physicians to patients should be 1 : 150, and the minimum ratio of nursing personnel to patients should be 1 : 8. The general physician-patient ratio is improving, as the following table shows:

Year.	Patients.	No. of state hospitals.	Total physicians.	Assistant physicians.	Ratio of patients to assistant physicians.
1923.....	225,430	153	962	809	278.7
1926.....	245,997	155	1,108	953	258.1
1936.....	357,861	171	1,600	1,429	250.4

The same advance may be noted in the ratio between patients and nursing personnel as the following table shows:

Year.	Patients.	No. of hospitals.	Nurses and attendants.	Ratio of patients to nurses and attendants.	Percentage graduate nurses.
1923.....	225,430	153	19,643	11.5	9.2
1926.....	245,997	155	21,687	11.3	8.3
1936.....	357,861	171	35,187	10.2	10.1

In a few places, as shown by the following table, the hospital standard is still better than these ratios, and in that fact one may feel satisfaction.

Hospitals.	Assistant physicians to patients.	Nurses and attendants to patients.
Delaware State Hospital	1 : 125.2	.....
St. Elizabeths Hospital, Washington, D. C.	1 : 135.7	1 : 5.3
Boston State Hospital, Massachusetts	1 : 143.7	.....
Utica State Hospital, New York	1 : 147.2	.....
Massachusetts	.....	1 : 6.6
New Hampshire	.....	1 : 6.9
New York	.....	1 : 6.7

5. There is a definite trend on the part of subordinate physicians to expect something more than room, meals and laundry. There is a widespread demand for training. In many parts of the country this focuses on the requirements of the American Board of Psychiatry and Neurology. In several regions special courses of instruction have been organized for the benefit of assistant physicians who wish to take these examinations. The same desire for greater knowledge has also been found in regions where the imprimatur of the American Board is not valued. This is a very heartening attitude on the part of our younger colleagues and in my opinion it should be encouraged by this Association in every possible way. We could not tolerate a situation in which the surgeon, the ophthalmologist, the roentgenologist and even the internist would be striving to acquire broader knowledge, but the psychiatrist would be content to make his rounds, attend to the incidental ailments of his patients, and remain uninclined and unequipped to give them the best available psychotherapy.

6. The tremendous need for study of causes as well as effects of mental disease is no secret to the physicians who direct the activities of our mental hospitals. It must be admitted that the word "research" is loosely used in many places. One even finds hospitals in which someone thinks that any physician who works in a laboratory is doing research. Nevertheless there is a distinct trend towards development of genuine research. Biochemical study has fired the fancy in more places than any other type of research, but as was shown in a recent study of hospitals in all parts of the country research is being prosecuted in various directions. In some

institutions the dream of doing research is apparently an effort to look away from the inadequacies that have been forced upon the physician by parsimonious legislatures. The traveller finds here and there a new plant or some new payroll positions allocated to research. In this connection there seems to be an increasing number of hospitals that have developed and prize a university connection. Some of the best hospitals, and particularly the small ones closely associated with schools of medicine, are well equipped with personnel and apparatus to probe carefully and thoroughly any project that it may seem wise to undertake.

7. Nurse training seems to be more difficult and in my opinion we are slowly losing ground. There seem to be few places where a full course is not given now but is planned for the near future. There are institutions that are likely to drop their full course as soon as employment conditions again become more difficult. More affiliate pupil nurses are coming in for a two-, three- or four-month course, and this is to the advantage of nursing education but is not so important for the proper care of our patients as is the full course of training. There is a tendency to employ more graduate nurses but it has to be done without much regard to the standards of psychiatric service that these young women have. (See previous table.)

The formal training of attendants proceeds very well in some places, but does not go ahead in others. There is perhaps a slight trend towards emphasizing it and towards strengthening the courses given.

8. The trend towards the development of special therapies in competent hands goes back some years. It is still strong. Occupation therapy, powerful or feeble, is found everywhere. Physical training is much more used than formerly, and superintendents who already have an occupation therapy department not infrequently say that they must as soon as possible get a salary for a physical training aide.

The introduction of hypoglycæmic treatment came just before this survey started, and we have watched with great interest the spread of the practice all over the country. There are still some hospitals that do not employ it. In certain places it has been an acid test of the capacity of the physicians and nursing staff. It is scandalous that there should be institutions named hospitals by their state legislatures that do not have enough physicians or nurses to

institute a type of treatment that is likely to make a material improvement in the condition of its patients. No matter what be the ultimate decision as to the value of hypoglycæmic treatment for the individual, its value to some hospitals has been great because it has focussed attention on weaknesses in organization and has led to their correction.

Metrazol treatment also has been widely used. The simplicity of its administration made it easier to institute in understaffed hospitals. The care with which it has been administered in some hospitals and the crudity in others has been enlightening to the visitor.

Prolonged narcosis is used in a considerable number of institutions but is not popular. Unless proper psychotherapy is added the procedure seems to be disappointing and hospitals that are weak in psychotherapy are unlikely to make use of this treatment. Hospitals that are relatively strong in psychotherapy sometimes think that prolonged narcosis adds little to their existing effectiveness.

9. Psychotherapy in one form or another has always been practiced by able physicians. There is a tendency to organize it better now. General psychiatry owes much to psychoanalysis in this regard. Indeed there are several hospitals that have one or several competent psychoanalysts on their staff. Such hospitals are of course in a small minority but there are many whose older staff members are quite competent psychotherapists.

10. Pathological work tends to be more actively pursued. The spur to clinical pathology seems to have come largely from the American Medical Association. There are far too many institutions that have little or no equipment for neuropathology. Every full-time pathologist is likely to develop his laboratory for procedures in this special field.

11. The demands upon the state hospital staffs for advice and treatment of patients young and old who are not and perhaps never need to be within the hospital are always in excess of the amount of time that can properly be taken from the needs of the patients in the institution. Generally speaking, the trend of twenty years ago to meet these requests continues unabated. One finds various hospitals extending their out-patient service. The tendency is to give preference in this work to the needs of children apparently because it is believed that on the whole a larger return for the investment of time and effort can be got from the early age groups. Social agen-

cies and children's courts appear to be asking and receiving broader service. There is increasing penetration of psychiatric methods into educational activities in the community.

12. Quite recently there developed in Chicago an interesting effort to mobilize the recovered patients of the Psychiatric Institute to create a public attitude more matter-of-fact towards the problem of mental illness. Removing the stigma of hospitalization was taken as the object of this work. No little attention has been attracted, and there are other regions where similar efforts are under way.

13. A vast amount of new building has been done. Credit must be given to the federal government for insisting on good standards of construction wherever governmental funds are spent. Many of the new buildings are well planned. Not all had criticism from men with special knowledge in this field, and therefore some new buildings are not very well designed to accommodate the special groups for whom they were built.

Former practices regarding the height of buildings seem to be carried out in the new ones and in some places one finds three- and even four-story structures. On the other hand, the bungalow is being more widely used. In California it is especially practical because of earthquakes. Illinois, which does not suffer from ground tremors, has used many in its newest institution at Manteno. One may venture to hope that more one-story structures will be built, particularly in these days when the number of old and feeble patients is increasing. Such persons should be kept near the ground, and bungalow construction is very suitable for them.

The movement for separate reception buildings is still in progress. There is an unfortunate tendency in many places to combine accommodations for the new patient with accommodations for the physically ill—an arrangement that is good for neither group. Many beautiful special buildings for the physically ill have been added to mental hospital plants.

The tendency in dining rooms is to have fewer and to equip them for cafeteria service. This involves the construction of corridors either out of doors or underground to make passage to and from the dining room safe and simple at all hours of the day.

14. The economic depression brought some political raids in places where relative stability had existed. It is to be expected that local leaders interested in obtaining work for their constituents who

have supported the successful party faithfully and who need help in supporting their own families should look upon work in a public institution as something to be parcelled out on political grounds. It is believed that this trend has not gone very far. The greatest barrier is a public opinion that recognizes the impossibility of mixing medicine and politics without damaging the welfare of the patients. Another protection in some communities is a standard of relief sufficiently liberal that the ordinary jobs in a mental hospital are not attractive.

15. There has been some movement towards better standards of administrative control. In one state this step was taken so late in the term of an expiring administration that the political overturn of November has destroyed the better organization. Some of the boards of control have been broken up. Their interests were too varied, and the mentally sick have seldom received first consideration from them. Where there are enough hospitals and schools to justify the existence of a separate board, better results are likely. Professional direction of the mental hospitals has received attention in Kentucky, Michigan, Virginia and Wisconsin, and the central board or department has been implemented with an official who could give real psychiatric leadership.

This has been for the mental hospitals a very trying period as regards the necessity for stretching old appropriations over enlarged numbers of admissions. Depressions are advantageous in at least one regard; when many persons are out of work it is possible to build up a stronger attendant organization. Few states have a definite policy about the size of institutions and the adequacy of personnel. Even the location of hospitals has not seldom depended on other than medical reasons and in some states a trip of 500 miles is required to reach a hospital and at least 1000 miles of travel to visit a patient and return home. Very encouraging features are the earnestness and ambition of the medical staffs and an apparently increasing popular demand that competent administrators shall be supported in their policies by their state governments.

DISCUSSION.

DR. ROSS MCC. CHAPMAN (Towson, Md.).—Dr. Overholser makes a strong case for central administrative control. I agree with him that the central authority should be a psychiatrist; and that the leadership should be essentially medical. This leadership obviously should have the respect and confidence of the State Medical Society which in time of political danger may well be a tower of strength. It is regrettable that politics must creep into this discussion of administration, and I have no doubt Dr. Overholser will agree that there is danger in too powerful central authority. A middle road should be followed, developing team work but giving plenty of authority to the superintendents of hospitals.

There is a great advantage in having a central authority to speak for the various hospitals in dealing with legislative committees and with other state departments, particularly the Department of Education.

As for the political dangers, the only way in which they may be slowly but surely eliminated is through the patient, constant education of the public to the fact that mental disease is a public health matter, that the state hospitals with their intramural as well as community activities are deeply concerned with the public health.

In Dr. Ebaugh's excellent presentation I was taken back at first with the impression that there was too much talk of apathy, lack of sympathy on the part of the state hospitals; but that impression was corrected in the last part of the paper most emphatically by the response made by a large number of hospitals to his proposition as to the inauguration of institutes. I am in complete sympathy with that idea, as I believe we all are.

To carry out their expanding programs the state hospitals will need money, and they will get it in proportion as the public realizes that they are dealing with the most important of all public health problems and gives them adequate support. It is in the state hospital that teaching and research must finally be largely done.

It is not a waste of time for all of us to talk to church organizations, civic organizations, parent-teacher organizations, about the facts of personality development, mental health and the prevention of illness, in an effort to promote a wholesome attitude on the part of the public which is so essential.

As to Dr. Hamilton's paper, his work is not completed; it will go on and the effect of his and Dr. Kempf's surveys, the facts they have assembled, the basis they have laid for sound teaching and research in the state hospitals of this country, will endure after all of us have moved on. I feel that when one looks back over the past twenty years, taking note of what has been accomplished and what efforts are being made today, we shall find reason for encouragement and hopefulness.

DR. NEIL A. DAYTON (Boston, Mass.).—Dr. Overholser mentioned our centralized statistical system in Massachusetts. This has been in operation

since 1928 and has functioned fairly efficiently. The superintendents may object to centralization of certain items but there is one thing that they are glad to pass to a central department, namely, the compilation of statistics. This results in greater uniformity of findings. The various hospitals are naturally prone to make their own interpretations; unless you establish definite criteria, your figures will not be of much use, and a central department can set up the criteria for the various institutions and see that they are observed.

We prepare the statistics for the 16 state hospitals and schools in Massachusetts on the 18 standard tables of The American Psychiatric Association and pass them back to the institutions. The superintendents discuss them and then include them in their own reports.

Another advantage that has accrued from this system has been the monthly reports on discharges, deaths and patients placed out on visits. There is a report made available for our commissioner which shows what each hospital has done in the preceding month with discharges. A separate table is offered showing the number of deaths and a third showing the number of patients placed on visit. It is useful to the superintendents, too, because they can take these reports to their staff meeting and say, "Why are we ninth or tenth in discharges this month? Why is such-and-such hospital first? Why did this hospital get out 150 cases on visit last month, while we only got out 50?" It gives the superintendent a means of talking to his staff about the machinery of his own organization.

Dr. Ebaugh spoke of the desirability of organized research. The more I look at investigative work, the less interested I become in research in the abstract, the more I become interested in persons who do research. The building up of large organizations for research has to be carefully supervised. It may get to the point that the research organization feels that they have to keep to schedule, with so many papers a year. The difficulties in this respect are obvious. Every hospital has a man who is interested in research, and interested in writing. I believe in building up these individuals, including the young man who has not yet made a start. If you take a group of 15 or 20 hospitals and have one such man in each institution, in five or ten years you will have 15 or 20 men who will be in a position to do something, rather than place 20 men in one large organization.

Dr. Hamilton tells us in his first summary that the demands for hospitalization have been increasing for many years and the end is not in sight. Several reasons are obvious. First, the average duration of human life has been materially increased and much larger numbers of persons live to the period when they are subject to senile sclerotic conditions and psychoses. This is more apparent than real however. Checking up in Massachusetts, we find in 1920, 18 per cent of the population were aged 50 years or over; in 1930, 20 per cent were 50 years or over. That isn't a great increase. The expectation of life has increased markedly over the past 30 years, but this increase has taken place through the saving of lives in infancy and the first two or three decades of life.

When we consider the situation of a person aged fifty, we find that he has not improved his condition a great deal; there is only a slight increase in expectation of life.

The actual situation is that mental disorders are increasing in persons over fifty years of age far in excess of any increase in the population, and we can only say that the economic and social conditions that have prevailed in this country in the past twenty years have had a particular selective effect on persons over fifty years of age, both physically and mentally.

DR. ROY D. HALLORAN (Waltham, Mass.).—I consider it a distinct privilege to have this opportunity to discuss Dr. Ebaugh's comprehensive paper. It is my purpose to offer the substance of a paper prepared by Dr. Paul I. Yakovlev and the reader, covering our experiences of three years with post-graduate seminars for physicians in Massachusetts. Time permits here only a general summary but it is hoped that a later opportunity may provide a full description of this project.

Examinations by the American Board of Psychiatry and Neurology not only pointed out the immediate need for practical post-graduate review courses in this field but have indirectly placed in our hands a valuable and timely implement for raising the standards of specialized practice in both hospital and community.

A state hospital forming a link in a large system of such hospitals in close proximity to a large academic medical center is the ideal place for building the post-graduate training of the entire system. Here both instructors and material are available. The first step in the realization of such a plan is the organization of yearly seminars in neuropsychiatry.

Several attempts in various places throughout the country were made in this direction during the last year. We felt, therefore, that a brief account of our experiences with the organization, management and development of seminars in Massachusetts might interest the section on psychiatric education.

The program developed from what originally was intended as a "refresher" course in those subjects required by the American Board of Psychiatry and Neurology which are difficult to prepare without assistance. The subjects were neuroanatomy, neuropathology and neuroroentgenology. However, the growing interest in and popularity of the courses from year to year clearly indicated the need of a broader training program and accordingly the number of lectures and of subjects taught each year were increased. Thus, the first seminar held in the fall of 1936 was attended by nine physicians and the program consisted of ten two-hour lectures. The second seminar was attended by twenty-five physicians; the number of lectures was increased to fourteen. The third seminar held in the fall of 1937 was attended by thirty-six physicians. The program included neuroanatomy, neurophysiology, neuropathology, roentgenology, clinical neurology and clinical psychiatry, and consisted of a total of forty-seven lectures given one day a week through eleven weeks. The fourth seminar, held in the fall of 1938, was attended by forty-eight physicians. Only twenty-one of these were primarily interested in preparation for the Board examinations. Several physicians in the class came from other New England states. The program was enlarged to sixty two-hour lectures, distributed through twelve weeks, Thursday evenings and all day Fridays each week.

Many of those who attended stated that the seminars helped them to find new interest in their routine hospital work. Several superintendents commented favorably on the gratifying influence on staff morale.

We propose in the future to divide the seminar into two semesters—one neurological in the fall and the other psychiatric in the spring, with two months interval between. Such an arrangement would permit still further development of subjects requiring laboratory and demonstration facilities and a really substantial course of some thirty-six to forty-eight lectures in psychiatry. This is our immediate aim. It is evident that in teaching neuropsychiatry, it should be envisaged primarily as a branch of practical medicine. There should be room in the program of seminars for various topics of internal medicine, general pathology, pediatrics and hospital administration of practical value to a state hospital physician. Such an ambitious program exceeds, of course, the scope of a mere seminar and is that of a post-graduate training school.

The value and, in a sense, the originality of the seminars consisted in the fact that they are planned as a unit. There are ample opportunities to take advanced courses in any single subject of psychiatry, neurology and other medical specialties. Such courses are offered by many hospitals, institutes and professional organizations. However, there is decidedly a need for courses in basic disciplines, on which the every day practice of psychiatry and neurology are founded. Indeed all of us, even the most competent, need from time to time to go over the abc's of medicine, not necessarily because one needs to learn them but because one needs to revalue them more critically in the light of a longer personal experience.

These seminars would not have been possible without the generous collaboration of several leaders in our special field in Boston and the wholehearted cooperation of the State Department of Mental Health. We wish to acknowledge the contributions of Drs. A. Leo Alexander, C. Macfie Campbell, Myrtelle M. Canavan, Stanley Cobb, Malcolm J. Farrell, William G. Lennox, H. Houston Merritt, Tracy J. Putnam, David Rothschild, Harry C. Solomon, Theodore J. C. von Storch, Richard C. Wadsworth and Walter Wegner.

These seminars have been an experimental group project, utilizing the Metropolitan Hospital as a center, without charge to applicants, the hospital absorbing minor costs involved. Where the material support should come from in the future is a question that requires considerable study.

In summary, we believe that our experiences indicate a need not only for review courses for examination purposes, but also point the way for raising the professional standards of those upon whom the care and treatment of mentally ill most immediately depends.

DR. C. M. HINCKS (Toronto, Canada).—I would like to touch briefly on three points that seem to me to be of particular importance in connection with state hospital programs; three points that have already been discussed by candidates in this symposium.

The first relates to reception services, to diagnosis and treatment arrangements for the first year of hospitalization. We all realize that, while recovery from mental illness may take place two, three or more years after the admission of the patient to the hospital, nevertheless, on the basis of averages we can expect higher recovery and improvement rates when illness is in its incipiency, within a year of its onset. This being the case, it seems to me that we are justified in focussing attention on the first year of hospitalization. During this year, there should be no such thing as overcrowding, no herding together of incompatible types, no pious gestures in the direction of occupational therapy, but carefully conceived programs. There should be no psychological abuses—abuses that are bound to occur when patients are treated *en masse* with a minimum of individual attention.

Dr. Hamilton and Dr. Kempf found in their surveys that very few hospitals in America measure up to the minimum standards advocated by the Association and, while we would like to have all our institutions living up to the minimum standards or requirements that we consider desirable, nevertheless, we are forced to be realists—we are forced to make progress by stages, if we cannot initiate comprehensive programs all at once.

And the logical first stage would seem to be the improvement of reception services. To abet progress in this direction, it would seem to be a sound move to rate the mental hospitals in America with reference to reception services, and to accredit only those institutions with adequate arrangements for scientific, humane and understanding treatment of patients during the first year of treatment and hospitalization. Ultimately, the rating of hospitals should extend of course to all the services of mental institutions.

A second point that strikes me of extraordinary significance relates to parole and rehabilitation. I expect that readmission rates could be considerably reduced through a larger measure of attention to the whole question of parole. Our colleagues in the field of tuberculosis, as we know, are tremendously concerned with the fitting of patients for life in the community, patients who have received treatment in sanatoria. In some of the more advanced hospitals for the tuberculous, programs have been developed for vocational guidance, vocational training and, finally, vocational placement, with consideration to the home situation. With necessary modification, we should be engaged in the same type of work in the mental hospital field. The days of perfunctory discharge and parole are over.

The third and last point relates to research and scientific work. Unless each medical member of a mental hospital staff is imbued with a keen scientific and clinical spirit there is danger of a sterile routine creeping into the diagnostic and therapeutic procedures. The antidote lies in the conduct of research and original endeavor in all our hospitals. We are concerned here with a larger issue than that of adding to the sum total of psychiatric knowledge. We are concerned with the scientific life of every last hospital psychiatrist and, even if the scientific returns are modest in terms of additions to knowledge, much will be gained through development of hospital staffs who will make our institutions *hospitals* in deed as well as in name.

DR. NOLAN D. C. LEWIS (New York, N. Y.).—I think there is little in these excellent papers with which to disagree and some points would well bear elaboration. I agree with the comments of Dr. Overholser regarding central control. I believe it is the right principle because it follows the main lines of biological evolution.

From the first in evolution, there has been an increasing tendency to centralize. We may think that in providing us with only one pair of kidneys instead of segmental ones which certain forbears had, nature made a mistake in so much centralization, particularly if you get nephritis. The same might be said about other organs that have dropped out and become centralized in one set of organs. However, you would be in a worse case if you had to take care of a great number of segmental organs of that character.

I think that the same principles apply in our social and human biological developments in the centralization of function. But we must not over-centralize, I mean not overstructuralize, because whenever nature over-structuralizes, then there is difficulty and death of the whole species or a part.

The second point is that success of a central control of hospital systems depends almost entirely upon the personality of the individual who is in the saddle. An absolute monarchy isn't a bad type of government. If you have a good king who lives long enough it will be successful. If physicians, as Dr. Overholser indicated, are in the hands of spoilsmen, then your central system fails.

Regarding research, there are people who do research in spite of anything. They have energy, curiosity, and in addition, they have a special type of intelligence which is able to deal with what we call analysis and synthesis and they have, according to the old Chinese proverb, three pairs of eyes—one set of eyes by which they look back into the past and evaluate what has been going on; another set of eyes that takes into account the immediate surroundings and activities, and the third set of eyes that gazes into the future and has research vision. Those individuals will do research though they starve. A man whose name you have seen on many research projects told me the other day that he didn't know what he should do because a hot dog stand moved away and he had no place to get his meals. These men work without money. They work without any stimulation. You don't have to educate them; they educate themselves first. There are a few of those geniuses, men of great talent.

There is another individual with curiosity and with great energy who is doing research whom you can't stop, and he shouldn't be doing it because he doesn't have research intelligence. He suffers from what has been called energetic ignorance, a most terrible thing to deal with when it gets going.

Between those two falls a group to which Dr. Ebaugh's remarks apply very strictly and distinctly. Probably every individual who is interested enough to enter psychiatry or state hospital service, has somewhere in him a streak of talent, sometimes narrow, sometimes fairly wide which may require stimulation in an organized way. I always tell these young men there is a way to test whether they have research ability or not. There is not

one single aspect of practical psychiatry or laboratory investigation which is as yet exhausted. Always there are matters that will bear more observation. If the young state hospital man doing his routine every day would make just one more examination, something that isn't routine, something that would throw light on some type of case, some form of delusion or hallucinatory experience or the condition that we have been calling deterioration and emotional deviation, or examine some cases that fall outside of the usual categories and would accumulate those observations and study them, he would soon find out whether he was equipped to do research or not.

In a scheme such as Dr. Ebaugh has advocated, these men would begin to see the problems or problems could be pointed out to them, and by concerted organized effort there would eventually accumulate something of significance in the research program.

Dr. Hugo Mella (Washington, D. C.).—With reference to central control, it is a foregone conclusion that in any federal activity this is elemental; but central control can function as a stimulus to progress only when the control body acts as an integrator, bearing in mind constantly that service to the patient must always be the paramount factor in all policy determinations.

The Veterans' Administration is at present operating 84 hospitals throughout the United States, 27 of which have been constructed primarily for the care of psychiatric cases, and these latter are so constructed, equipped and staffed that they are prepared to care for any emergency, medical or surgical condition, that might arise in a veteran in the geographical area covered. It is believed that the requirement to be prepared to care for medical and surgical emergencies reacts favorably upon medical staffs by compelling close association with physicians practicing branches of medicine other than psychiatry and thereby also keeping the care of the physical condition of the psychotic patient at a high level. In an organization of the scope and widely distributed character here outlined it is obvious that there must be a central control agency to coordinate policy and procedure.

There are at present available for the care of neuropsychiatric cases a total of 31,214 beds in Veterans' Administration facilities. There are 1,575 full-time medical officers on duty in the Veterans' Administration, 440 of these giving their full time to the care of neuropsychiatric cases. Within the next fiscal year there will be required a minimum of 300 additional medical officers. They will be drawn from United States Civil Service registers upon which the doctors will be certified in the Veterans' Administration.

This group of physicians will be brought into the service as associates, will be given from two to three months' didactic instruction in Veterans' Administration hospital procedure and a brief medical review, this period to be followed throughout the remainder of their first year by continued clinical instruction with practical training on wards under the supervision of senior medical officers. If found acceptable at the end of this training year, they may become permanent members of the medical service.

Particularly in the field of neuropsychiatry it has been found necessary to commence instruction with the very fundamentals of the specialty, to the

point of giving instruction in the selection of medical literature and the use of the Quarterly Cumulative Index Medicus. In other words, only a small percentage of recent graduates are found to be adequately equipped in the fundamentals of neuropsychiatry to permit them to step immediately into advanced work, although every one is a graduate of a recognized class A school. Therefore, it has gradually become one of the important functions of our neuropsychiatric hospitals to take part in the training of physicians.

In addition to the teaching, every effort is made to encourage research, bearing in mind that research in pure science can best be conducted in special units established for the purpose directing the efforts of the clinicians interested in research more into the channels of applied science.

DR. L. M. ROGERS (Lexington, Ky.).—This symposium points out the lack of uniformity which exists in state mental hospital programs.

The advantages and disadvantages of the different patterns of state hospital service have been discussed from several points of view.

From a public health point of view, the mental hospital program cannot be evaluated as a single item. Its relation to a centralized organization for mental health and the relationship of that organization to a general program for the prevention and treatment of disease must also be considered.

A broad health program must include facilities for both the prevention and treatment of disease. It must also distinguish between prevention and treatment and between individual and group prevention in order to determine the basic rôles and operational limits of the organizations which make up such a program.

Fundamentally treatment is a process which is applicable to the abnormal. In mental disorder it consists of two essentially different but related procedures. First, there are measures which must be applied directly to the individual for the purpose of bringing about desirable changes in him as a person. Second, there are measures which must be applied to the environment in which he functions in order to influence adverse social and material factors which interfere with his adjustment.

Fundamentally the prevention of disease is a process which is applicable to the normal. Like treatment its measures deal with individual and environmental factors but true preventive measures differ from those which are required in treatment.

The mass or group prevention of disease must also be distinguished from individual prevention. Mass prevention is concerned with the application of general measures to groups of normal individuals and to environmental conditions which are potential causes of disease.

Although there is considerable overlapping in the practical application of measures for the prevention and treatment of disease, the fundamental differences between them is sufficient for a determination of the primary rôles of the organizations which constitute a health organization. Therefore, it is reasonable to assign rôles to them.

On a basis of primary purpose, a centralized mental health organization would have a dual rôle in a general health program. It should establish

facilities for both prevention and treatment. A hospital service within such an organization, however, would be limited to a single rôle—the treatment of mental disorder.

From this assignment of a primary rôle it does not necessarily follow that a hospital service should be confined within its walls and that it should perform no preventive service. The fundamental nature of treatment, which requires the application of measures to both individual and environmental factors, demands that certain hospital facilities be extended into the community. Through this extension, the early recognition and treatment of individuals with slight mental abnormalities or abnormal trends would also become a hospital function. Although primarily a treatment technique, this process also has a preventive value to the individual served.

A fundamental distinction between prevention and treatment and the assignment of a primary rôle to hospital services would extend rather than limit hospital activities. Furthermore, such an assignment has the additional advantage of definitely placing the responsibility for the improvement of remedial methods. Moreover, by relieving mental hygiene and public health departments of the burden of remedial duties, it makes it possible for these organizations to develop true preventive techniques.

It seems, therefore, that a public health point of view offers a definite and logical basis not only for the development of a uniform mental hospital program but also for a centralized mental health organization.

DR. M. A. TARUMIANZ (Farnhurst, Del.).—The question of central administrative control of state mental hospitals has not yet been adequately solved. It stands to reason that any state setup which is apt to lead to a complete or partial change of personnel at every change in political control is seriously detrimental to the organization. Well qualified superintendents are inclined to protest against central control unless they are assured that their institutions will not be injured by such a control. There is no well trained psychiatrist anxious to have his hospital under the control of a lay person who does not thoroughly understand the difference between an institution for the care of the ill and one which cares for the delinquents, orphans or other state dependents. However, in states in which there are several institutions to care for the psychotics, it is essential that the same care be given to the patients in all institutions. A central organization might seem to be essential to serve this purpose.

Uniformity of record keeping throughout all institutions, depending on the nature of the particular institutions would be of great help. By this I mean that the same type of record keeping should be used not only in state hospitals but in other state institutions. The importance of this procedure as an aid to research and diagnoses is probably brought out more clearly in Delaware than in any other state in the Union since we can determine the percentage of patients who have spent time in other state institutions. It would be of great value had we detailed information concerning the behavior of the patient during his residence at the other institution.

In a small state having only one institution of each kind, it would seem sufficient to have a committee, made up of various superintendents and heads

of organizations, to act as a central agency. The chairman might be an individual who is not connected with any institution.

Dr. Overholser states that any hospital would welcome the opportunity to divide the responsibility of releasing patients about whom there is some question as to whether they are dangerous. Personally, I do not feel that dividing the responsibility releases the hospital from its moral obligations should a tragedy occur. It would seem to me that the officer who is with the patients constantly is the one responsible for the release of such an individual and that a central board unfamiliar with the patient's daily behavior should not share the burden.

The supervision and licensing of the private hospitals is an important matter. It is all too true that an inadequately prepared physician often opens a hospital for the care of mentally ill, charging exorbitant sums yet not adequately equipping his institution or giving his patients proper care. Some private institutions, indeed, are excellent but there are others which are merely places of detention. Such treatment should be considered mal-practice just as obvious neglect of the physical status of a patient by a general practitioner is considered mal-practice. However, inspection of private institutions by a central agency would not seem to solve the problem unless a legislature would pass a law preventing the continuation of institutions which are not approved by the agency.

I agree thoroughly that the extramural work should be entirely in connection with the state hospital. The clinic of the Delaware State Hospital not only handles adults and children, psychotics and pre-psychotics and behavior difficulties but also does work in the school in cooperation with the Department of Mental Hygiene of the public school system.

One wonders whether the plan of centralized administration is a result of present trends toward regimentation of all types of activities and may not be considered another step in the direction of state medical control. One wonders if it is possible to find enough men medically, psychiatrically trained with administrative ability, unprejudiced and non-partisan to head such a central agency in each state. I also question whether a central organization would solve the problem any better than a close and friendly coordination between the superintendents of the various state hospitals in the state. I do not know why so many state institutions are opposed to central control, but in my opinion it is simply a step further in making puppets of trained, scientific men and placing them in an army-like formation. I am just wondering if we could not run our state institutions as general hospitals with broad policies adopted by The American Psychiatric Association and rating them as the American Medical Association does. This would give each superintendent a goal to strive for, a necessary stimulus to bring forth the best that is in him. The efficiency of the individual institution under a non-central control is dependent upon those who are appointed on the board who hires the superintendent. It would seem that The American Psychiatric Association could set up standards of salaries as well as hospital administration which would encourage the individual institutions to strive for a higher rating. These ratings would be recognized by the individual legislatures as a standard

that they would demand of their institutions. Publicity would lead the public to demand the best.

I feel that Dr. Overholser has prepared a very fine paper which shows careful study of situations as they exist, and demonstrates the necessity of considering administrative details as well as scientific ones. I fully agree that The American Psychiatric Association has paid too little attention in the past decade or so to the important question of administration.

I cannot entirely agree with Dr. Ebaugh that most of the physicians in the state hospitals are not interested in the cause and cure of mental disease. This has not been my personal experience. The ward physician, however, has his time so occupied that he has very little for other work, and I do not approve of physicians using their time off duty for psychiatric work.

Only too frequently state hospitals are removed from medical centers and it is necessary for the staff to devise their own program of instruction under the clinical director. Although the younger physician might profit by a two months course in special training, such a procedure in an already understaffed hospital is detrimental to the patients. Until the situation of understaffing in state hospitals is corrected, I see no solution to the training of resident physicians in the outlying institutions.

I agree with Dr. Ebaugh, that to carry on intramural training requires a clinical director who is enthusiastic and who has a personality that can inoculate the staff with his own enthusiasm. We have found in Delaware that our residents are inclined to remain at the hospital after their year's residence is completed. We are probably more fortunate than some other state institutions in being near Philadelphia, New York and Baltimore—all medical centers affording easy access to special courses, libraries and the like. I still feel that if The American Psychiatric Association would demand that state hospitals live up to the Association's requirements, the personnel would improve and interest in research would increase. Dr. Ebaugh has done a good job in training psychiatrists, but unfortunately he has not trained enough men and women to fill the demand.

The survey reported by Dr. Hamilton and Dr. Kempf helps us strive for and maintain better standards. All superintendents have felt the difficulties described and all are feeling somewhat baffled in attempting improvements during the present economic situation throughout the entire country. The Delaware State Hospital as well probably as all other institutions is suffering from an increase of admissions due to cases of senility and the economic impossibility of families keeping mildly ill patients within the home. Relief agencies are also anxious to have all cases committed no matter how mild when the family seeks their relief. Whether it would be practicable to give additional relief to such families is questionable since there is a danger that relief agencies will tend to throw the burden of support of the entire family on the state institution. Undoubtedly all institutions have senile cases who could make a satisfactory adjustment in the home if this were economically feasible.

Although state hospitals may refuse to admit patients until there is a vacancy this refusal is necessarily limited to those cases who are not a menace

in the community. Placing patients in workhouses and jails seems to me a step backward and more detrimental than overcrowding our state hospitals. I would also like to state here that it is still difficult to obtain properly trained assistants to fill staff vacancies, since state institutions must be maintained on a budget, often insufficient. The salary scale of the institutional physician is not high enough to attract the better class of medical student, unless he is fundamentally interested in institutional psychiatry. Institutional work is still used to some extent as a means to obtain finances until the young physician is able to either start his own practice or take post-graduate work. With the more rigid standards which are now being required by The American Psychiatric Association, this problem will be solved.

The administrative control in Delaware has presented no difficulty since there is only one hospital and the Board of Trustees is the controlling organization, under the supervision to a slight extent of the State Board of Charities which does not interfere in any way with the professional work of the hospital; but which has the power to inspect the institution to determine whether the physical plant is properly cared for and to see whether the material comforts of the patients are neglected. This inspection has not been carried out in recent years, since, due to the smallness of the state, both in area and population, the administration of the hospital is well known to the majority of the people of the state.

Despite the depression we have been unable to build up a strong and efficient attendant organization. The legal residence requirement may be a contributory factor. All employees are fingerprinted, in cooperation with the Federal government, and are given psychological and physical examinations. This does not mean that we hire no one who has had any difficulty with the law since it is a definite part of mental hygiene work to help individuals rehabilitate themselves; but it does give adequate information in certain cases and allows the institutional head to give added training, when such is needed, as well as additional encouragement. On the other hand, no matter how we feel about the rehabilitation of criminals, the repeating offender of years duration is not the type of individual who should work with the mentally ill. This is a new procedure and policy of the hospital, whether wise or not cannot yet be judged.

In closing, I would like to say that a repeated survey of the hospitals by The American Psychiatric Association, as carried on by the American Medical Association and the American College of Surgeons would be a great help in keeping up the psychiatric standards of the individual institution. Institutions recognized by the two other organizations are surveyed yearly as to their qualifications for classification. We feel that The American Psychiatric Association should adopt the same standard and rate institutions from a psychiatric viewpoint at regular intervals.

The paper by Dr. Hamilton and Dr. Kempf shows careful study and analysis. It should encourage all institutions to strive to meet the qualifications of The American Psychiatric Association. Let us hope that means will be found to enable Dr. Hamilton to carry on this work.

THE SIGNIFICANCE OF SPECIAL MENTAL TESTS FOR DIAGNOSIS AND PROGNOSIS IN SCHIZOPHRENIA.*

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The treatment of schizophrenics with insulin and other similar somatic methods is apt to push into the background all interest in the psychological investigation of schizophrenia. There are several reasons, however, which should induce us not to neglect the psychological investigation. Although these treatments have yielded considerable results, we cannot overlook the fact that we know very little as to whether or not these results will be permanent. Furthermore, we do not know whether or not psychotherapy should play an important rôle alongside somatic treatment. There is another reason why psychological investigation demands special consideration. The somatic treatment certainly does not help in all cases, and we cannot predict which cases will or will not be helped. The facts show that the duration of the disease is not a reliable indicator as to the chances of improvement by that treatment. One is often inclined to take duration as such an indicator; however, the clinical data cannot support such an assumption. There are cases where spontaneous recoveries occur even after an illness of long standing, while in other cases the typical schizophrenic end-state might develop after a short acute attack. Perhaps one day we shall find somatic signs which will enable us to decide which cases have a bad or a good prognosis; chemical research particularly may become instrumental for this purpose. In any event, psychological investigations can be of assistance. Examinations with specific tests have taught us that there is a definite mental change characteristic of schizophrenic patients. However, not all patients with the clinical diagnosis of schizophrenia show this change. Thus far we are not able to differentiate clinically those cases which show this change and those which do not. The findings suggest that by means of psychological investigation types of the disease can be distinguished

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which do not seem to correspond to the usual clinical classification. There is some hope with the help of this demonstrable mental change, to differentiate groups with different prognoses.

The psychological investigations can aid further in determining whether the recovery after treatment consists merely of a clinical, symptomatological or social improvement, or whether it is a real rehabilitation. Such a clarification would be of great importance not only in cases which have been treated, but also in cases of so-called spontaneous recovery. Finally, psychological studies have a special bearing on the question as to whether and in what way psychotherapy should follow somatic therapy. Explanations given later (see p. 584) will serve to demonstrate that psychotherapy deserves special attention.

Psychological explorations of schizophrenics have been carried out in a great number of studies. (The book of Dr. Nolan Lewis gives a good review of them.) I should like to refer only to some earlier publications by Bleuler,[5] Storch,[20] Meyer-Gross,[16] and others, further to some experimental studies, especially the more recent ones from the psychological department of Worcester State Hospital.[14, 18, 19]

Most of the earlier publications present chiefly theoretical discussions based upon an analysis of clinically outstanding symptoms like changes of thinking, language, etc. As far as they involve experimental work, the research is focused upon single psychological capacities by studying separately reaction time, memory, attention, etc. Both procedures are not very fruitful for the problems we are interested in. The material used in the first procedure is too complex to permit its analysis to evolve much more than somewhat speculative general ideas about the mental change in schizophrenia. In the second procedure the testing of single capacities, no matter how minutely examined, yields more or less piece-meal material of rather peripheral significance. The same holds for the investigation of the scatter in the Binet-Simon test. To know the change of reaction time, attention span or retention, etc., in itself does not help us to gain an understanding of the essential personality change in the patient.

The type of approach which I wish to emphasize differs from most of these studies. It places the changes of the *total personality* in the foreground and tries to attack them *experimentally*. This

approach goes back to research on patients with organic brain lesions by my co-worker A. Gelb and myself.[8] Also in the field of organic lesions, separate symptoms have usually been singled out for study. Contrary to this procedure we tried to study the behavior of the patient in its entirety and to determine the changes of the total personality. From these investigations we arrived at a distinction between two kinds of human behavior which we called the abstract and the concrete.

These attitudes are neither acquired mental sets or habits of an individual; nor do they represent specific isolable aptitudes such as memory or attention. They are rather capacity levels of the total personality, each furnishing the basis for all performances on a certain plane of reference to the outerworld situation.

The concrete attitude is realistic. In this attitude we are given over and bound to the immediate experience of the given thing or situation in its particular uniqueness. Our thinking and acting are directed by the immediate claims which one particular aspect of the objects or situation in the environment makes.

In the abstract attitude we transgress the immediately given specific aspect or sense impression, we "abstract" from particular properties. We are rather oriented in our action by a more conceptual viewpoint, be it a category, a class, a general meaning under which the particular object before us falls. We detach ourselves from the given impression and the individual thing represents to us an accidental example and representative of a category. Therefore, we also call this attitude the categorical or conceptual attitude. The abstract attitude is basic for the following potentialities:

1. To assume a mental set voluntarily.
2. To shift voluntarily from one aspect of the situation to another.
3. To keep in mind simultaneously, various aspects.
4. To grasp the essential of a given whole; to break up a given whole into parts and to isolate them voluntarily.
5. To abstract common properties; to plan ahead ideationally; to assume an attitude toward the "mere possible," and to think or perform symbolically.
6. To detach our ego from the outerworld.

The abstract behavior is the more active one, whereas the concrete is the more passive one. For the concrete behavior the above mentioned potentialities are not prerequisite.

We have to differentiate various degrees of both, the abstract and the concrete behavior. First, there are various degrees of abstract behavior corresponding to the degree of complexity which the performance in question involves. For instance, the highest degree of abstract behavior is required for the conscious and volitional act of directing any performances whatsoever and for the act of accounting on these to oneself or to others. A less abstract degree is required for conceptual behavior if unaccompanied by the mentioned awareness of one's own doing. As a special instance of the latter behavior may be considered symbolic or metaphoric thinking and intelligent behavior in everyday life.

The gradation applies just as well to the concrete behavior. The most concrete way of dealing with situations or things is to react to one aspect of them exclusively; for instance, reacting alone to one color or a particular form of the object, to one property of it, such as its practical use. A less concrete approach is indicated when the person is embracing in his scope the total concrete configuration of an object or situation and is not determined in his response by one impressive particularity of it alone.

The normal individual combines both attitudes and is able to shift voluntarily from one to the other at will, according to the demands of the situation. Some tasks can be performed only by virtue of the abstract attitude, for other tasks the concrete attitude is sufficient.

Organic pathology in patients with brain disease disintegrates human behavior in a way that the capacity for abstract behavior is impaired to a greater or less degree. Thus the patient becomes more or less reduced to a concrete level so that he can perform only such tasks as can be fulfilled in a concrete manner.

In order to avoid misunderstanding, we have to emphasize that the process of disintegration toward the concrete does not abolish the arousal of ideas and of thoughts at all. What the deterioration affects and modifies is the *way of manipulating and operating with ideas and thoughts*. Thoughts do, indeed, arise but can only become effective in a concrete way: just as the patient cannot deal with outerworld objects in a conceptual frame of reference, so he deals with ideas simply as things which belong to an object or situation. Concepts, meanings, categories other than situational means-end relations are not within his scope.

Hence, the characteristic change in patients with brain lesions could be determined as an impairment of the attitude toward the abstract; the various symptoms are now considered to be manifestations of that basic defect in various fields. From this basic personality change many of the otherwise separately described disturbances could be made understandable in a unitary way.

We came to our result especially through the use of various sorting tests (Zuordnungstests).[8, 9] Our general concept and methodological procedure have been adopted by the late Russian psychiatrist, Vigotsky [21] in his attempt to determine the basic intellectual change in schizophrenia. He, too, used for his studies a sorting test, a modification of a test devised by the psychologist N. Ach.[1] This test now named the Vigotsky test became known in this country through the publications of *Hanfmann and Kasanin*.[13, 15] Vigotsky believed the change he found in schizophrenics to be similar to the impairment of the attitude toward the abstract which we found in organic cases. Being inclined to relegate the change to an impairment of the thought process proper, he spoke of an impairment of "thinking in concepts." Kasanin and Hanfmann [15a] have confirmed Vigotsky's findings on a greater number of patients and have tried to place the Vigotsky test on a quantitative basis. Because, in my opinion, this personality change does not concern thinking alone, I prefer to speak of a change in the total behavior and to consider the impairment in thinking a special expression of the latter.

No matter how useful the Vigotsky test may be for the exploration of schizophrenics, I favor the tests which we introduced for examining organic cases and those which my co-workers and myself have subsequently developed and improved. The reason is that these tests seem to me much simpler than the Vigotsky test and therefore more easily applicable to patients. There is no time for describing and discussing these tests in detail here. They are partially described in earlier papers of Gelb and myself [8] and our students in Germany.[22] In this country, the tests were used in studies of my co-workers Bolles [2, 4] and Nadel [17] and myself.[3, 11a] Very soon a detailed description of all these tests will be published by Scheerer and myself.

The battery of tests contains the following: the so-called stick test, the color-sorting test, the color-form sorting test (Weigl), the object sorting test (Weigl), my block test with the Kohs block

material and others. Usually the Vigotsky test and the Rorschach test are also administered. The investigations upon which my results are based have been carried out with the kind permission of Dr. N. Lewis, mainly at the New York Psychiatric Institute in the Psychological Department of Dr. Landis. First I worked together with Dr. M. Bolles, later this work developed into a collective project with the participation of several students, headed by Dr. Piotrowsky and myself.

All these tests are in principle of the same character. They all represent similar procedures to discover personality changes. However, it would be fallacious to confine our exploration to the application of one test. We are using a battery of these tests with the same patient because at times one test may be more effective in revealing the change and other times another test may serve the purpose better; the reason for these different results will disclose itself when we know better all the implications of the individual tests and the structure of the personality change. Particularly with respect to the possibility of their quantitative scoring there is a great difference between the various tests. My block test lends itself especially well to quantitative studies. Therefore I like to deal here with this test somewhat more in detail.

In the test were used the Kohs block designs which the patient has to reproduce with four blocks. I modified the procedure as follows: first we present the original design. If the patient does not succeed, we present him consecutively with the following modifications: first, an enlarged design of equal size as the four blocks; second, the original design divided by lines which break up the figure into four parts according to the four blocks required; third, the enlarged design also divided by lines; and finally, a model built by the four blocks proper. *This modification originated from our experience that in impairment of abstract behavior the subject's difficulty is mainly due to the inability to abstract* (1) *from size and* (2) from the *given configuration, the pattern of the design.* This concrete dependence upon the size may be overcome by introducing the *enlarged* designs. The concrete dependence upon the given *design-pattern* may be overcome by presenting the designs *broken up into squares* corresponding to the individual blocks. This division can be carried out by inserted lines or by presenting the blocks proper. Now the patient may be unable to copy the original design

but be able to copy the enlarged design or the design with lines or only the block model. The impairment is verified by the fact that the patient cannot copy the originally presented design, even after he had succeeded with any one of these modifications. That shows that he cannot learn the abstract attitude. The modifications presented here may be evaluated as representing different levels or degrees of greater concreteness in order to prompt the patient's performance. The enlarged design with lines is still less concrete than the final presentation of the model by the blocks themselves. There is still one step beyond this, namely, to present the four blocks separately instead of together so that the pattern in itself is not presented and the reproduction is reduced to placing four single blocks side by side according to the four models.

Certain normals show also a preference for concrete procedure, some for abstract procedure, which may be characteristic of underlying personality traits. Notwithstanding such preference of abstract or concrete behavior in some normals there is one decisive difference between the normal and the patient; while the normal can be induced to proceed in the abstract way, the patient can never learn that.

The most significant result of the findings with these tests is that in a certain group of schizophrenics there is a characteristic impairment of the attitude toward the abstract; the patients behave much more concretely than do normals and do not learn to understand even after demonstration, that the abstract behavior is required. In principle, these results are in agreement with the findings of Kasanin and Hanfmann. They give rise to various problems:

1. *Is it possible to understand, from these findings, the symptoms which usually are considered characteristic for schizophrenia?*

For want of time I can only mention here this question, its discussion would demand detailed elaboration of observations.

2. I should like to confine myself to a second problem which concerns the *general concept of the change in schizophrenia.* This can be formulated as follows: Are we in the *mentioned impairment of the attitude toward the abstract dealing with the basic change in schizophrenia, or with a secondary phenomenon?*

Distinction between primary and secondary symptoms in schizophrenia has been the objective of many discussions. I mention par-

ticularly the reports of Bleuler [5] and Meyer-Gross [16] concerning this subject at the Psychiatric Meeting in Basel, 1929.

One has spoken of a fundamental, primary change and of secondary phenomena which are to be considered as psychological reactions to the primary personality change, *i. e.*, as expressions of the struggle on the part of the pathologically modified psyche with the demands of the environment. The primary change was considered either as an organic nucleus, as a consequence of which bodily and mental symptoms appear (so for example the catatonic phenomena or the disturbances of associations); or the primary change was conceived as being a mental one, *e. g.*, an insufficiency of mental activity, intrapsychic ataxia or split personality and so on.

There is no doubt that the clinical picture of schizophrenia contains symptoms which may best be understood as expressions of a disturbed function of some apparatus of the brain, particularly of the frontal lobes and the subcortical ganglia, and also symptoms which are better interpreted as psychological reactions. Whether one might consider these two types of symptoms as being primary or secondary changes, in any case, a twofold therapeutic procedure is imperative. We have to attack the disease underlying schizophrenic symptoms by physical and psychological ways. Which way we should go is not simply decided from theoretical consideration. As far as somatic therapy is concerned, it certainly can be applied in any state of the patient, if his physical condition permits. It is to a high degree independent of the patient's mental state. This does not hold for psychotherapy. There is no sense in trying psychotherapy if the patient's mental and somatic condition renders it impossible for the examiner to come into a psychological contact with the patient. This lack of contact is not necessarily caused by a mental seclusion of the patient from outerworld influences in general, it can even be caused by the impairment of the patient's abstract attitude. In this latter case the patient might not be without any contact with the outerworld but he might be incapable of that specific contact which usual psychotherapy asks for. This procedure is successful only if the patient is able to grasp the explanations of the psychotherapist. But there arises a great difficulty. The therapist's explanations take place on a rational level which exceeds the patient's capacity. This is the case particularly because the impairment expresses itself in a special change of language.

One can imagine how difficult any communication must be with an individual whose words are so entirely concrete or so changed in meaning that they imply something totally different for both the patient and the physician. From these considerations it becomes comprehensible that the usual psychotherapeutic procedure is doomed to failure so long as this impairment of the abstract attitude persists. Dr. Fr. Reichmann's paper, presented at this meeting, supports my view. Her procedure aims at a more direct concrete communication with the patient based on empathy and cooperation with him, a communication which takes into consideration all the pathology the patient might disclose. The physician tries to adapt himself to the patient's capacities. Dr. Reichmann's success is a plain confirmation of the appropriateness of such an approach. In this connection the work of the Swiss psychiatrist Klaesi is worthy of mention.

For usual therapeutic procedures the impairment of abstract attitude is certainly a great hindrance, and if we wish to apply psychotherapy we must try to remove this impairment as much as possible. In this attempt a comparison of the personality change in schizophrenics with that found with the same tests in organic patients can guide us. There is no question that there is a great similarity in the personality changes in both conditions. This similarity does not permit the rash assumption that schizophrenia is fundamentally an organic disease; the great differences in the behavior of organic and schizophrenic patients in various directions will prevent such conclusion. However, the similarity points to an organic process as cause of this impairment, whether this process be primary or of secondary origin.

In any case our endeavor must be to *remove this change* in the brain's function, *and here the somatic therapy appears especially promising*. From the few experiences I have, I do not dare to say that this deterioration is really improved after treatment. Systematic psychological examination before and after treatment with the tests will be here of great advantage and is one of the tasks of further research.

If our first experiences with the tests should be confirmed in general, *i. e.*, if these tests can show that the patient has improved with respect to his deterioration, we may be able to use them also for prognostic purposes.

Determination of the quantitative degree of mental deterioration preceding the somatic treatment may furthermore enable us to differentiate the cases with probably better prognosis for improvement through treatment from those with worse prognosis. We may assume that the degree of mental deterioration goes somewhat parallel to the severity of the organic process and that an organic distortion of small degree might be removable but a distortion exceeding a certain degree cannot be rehabilitated. This assumption is confirmed by observation in so far as patients who show less deterioration in these tests seem to give a better prognosis for insulin treatment. These findings are published in a preliminary report by Bolles, Landis and Rosen,[4] they must, however, be evaluated with great caution. Not only, because the number of cases is too small to draw any definite conclusion, but also because the quantitative evaluation of the test results regarding the amount of deterioration has yet to be refined and the scoring basis gauged. It remains our task to organize these tests which are primarily suitable for qualitative analysis in a way that they can be used for quantitative scoring as well. The present findings can only offer hints in which direction we should proceed. Investigation of a very large number of patients is needed in order to allow generalizations.

We started out with the statement that the impairment of the attitude toward the abstract is a great obstacle to psychotherapy. If that is right, removal of this impairment will facilitate the application of psychotherapy. Indeed this state must oblige us to apply psychotherapy, because we have to assume that we can now help the patient along the same lines as his own endeavor. The patient, now freed from his deterioration, might try on his own accord to come to normal terms with the outerworld. He will try to overcome his conflicts. But he might not be able to do that successfully on his own and so be always in the danger of relapsing into the state of deterioration.

We have stressed that we do not know how the deterioration originates. If it is a secondary phenomenon, the state of greater concreteness may be considered to be a way out from the organism's unbearable conflicts, a protection against the danger of severe catastrophy. Many a symptom found in organic and so-called functional disease is understandable from this viewpoint.[12] Thus the patient whose conflicts are not relieved by somatic treatment

will easily fall back in this protective state of deterioration. This might be one cause of the great number of relapses after the treatment. Here psychotherapy can give the patient support and help him to get rid of his difficulties, which he could not achieve alone.

Exploration with the mentioned tests can furthermore be very useful for psychotherapy because it can provide a reasonable basis for the decision of what specific psychotherapy should be applied. It is self-evident that psychotherapy must be based on a knowledge of the particular mental change in the individual case. In this respect the application of these tests offers advantage because the special way in which the patient behaves during the tests and the comments he gives may disclose many peculiarities of his personality, of his ideas, of his emotional conflicts, which otherwise might not be disclosed.

From this analysis we see that psychological investigations, especially with those tests mentioned above, appear at least as a useful even necessary method of handling the problem of schizophrenia, also in the cases where somatic treatment is successful.

At present this research has not been developed to the degree which meets all the necessary standards. However, the results are promising enough that a continuation of investigation seems indispensable. The work which has to be done is so extensive that only by a joint effort of workers in many groups may we expect real success. It is especially the purpose of this paper to point to the necessity of that kind of research on a large scale. The tests will soon be available in published form so that these investigations can be carried on in different clinics on a common basis. Only a widespread project will make it possible to study a great enough number of patients, a prerequisite for reaching valid decisions on any of the questions discussed here.

The present seems particularly opportune for the execution of such a project. Research in schizophrenia, in general, has received a new impulse by the recent success of the somatic treatment. Especially chemical investigations and studies of other somatic processes are under way on a larger scale than ever before. One should not let the occasion go by for *comparative investigation of the same patients with methods of physical and psychological examination.* Only such a parallel study promises to open the way

to attain real knowledge of the essentials of this disease and to improve our therapeutic measures—and this is without any question our paramount concern.

BIBLIOGRAPHY.

1. Ach, N.: Ueber die Begriffsbildung. Bamberg: Buchner, 1921.
2. Bolles, M.: The basis of pertinence. Arch. Psychol., *212*: 52, 1937.
3. Bolles, M., and Goldstein, K.: A study of the impairment of "abstract behavior" in schizophrenic patients. Psychiat. Quart., *12*: 42, 1938.
4. Bolles, Rosen and Landis: Psychol. perform. tests as prognostic agents for the efficacy of insulin therap. in schizophrenia. Psychiatr. Quart., *12*: 733, 1938.
5. Bleuler, E.: Primaere and secundaere Symptome der Schizophrenie. Zeitschr. f. d. gesamte Neurolog. und Psychiat., 124: 607, 1930.
6. Cameron, Norman: Deterioration and regression in schizophrenic thinking. J. Abn. and Soc. Psychol., 34: 265-270, 1939.
7. Fischer, Franz: Ztschr. f. d. ges. Neurol. u. Psychiat., 124: 246, 1920.
8. Goldstein, K., and Gelb, A.: Ueber Farbennamenamnesie. Psychol. Forsch., 6: 127, 1924.
9. Goldstein, K.: The problem of the meaning of words based upon observations of aphasic patients. J. Psychol., *2*: 301, 1936.
10. Goldstein, K.: The modifications of behavior consequent to cerebral lesions. Psychiat. Quart., 10: 586, 1936.
11. Goldstein, K.: The significance of the frontal lobes for mental performances. J. Neurol. and Psychopath., 17: 27-40, 1936.

11a. Goldstein, K., and Katz, S.: The Psychopathology of Pick's Disease. Arch. Neur. Psychiat., 38: 473, 1937.

12. Goldstein, K.: The organism. A holistic approach to biology derived from pathological date in man. American Book Co., New York, 1939.
13. Hanfmann, E., and Kasanin, I.: A method for the study of concept formation. J. Psychol., 3: 521, 1936.
14. Huston, P. E., Shakow, D., and Riggs, L. A.: Study of motor. function in schizophr. II. Reaction time. J. General Psychol., 16: 39, 1937.
15. Kasanin, I., and Hanfmann, E.: An experimental study of concept formation in schizophrenia. I. Quantitative analysis of the results. Amer. J. Psychiat., 95: 35, 1938.

15a. Haufmann, E.: Analysis of Thinking Disorder in a Case of Schizophrenia. Arch. Neur. Psychiat., 41: 568, 1939.

16. Meyer-Gross, W.: Primaere und secundaere Phenomene in Schizophrenie. Z. f. d. ges. Neur. u. Psychiat., 124: 607, 1930.
17. Nadel, A.: A qualitative analysis of behavior following cerebral lesions. Arch. Psychol., No. 224, 1938.
18. Shakow, D., and Rosenzweig, S.: Play technique in schizophr. etc., I and II. J. Orthopsychiat., VII, No. 1, 1937.

19. Shakow, D., and Huston, P. E.: Stud. of mot. funct. in schizophr. I. The Journ. of Gener. Psychol., 15: 63, 1936.
20. Storch, A.: The primitive archaic forms of inner experiences and thought in schizophrenia. J. Nervous and Ment. Dis., Monogr. Series, 36, 1924.
21. Vigotsky, L.: Thought in schizophrenia. Arch. Neur. Psychiat., 31: 1063, 1934.
22. Weigl, Eg.: Zur Psychologie sogenannter Abstractionsprozesse. I. Ztsch. f. Psychol., 103: 2-45, 1927.

DISCUSSION.

Dr. C. Macfie Campbell (Boston, Mass.).—Dr. Goldstein has covered such a broad and complicated field that it is not easy to choose one topic or to formulate clearly any single issue. Even the term "schizophrenia" might be open to discussion. The difference between the organic and the functional might keep us here many hours.

The term "deterioration" has been used in a sense that is somewhat unfamiliar to me, because it has usually referred to conditions in which we do not get restoration of function and deterioration as an escape from a difficult adaptation is a rather novel concept.

The attitude towards the abstract and the concrete is difficult for me to be quite clear about, and the special tests which are perhaps the key to the communication have had to be eliminated owing to want of time.

There is one point I think that Dr. Goldstein has emphasized which is important. We have learned that in studying the basal functions, merely to take one element and contrast that element in so-called schizophrenics with normals or in the individual from time to time is of comparatively little value. We have to take a variety of biochemical or physiological factors and see whether in the totality of these reactions we find some abnormality of importance, and we have to study these elements in their totality in reaction, perhaps to certain environmental stimuli, and that, I judge, is essentially the crux of Dr. Goldstein's paper; namely, it is not by studying traits or individual elements, but by studying a group of reactions to certain concrete situations, with regard to which Dr. Goldstein has already attained considerable success in the study of the organic condition.

We shall therefore wait, I think, with considerable interest the publication of these tests of Dr. Goldstein's and apply them to our cases, not to schizophrenics, but rather clearly defined conditions which we can more or less formulate and think of in less glowing terms than terms of schizophrenia.

Dr. Arnold Hackfield (Seattle, Wash.).—A question that arose in my mind was, How does Dr. Goldstein apply these tests to the catatonic where there is mutism and no response?

We are all familiar with the use of sodium amytal in catatonics and when given they will respond to the environment, whereas without it, they will not. Dr. Eisner just read a paper that somewhat corroborates what Dr.

Goldstein said about the use of the Rorschach test before and after treatment, and he found that there wasn't any essential change as far as the schizophrenic personality was concerned, because experience with the use of the pharmacological shock treatment shows that the relapse rate is just as great as in those cases that had spontaneous recovery.

All those who are acquainted with the Rorschach test know that a certain personality past puberty causes the test to remain more or less constant and will not change; but in working with the Rorschach, I have found that quite marked states of intoxication will produce a change in the basic elements of the Rorschach.

I would ask Dr. Goldstein whether or not he has used any pharmacological drugs, whether they be sodium amytal or alcohol in large quantities to see whether the test results would change.

As far as metrazol is concerned, I know from practical experience one particular case who was unable to transfer in psychotherapy under ordinary circumstances, and I gave him a shot of metrazol about eight o'clock in the morning, had him in for psychotherapy about ten or eleven, and he was doing fine. But the next day there was a gradual recession of his affect and basic element inhibitions were coming out again. I think that in some measure explains why these patients relapse but, on the other hand, I think you can change the rapport of a schizophrenic with pharmacological methods.

Dr. Kurt Goldstein (New York, N. Y.) (Closing).—Concerning the question of catatonic patients with mutism, these tests are especially applicable, because all these tests are performance tests and the patient doesn't have to say any words. I have seen patients in mutism and in the catatonic state who didn't move and didn't do anything, but when we began to play on a table with some blocks which we use, they come nearer and gain interest and do things like a normal person. They behave as if they would be normal, but they behave only in a concrete way; but they do something. I think there is some preference for these tests. They bring the patients into that condition in which they are able to do some things. They cannot do some things best because of the usual environment, and especially when we speak to them, they cannot react in the right way because they don't understand our language, because they are living in a concrete level and we are speaking from our more abstract level. Therefore, I think these tests are revealing, that they are applicable even to the catatonics and to schizophrenics because they are to be performed in the concrete level.

I have no experience with the effect of pharmacological treatments in this test, but as far as the Rorschach is concerned, I think the Rorschach gives more an aspect of the general personality of the individual, not so much the change of the personality by the schizophrenic disease. So the Rorschach after treatment is not so different. But in other tests you can find this more concrete behavior is changed to a certain degree and the patient is more inclined to behave a little more abstractly. Therefore, I think we have to use many tests, a whole battery of tests, because each of these tests will give us a little insight. But I am also conscious of the fact that we are in the beginning of all of this work.

USE OF METRAZOL IN THE TREATMENT OF ACUTE ALCOHOLISM.*

By LEO L. ORENSTEIN, M. D., KARL M. BOWMAN, M. D., JULIA R. KAGAN, M. D., and WALTER GOLDFARB, M. D.,
New York City.

At the psychiatric division of Bellevue Hospital we have the opportunity of studying alcoholism in its various manifestations and observe the many problems it creates. In 1938 we examined and discharged 12,582 patients with a diagnosis of alcoholism (Table I). Of these 1301 were psychotic. There were 10,361

TABLE I.

Patients Treated for Alcoholism in 1938.

	Males.	Females.	Total number patients.
Psychotic	1,026	275	1,301
Not Psychotic	9,325	1,956	11,281
Total	10,351	2,231	12,582

males and 2231 females. About 81 per cent of these patients are treated on the alcoholic ward.

While the chronic alcoholic requires longer periods of treatment and affords greater opportunity for observation we have been able to make several interesting studies in cases of acute alcoholism. Goldfarb, Bowman and Parker [1] recently reported the effect of glucose and insulin on the rate of disappearance of alcohol from the blood. Bowman, Orenstein, Wortis and Goldfarb [2] have found significant changes in the carbohydrate tolerance in the alcoholic patients.

From the clinical point of view acute alcoholism presents two difficult problems. These include the disturbed, resistive, violent

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alcoholic and the patient in an alcoholic coma. Ordinarily one resorts to sedatives, restraints and seclusion for the former group and various stimulants for the latter. It was suggested to one of us (K. M. B.)[3] that metrazol (penta methylene tetrazol) might be used as an aid in the management of the problem of acute alcoholism. Since our service admits such a large number of alcoholics, methods which facilitate the management of such patients are desirable.

Work on the pharmacodynamic effects of metrazol has revealed that this substance stimulates the respiratory center. It is also reported to act directly on the cerebral cortex. Masserman[4] in a recent publication refers to the literature describing the action of this drug. It has been stated that alcoholism in its milder stages of narcosis depresses inhibitory functions of the cerebral cortex. Such action then permits the more primitive tendencies to come to the fore and result in the clinical picture best described in the non-psychiatric terminology as the "fighting drunk." If the narcosis is carried further a state of coma results, in which respiratory depression and at times acute failure may occur. We are interested in studying the effect of metrazol on alcoholic narcosis.

METHOD.

Two groups of patients were selected at the time of their admission to the hospital. The criteria determining the choice were: group A had (1) history and evidence of recent alcoholism, and (2) a clinical picture of acute disturbance, resistance and violence; group B had (1) the same history as group A and (2) deep coma or (3) coma complicated by respiratory failure.

Immediately after admission such patients were taken to the emergency room and received 5 cc. of 10 per cent solution of metrazol intravenously, the rate of injection being about 30 seconds for the 5 cc. We found that in a person narcotized with alcohol, this speed of injection failed to induce a general convulsion. In some individuals, twitching of the face appeared. We refer to this dose time factor as a "subconvulsive dose."

It has been shown[4] that metrazol given intravenously in large enough doses, or applied directly to parts of the central nervous system will cause stimulation of this organ. By subconvulsive dose

we mean that the injection is given fast enough to permit a concentration of metrazol in the blood per unit of time to be large enough to stimulate the central nervous system notably but not enough to lead to a generalized seizure. After the injection the patient was kept in the emergency room for a short time and then sent to the ward best suited for his clinical condition. Sedation and other treatment were ordered as indicated. It was found, however, that a large number of the excited alcoholics would quiet down in two to ten minutes after the injection and no further special treatment would be necessary. Comatose patients frequently regained consciousness in five to thirty minutes and were able to give an adequate admission history.

In attempting to evaluate improvement we considered the time and the patients' response clinically (Table II). The acutely excited

TABLE II.

50 Cases Treated with Metrazol.

Type of patient.		Sedation.	Improved.	Not improved.	? Improved.
Excited	34	10	25	7	2
Comatose	16	2	10	4	2
Total	50	12	35	11	4
Per cent..... ..		24	70	22	8

Under "sedation" we include those patients who received medication during the first 24 hours in the hospital.

patient was classified as improved if he showed a change either immediately after the injection or within twenty minutes. The improvement in the clinical picture consisted of disappearance of the acute symptoms of excitement. In the comatose group the patient was considered improved if he awakened immediately or within thirty minutes. In those with acute respiratory failure, we felt that an immediate change in the respiratory function with clearing of cyanosis and other alarming signs was sufficient change to consider such patient as improved.

Since the disappearance of alcohol from the body leads to clinical improvement we analyzed samples of blood from a number of our improved cases. We found no notable change in blood alcohol concentration as determined by Abel's oxidation method.[5]

We present brief summaries of case histories:

CASE I.—E. P., male, 38, was well developed and in good physical condition. A friend who brought the patient to the hospital stated that they had been drinking. The patient had more than the others in the party and became agitated, violent and shouted, "I want to kill somebody." When seen in the admitting office he was noisy, resistive to examination, violent, continued to shout "I'm going to kill somebody, I want to kill them." Contact was impossible.

The patient was restrained on a stretcher. It required several male attendants and nurses to do that. He was given 5 cc. metrazol intravenously without immediate effect. It was felt that the injection was given too slowly and another 5 cc. dose was injected and he became quiet at once. His breathing became deep, his face flushed. He remained quiet from this point on; slept without sedation. The next morning the patient stated that when he is drunk he becomes very difficult and tends to be violent. He added that two years before he attempted suicide while in a state of intoxication.

CASE II.—J. S., male, white, age 44. At the time of admission the patient was noisy, restless, thrashed about on the stretcher, totally uncooperative. He was given 5 cc. metrazol intravenously and became quiet and cooperative at once. No sedation was required. On the following morning the patient said, "I was very sick when I came here. You gave me an injection, for a moment after that I saw stars, it was all over soon and when I came to the ward I was getting very sleepy."

This patient had four previous admissions because of alcoholism; each time he was described as noisy, uncooperative, resistive and was given sedation in the form of paraldehyde.

CASE III.—J. R., colored male of 29, was found on the street in deep coma. At the time he was seen by one of us he showed pinpoint pupils, strong alcohol odor to breath, complete areflexia; a divergent squint. No response to painful stimuli could be elicited. He was given 5 cc. of metrazol intravenously and awakened immediately. He answered questions, gave his name and age correctly. Three minutes after injection he vomited after which he continued to be in good physical condition. He slept well and was ready for discharge the next morning.

CASE IV.—A. A., a middle-aged white male was brought to the admitting office, in deep coma, pupils small and fixed. No response to stimuli. There was moderate cyanosis of the skin and respiration was shallow. Pulse was regular and of fair quality. While the ambulance surgeon who brought the patient to our hospital was writing his note, the patient became very cyanotic. His face appeared bluish-black, respiration began to fail. The patient's mouth was opened and his tongue pulled forward. This however, did not change his condition, nor did respiration improve. He was rushed to the emergency room and was given 5 cc. metrazol intravenously. Immediately after injec-

tion he sneezed a few times, then he coughed. It appeared that he might vomit and his head was supported by a nurse; however, he did not vomit. The respiration began to increase in rate and depth. The blue-black color of his face began to disappear and in about five minutes he showed flushing of the face and mild perspiration. The pupils were wide and responded to light. He still made no response to painful stimuli. The patient was taken to the ward and placed in bed after a spinal puncture revealed clear cerebro-spinal fluid. He continued to breath regularly and deeply. About one hour after the injection he responded well to stimuli. The next morning he appeared in good condition, was up and about the ward, had no knowledge of what had happened to him on the day of admission. He said, "I was drinking and that is all I can remember. I feel pretty good now. May I go home?"

DISCUSSION.

The narcotic effect of alcohol has long been known. Primarily in small doses it acts on the cerebral cortex and depresses the inhibitory centers. Clinically this results in acutely agitated patients. When the narcosis is carried further, the patient lapses into a comatose state. In the present study we have chosen a group of patients who appear to fall into either of these classifications. The administration of metrazol to these individuals was followed by a marked improvement in the clinical state. Such improvement manifested itself in the arousal of the comatose cases, and sedation of the excited group. This improvement was not due to any changes of the concentration of alcohol in the blood. It is suggested that this apparent biphasic effect of metrazol is due to a direct stimulation of the narcotized cerebral cortex. In the mildly narcotized, or agitated group, the improvement is ascribed to a stimulation of the depressed inhibitory centers. Improvement in comatose cases is considered to be due to general stimulation of the central nervous system.

SUMMARY.

The problem of acute alcoholism in a large psychiatric service is considered. Metrazol was given intravenously in "subconvulsive" doses and found to be efficacious in the treatment of certain types of alcoholic intoxication. The result with metrazol in acute alcoholism is ascribed to the action of the drug as a direct stimulant of the central nervous system.

BIBLIOGRAPHY.

1. Goldfarb, W., Bowman, K. M., and Parker, S.: The treatment of acute alcoholism with glucose and insulin. Am. J. Physiol. (Proc.) 1939.
2. Bowman, K. M., Orenstein, L. L., Wortis, J., and Goldfarb, W.: The sugar tolerance of alcoholic patients. Proc. Soc. Exper. Biol. & Med. (in press) 1939.
3. Preston, G. H.: Personal communication.
4. Masserman, J. H.: Action of metrazol (penta methylene tetrazol) on the hypothalmus of the cat. Arch. Neurol. and Psychiat., 41: 504, 1939.
5. Abels, J. C.: Simple method for determination of alcohol in blood. Proc. Soc. Exper. Biol. and Med., 34: 346, 1936.

PSYCHOSES ASSOCIATED WITH EPILEPSY.*

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During the course of one year 22 patients were admitted to the Boston Psychopathic Hospital because of psychoses associated with epileptic seizures not complicated by syphilis, brain tumor, recent head injury or other causes of extensive damage to the central nervous system. In the present communication these cases will be reviewed, together with the literature on the subject, and theoretical considerations will be discussed.

Literature.

In 1881, Hughlings Jackson [1] described the post-epileptic psychoses as stages of reevolution from the dissolution occurring in the seizures, with various degrees of loss of control by higher neurological levels, and consequent irruptions from lower levels, the degree of organization of which depended on how far reevolution had gone.

Raecke [2] in 1903, classified postparoxysmal conditions as stupor, delirium, mixed stuporous and delirious states, and paranoid and maniacal conditions. Kraepelin [3] added the "besonnene Delirium" with less clouding and more complicated activities, and spoke of the propensity toward dipsomania of epileptic patients suffering from depression (Verstimmung). Binswanger [4] found that post-epileptic psychoses usually followed repeated successive seizures. The shorter the intervals between seizures, and the deeper the exhaustion, the more likely was there to be a clouded state. Intoxication, affective shock, or trauma might help bring on a psychosis. The descriptions and classifications remain essentially the same in more recent publications, such as those of Bleuler [5] and Strecker and Ebaugh.[6]

More searching analysis first concerned the intellectual processes. Christoffel [7] stated that in clouded states of deteriorated epileptics

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the thought process is slowed, with a tendency to perseveration. Comprehension, and ideation are unclear. Orientation is disturbed, memory full of gaps. The patients cannot find words, their words are often mutilated, and syntax so suffers as at times to amount to agrammatism. Since precise expression is hard, they speak in generalities.

With growing interest in psychological dynamics, they were soon applied to epileptic states. Maeder,[8] the psychoanalyst, noted the frequency of sexual material in epileptic psychoses, relating it to early sexual development. He described masturbation, sucking, smearing, homosexual and heterosexual activities, exhibitionism, and erotic fantasies often related to religious delusions. Rosenhain [9] told of a soldier wounded in the leg with rapid complete recovery, who two years later in a post-epileptic clouded state developed in the wounded leg a psychogenic limp which disappeared the next day. Schilder [10] determined after recovery from clouded states the retention of poems and pictures learned during them, finding great variability often not corresponding to the apparent confusion during the psychoses. He concluded that the amnesia was not entirely due to faulty perception, but also (as in memories of dreams, hysterical episodes, and hypnotic states) partly to repression. He noted how frequently the idea of rebirth, so common in dream fantasy, appears in clouded states. Binder [11] concluded from a very interesting case that epilepsy is an organic brain process with slow progression in whose course transitory crises arise, taking the forms of anxiety attacks, absences, and seizures. The organic shock brings repressed material to the surface. The amnesia after lighter clouded states may be lifted by hypnosis, but not after deeper states, where the organic disturbance is too severe. L. Pierce Clark stated: [12] "The fit disorder and its periodic loss of consciousness may thus be regarded as condensed episodes of intensive epileptic moment. The furors, deliria and the manic excitement are similar chaotic rushes of the organism to a more or less certain final goal in status or dementia."

In accord with his theory of a disordered function of the vestibular system in epilepsy, Stauder [13] stimulated the vestibular apparatus in two cases in whose post-epileptic psychoses feelings of world destruction were uppermost. In one case calorically stimulated during an attack the feeling of world destruction appeared.

The other patient who had had false perceptions of movement while in an attack was vestibularly stimulated when not in one. While imagining the same scenes he had seen in the attack, the same false perceptions arose again, and with them a feeling of world destruction and unpleasant mysteriousness. Schönmehl [14] provoked convulsions with metrazol during three clouded states, concluding that their duration or severity was thereby decreased. Because he found that epileptics in clouded states could more easily name objects related to themselves than those related to other people, Stengel [15] supposed that ego relations were profoundly disturbed.

Several authors, citing cases of chronic psychoses among epileptics, and of seizures occurring in those already having schizophrenia or manic-depressive psychosis, have written on the relation of epilepsy to other psychiatric conditions.[16, 17, 18, 19] Lange [20] suggested a serial classification, beginning with epileptics never having clouded states, passing to those who have had them, thence to those with states accompanied by isolated or outspoken schizophrenic symptoms, next to schizophrenics with isolated seizures, and ending with schizophrenics without convulsions. A similar series may be constructed with relation to manic-depressive psychosis. Strauss [21] described an excellent case of a woman, long subject to post-epileptic clouded states, who had an infant during the course of one. On awakening she denied the child was hers. This and associated delusions persisted (interrupted by seizures and clouded states) until death years later. Strauss believed the delusionary condition to be secondary to the damage of the epileptic process, rather than that there were two independent coexisting processes. Such conditions are called "symptomatic schizophrenias" by Gruhle.[22]

Personal Cases.

Of our 22 patients, 14 had clouded states, three chronic psychoses, one deterioration with psychoses, one a severe behavior disorder, and three were unclassified. Their ages on first becoming mentally ill ranged from 12 to 49 years. In all a considerable interval (from 2 to 45 years, averaging about 15 years) elapsed between the onset of their seizures and their first attack of mental illness. Twelve were male and ten female.

Their family histories were, in general, not remarkable. An exception was a patient who had many clouded states of short duration, whose father was said to be epileptic and alcoholic, whose

mother had been in the Boston Psychopathic Hospital and diagnosed paranoid condition (recovering in 5 years), whose three paternal uncles were alcoholic and whose father's two nephews were said to have been epileptic. Another patient's maternal aunt died in a state hospital. Only four other patients were known to have a parent with sign of nervous abnormality; two alcoholic, one depressed and one migrainous.

Eleven cases may have had early brain damage although there were no residual neurological signs.*

Partly because many informants knew little of the patients' early lives, special traits before seizures began were seldom found. One patient had enuresis until 10 or 11 years, and was described as a stubborn and self-willed child, greatly attached to his mother. Another was said to have been very stubborn. The patient who later had so many short clouded states did not talk plainly until five years, and then stuttered for some years.

All of our cases had typical grand mal attacks without localizing features. Five also had petit mal attacks. In very few was an aura described; none out of the ordinary. The frequency of seizures varied greatly—one having only nine observed seizures in the previous six years, another having grand mal attacks up to seven times daily. The average was about once a month.

Of those patients who had clouded states, eight had had only one, five had had from two to five each, and one had had numerous excited episodes lasting 10 or 15 minutes each. Not counting this last case, the clouded states were from about half a day to 35 days long, averaging 4.5 days for 21 episodes. Often it was hard to say just when an attack ended, because the longer ones approached normality so gradually and because just where clouded state ended and deterioration began was often impossible to say.

In a majority of cases, these states were preceded by one or more grand mal seizures.† One had several petit mal seizures as well.

* Seven had possible birth injury, due to instrumental birth, breech delivery, premature birth or prolonged labor. Three had head injuries (none severe) shortly before the onset of seizures. Two had convulsions in early childhood one associated with other illness. Another's convulsions began shortly after pneumonia.

† Six by one, three by two, two by three or four, one by six (in four days), one by eight seizures daily for five days before, and three by a series of seizures, the exact number uncertain.

Since the patients were not under constant observation, it could not be said for sure in the absence of a history of preceding seizures that none had occurred. The interval between the last seizure and the onset of the psychosis varied from a few minutes to nearly two days.

All those in clouded states were overactive at some time (from mere restlessness to extreme combativeness and assaultiveness). Only two were stuporous for short periods. Speech and mood were variable.*

Delusions were manifested by 12 patients—four having religious delusions, three sexual, four persecutory, one somatic. Some were most bizarre—for example, one said he had lead put into his testicles when he was confirmed and this was later changed to gold. In a second attack the same patient claimed that his sputum was really vaginal secretions being piped up from a room below from negroes kept there, and that in another room the Pope's gold was kept. Twelve had hallucinations, eleven of whom had delusions as well. Six had visions, including dead people, snakes, pigs, bugs, pigmy men and various living people. Seven had auditory hallucinations, including voices, music and the sound of airplanes. There were no olfactory, tactile, nor gustatory hallucinations. Of those who responded to questions, only two had unimpaired memories. The others had better preserved remote than recent memories. Where orientation could be determined, it was poor in all but one case. Insight during attacks was absent in those with great overactivity and disturbed moods, while in calmer patients insight was partially present. The degree of amnesia for the attack after recovery was greatest where the most excitement prevailed during the psychosis, and was least in the more elaborate, dream-like states.

Two of our patients took up Christian Science in hope of preventing their attacks.

The case histories of two patients with more dream-like clouded states are given in more detail:

CASE 1.—A 39-year-old salesman, single, white, high-school graduate, was admitted July 7, 1938. He was of normal antecedents and his childhood was

* Four patients were overtalkative with push of speech, five undertalkative, two spoke in fragmentary phrases, three were profane and obscene, one was slightly incoherent. Three patients were elated, seven depressed, two suspicious, two angry and irritable, one childishly labile in mood.

uneventful. Though a Catholic, he had gone to Church seldom since the age of 18. Since 19 he had had 10 or 12 grand mal seizures yearly, as well as petit mal attacks.

Because of a business service in 1932 to a convent whose patron was St. Theresa the nuns had written to him several times, saying they remembered him in their prayers. Eight months before entry, after a seizure, he had a clouded state beginning with a vision of St. Theresa. Feeling directed by an unknown force as though in a dream, he took a train to Washington. There he went to a priest he knew to confess and take Communion (for the first time in years). In a few days the patient cleared up.

He was practically well during the following months until he had five seizures in a day, four days before entry. The next day he visited his doctor. He saw a Catholic religious picture in his office, making him believe that the doctor (whom he had thought a Jew) must be "psychic" like himself. He believed he and the doctor had a great plan, shared only with God and Jesus, for the good of America. The next day he went to a railway station in Boston, always accompanied by the rhythm of the phrase "God help me," and hearing strains of popular music in his head. Men's voices told him to buy a book. With 50 cents gotten from a newsboy in return for his bank-book he bought the book and gave it to a passing boy. Later he felt as though already dead, but still among the living because not yet fit to enter heaven.

On entry he was restless, cheerful, self-confident, with a slight incoherence of speech, and only approximate orientation. He talked vaguely of his plan to aid America, and of hearing voices and music. After six days of gradual improvement he seemed himself, and joked about his dream. He was discharged August 12, 1938.

In these clouded states we have (with poor judgment and disorientation) dream-like ideas and acts harking back to long dormant early religious training, which had perhaps been stimulated by the letters from the convent. There was also a note of rebirth to a happier life, with strong feelings of self-confidence and personal value—attributes sadly lacking in his life previously.

CASE 2:—A 22-year-old single, unemployed female, high-school graduate, admitted October 20, 1938. Family history, birth and early development not remarkable. Frequent grand mal attacks since 14 prevented her working. When 19 she had a series of convulsions a week after being jilted by her fiancé, and went to a mental hospital for seven days, because she became excited and said she was dead and had been hypnotized. She was discharged recovered. Meanwhile she took up Christian Science. Until her mother became ill in August 1938 her seizures were infrequent. On October 18 she had five convulsions, the next day was depressed and restless. Her mother died that day. On being told of this the patient became even more morose. The next day, however, she became hilarious, laughed at her father for crying, and said the funeral preparations were unnecessary. She was admitted that day. She was overactive, talkative, distractible, elated, and said

she had dreamed her mother was dead, but she was not, being still present in an immaterial way. Though oriented, and with good remote memory, she could not relate coherently the events of the past few days, and insight was lacking. In two days she became quieter, and recognized that her mother was dead. Then on the fourth day after entry she had another seizure, and relapsed to her former state of elation and talkativeness. She then believed another patient to be her mother, that the hospital might be heaven, and that she was to be cured of her sickness. A day later, though still very cheerful and sociable, she had given up these ideas. When discharged on the tenth day she had returned to normality.

This patient expressed simple wish-fulfillment in her delusions. The elated mood, though opposite to that demanded by the real situation, was in keeping with her delusions.

The following is a complicated case illustrating diagnostic difficulties that may arise.

CASE 3.—A 16-year-old, unemployed boy, admitted last on May 16, 1938. Family, birth and developmental history not remarkable. Petit mal attacks since 12, grand mal attacks since 14, when he stopped school at first year high school. He was unsociable, egotistical and sensitive. At 13 he first misbehaved by cutting off his mother's house plants. Once or twice a week he had attacks of sudden yelling or throwing an object across the room, which his mother attributed to trying to get his own way. During the month before his first convulsion he hit his father with eggs, threw knives at boys, and the like. Later he shot out house lights with an air rifle, shot at his mother, broke dishes, stuck knives in the walls and fought with his father (all unrelated to seizures). He was admitted to the Boston Psychopathic Hospital in March, 1937, January, 1938, and May, 1938, because of similar behavior, the last time for putting bichloride of mercury in his mother's preserves. He told of deep resentment because of his father's attempts to masturbate him. At a state hospital he had grand mal attacks several times weekly. He escaped home once, having to be brought back by police, after barricading himself in the attic. At the hospital he was often irritable and tried to get patients to escape. Physical examination was always negative and there was no evidence of deterioration.

In this case some attacks may be unmotivated equivalents, others more in the nature of temper tantrums partly based on instability associated with his epilepsy, partly on resentment against his father. They are rarely related to convulsions. In no episode was there evidence of clouding of consciousness.

Because of recent interest in the relation of epilepsy to schizophrenia, abstracts of three chronic psychoses are given below:

CASE 4.—A 40-year-old housewife, grammar school graduate, last admitted December 16, 1937. Family, birth and development histories not remarkable. Grand mal attacks began at 17. She was married at 21 to an alcoholic who

abused her and called her unfaithful. She was quiet, unsociable, with no interests outside home and religion. Since marriage she had seizures nearly every day.

Admitted June, 1924, with a post-partum psychosis, refusing food and saying the baby was dead. Discharged improved, October 1925. She returned April, 1926, in a clouded state, probably post-paroxysmal, which cleared by June. Again she returned in March, 1927, after three months of depression, admitting hallucinations, saying electric wires were sending messages to her but well-oriented. She became symptom free in 20 days.

For the next 10 years she had five or six seizures about once a month. In 1937 she developed queer ideas again, and was admitted in December. She was underactive, with monotonous speech and some depression. She believed everyone's thoughts were read by a syndicate of great corporations through electricity and telephone wires. Orientation and memory were good. Hallucinations were not elicited. During the following year she did not improve, and convulsions continued unabated.

CASE 5.—A 46-year-old foreign-born, unemployed leather worker, admitted January 11, 1938. Since 28 he had seizures, lately up to two or three times daily. In the past year he heard God telling him he was a fool and would not live long, and saw God at intervals, and in the past three months he thought people were laughing at him. In the hospital he was quiet, seclusive, fairly cheerful, with ideas of reference and a belief that God told him to go to church. Because of language difficulty it was difficult to say if he were deteriorated. Orientation and memory were good. During eight months in another hospital he has remained about the same. His seizures have continued as before.

CASE 6.—A 48-year-old single, unemployed woman, admitted October 28, 1938. Uneventful history until aged 20, when she began to have grand mal attacks about once a year, and petit mal attacks more often, so she had to give up work as a telephone operator. At 41 after her mother's death seven years before entry she became more religious. At 44, four years before entry, she began to say she got letters from God. On holy days she sat idle about the house in her best clothes. Once she said she was the Virgin. After attacking her sister for intercepting her mail she was brought to the hospital. She was talkative and rambling, with a cheerful mood. She told of writing to the Cardinal, and of the priest saying she was the Virgin. She believed she had influence over the Pope. She saw miracles in commonplace events on the ward. Hallucinations were denied, orientation and memory were good. Her last seizure had been two months before entry. Three months after transfer to another hospital she was reported unchanged, without deterioration, and having had no more seizures.

Despite the onset in these three patients of chronic psychoses resembling schizophrenia, seizures continued as before.

Electroencephalographic Studies.

Through the courtesy of Dr. K. H. Finley, we were able to obtain electroencephalographic records on four of our patients. Two taken while clouded states were in progress showed generalized abnormally slow rhythm, another in a patient recently recovered from a clouded state showed both slow rhythm and a more rapid rhythm superimposed on normal alpha waves. One of the unclassified patients with a more chronic psychosis had an essentially normal record.

A case admitted since the present series was closed had interesting electroencephalographic findings. During a stuporous phase of a clouded state, a markedly abnormal record was obtained, while after he had cleared up the records were normal.

Comment.

Psychoses associated with epileptic seizures in the present series of 22 patients occurred in three forms, most commonly as transitory clouded states, less frequently as chronic psychoses bearing resemblance to what is called schizophrenia, and as psychoses associated with deterioration of the intellectual faculties. Though clouded states may occur during the course of both other varieties, each may best be considered separately.

In accounting for the clouded states, we must consider what occurs in the nervous system during a seizure which may help to explain the subsequent psychic symptoms. Since one seizure, with its exaggerated discharge of nervous energy, produces considerable fatigue in the central nervous system, the degree of exhaustion produced by a succession of seizures (in an already weakened brain) must be extreme. The electroencephalographic record during a seizure—very rapid oscillations of high voltage succeeded by almost entire absence of activity—sustains this reasoning. Clouded states appear during restitution from this exhaustion. Wilder Penfield [24, 25] has described transitory vascular changes in the cortex of patients with focal epilepsy, which he suggests may be the physiological correlate of "post-epileptic automatisms with amnesia, with effective anemia remaining in the region in which consciousness is represented." A patient in our series who had periods of confusion

lasting 10 or 15 minutes, occurring frequently not in association with convulsions, may have had some such process going on synchronously in his cortex.

Since it is known [26] that prolonged diminution in oxygen intake will result in mild reversible psychotic manifestations, it might be argued that with the oxygen deprivation occurring in a succession of seizures the effects are cumulative, especially in somewhat deteriorated epileptics. Since equivalents without preceding convulsions may occur, and since clouded states may appear after only one seizure, oxygen deprivation due to seizures can be no more than a small contributing factor.

Post-epileptic clouded states may be compared in many ways with states of pathological intoxication due to alcohol (see Binder).[27] In pathological intoxication consciousness is transitorily disturbed, with a marked disturbance of orientation associated with an unreal, phantastic outlook. Like the more overwhelming epileptic states, in many of the pathologically intoxicated the onset of the condition is sudden, with delusions, isolated hallucinations and frequent asocial behavior—profanity, obscenity, threats, assault, and sexual delinquencies. Amnesia is either complete, or only insular memories remain. Other cases—as with the more dream-like clouded states—have less disturbed orientation and mood, less prominent motor activity, and more hallucinations, and infrequent asocial behavior. There is less amnesia later on, and reconstruction of the events of the episode may be aided by hypnosis. In those predisposed, either by constitution or by psychogenic factors, alcohol may thus benumb the controlling nerve centers in a way very similar to that in which the epileptic seizure may. According to Gruhle,[28] epileptics suffering from depression (Verstimmung) develop pathological intoxication after ingestion of alcohol.

Chronic psychoses in epilepsy unassociated with deterioration present their own problems. To attribute the epilepsy and the psychoses to two distinct disease processes makes the matter no more clear. It is more logical and economical to consider them two manifestations of the same disorder. In the present state of our knowledge no more can be said. The mutual antagonism between epilepsy and schizophrenia of Von Meduna does not seem to hold in the present cases.

The psychoses associated with deterioration, because presumably secondary to brain damage, are more comprehensible, though it is impossible to explain in detail the individual phenomena.

The clouded states offer the psychopathologist an unusual opportunity to study the structure of the mind. In the dream-like states with little amnesia, material supposedly arising from subconscious levels may be observed during the psychosis, and later compared with the patient's memory of what occurred, and an attempt may later be made to account for it in terms of his past experience. Dream interpretative methods may be supplemented by the addition of contemporaneous observations made on the spot.

The frustrations and restrictions in the lives of epileptics are notorious. They cannot travel about freely, they have few friends, their careers are cut short, they seldom marry, so that their existence is nearly always drab and unhappy. The deeper clouded states, with their wild and aggressive behavior, may be looked upon in one sense as blindly furious rebellions against this fruitless existence, releasing tumultuously the pent-up feelings of years. In the more dream-like states this release is perhaps more gradual and at a higher level, with more symbolic expression in the forms of religious and wish-fulfilling phantasies, and less crude aggressiveness. In our series these dream-like conditions occurred in the more intelligent patients. Aggressive impulses may be turned inward, giving rise to the self-mutilating acts occurring not infrequently in these states. If such a mechanism is at work, making the lives of these epileptics more happy by stimulating their interests and capacities and broadening their friendships should lessen the liability to clouded states. It is perhaps significant that one clouded state (case 2) came on after the patient was jilted, and the other in the same case when her mother was on the point of death.

The dream-like states have many points in common with transitory schizophrenic attacks, just as the chronic psychoses do with chronic schizophrenia. There is more emotional display, accompanied by a certain amount of disorientation perhaps secondary to emotional turmoil and preoccupation. They are benign, with relatively little amnesia, but are subject to recurrence. Religious and sexual symbolism are prominent in both, often with vivid hallucina-

tions. The study of epileptic psychoses and of schizophrenia may throw light on each other.*

When successive convulsions occur, especially if the patient is known to have had a clouded state before, intravenous sedation, inhalation of a gas mixture high in oxygen content, and other measures such as those recommended by Lennox and Cobb [23] may be used, to prevent further attacks. One clouded state developed in a patient who had had a series of seizures following an unintentional reduction in the amount of phenobarbital she was taking. Greater care in the administration of sedatives would thus prevent a few psychoses.

For the psychiatrist the problem at present is to deal with the psychoses once they have arisen. Since the clouded states are temporary, efforts may be directed at attempting to shorten them. Evaluation of the results of such attempts, because of the extreme variability in length of different attacks, would be difficult, and could be made only after the study of a large series of cases. The treatment of chronic and deteriorative psychoses will probably remain for some time in its present unsatisfactory state.

Summary and Conclusions.

The literature on psychoses associated with epilepsy has been reviewed. Twenty-two personal cases are described. Fourteen had clouded states, which were found to be transitory obscurings of consciousness of varying depths, developing some years after seizures first began, usually occurring after several successive convulsions, characterized nearly always by overactivity, by variable speech abnormalities, wide variations of mood, visual and auditory hallucinations, delusions, disorientation, poor recent memory, defective judg-

*A case seen since the end of the present series (cited under encephalographic studies) is especially interesting psychopathologically. After recovery from a clouded state during which he had tried to put out his eyes with his fingers, the patient said that in that time he had first thought he was Christ, his father God, and his mother the Virgin. He thought his father was going to bring his mother in to his room so that he could have intercourse with her while the angels waited. A few days later he thought that if he killed himself he would go to heaven as a saint. Since beating his head against the floor did not succeed he tried poking his eyes into his head with his fingers. If he weakened he thought he would go to hell.

ment, and little or no insight. In some cases, the content of the psychosis could be partly traced to earlier factors in the patient's lives. The clouded states were compared to alcoholic pathological intoxication. Three had chronic psychoses resembling schizophrenia. One other was considerably deteriorated. The rest were unclassified. The neuropathological processes underlying these conditions are unknown. The clouded states contribute to the study of psychopathology. Psychotherapy might be of some value. There is great need of research in this almost untouched field.

BIBLIOGRAPHY.

1. Jackson, Hughlings: Selected writings, edited by James Taylor (2 vols.) London, 1932.
2. Raecke: "Die transitorischen Bewusstseinstörungen der Epileptiker." Halle, 1903.
3. Kraepelin: "Psychiatrie" (8th edition), Vol. III, Leipzig, 1913.
4. Binswanger, Otto: "Die epilepsie." Wien u. Leipzig, 1913.
5. Bleuler: Textbook of psychiatry (translated by Brill). New York, 1924.
6. Strecker & Ebaugh: Clinical psychiatry, 3rd edition, 1931.
7. Christoffel, H.: Zeitschrift f. d. ges. Neur. u. Psy., 55: 161, 1920.
8. Maeder, A.: Jb. f. Psychoan. u. Psychopath. Forsch., 1: 119, 1909.
9. Rosenhain, E.: Zeit. f. d. ges. Neur. u. Psy., 64: 96, 1921.
10. Schilder, P.: Zeit. f. d. ges. Neur. u. Psy., 81: 174, 1923.
11. Binder, H.: Schweiz. Arch. f. N. u. P., 19: 242, 1926.
12. Clark, L. P.: Ass. Res. in N & M Dis. monograph, VII, 67 ff, 1931.
13. Stauder, K. H.: Zeit. f. d. ges. Neur. u. Psy., 101: 762, 1934.
14. Schönmehl: Munch. Med. Wochenschr, 83: 721, 1936.
15. Stengel, E.: Jb. f. Psychiat., 54: 193, 1937.
16. Notkin, J.: Jour. of Nerv. & Men. Dis., 69: 494, 1929.
17. Weise: Arch. f. Psychiat., 95: 175, 1932.
18. Faver, H. E.: Psychiat. Quart., 12: 725, 1938.
19. Murphy, J. P. H.: Med. J. & Rec., 128: 77 & 110, 1928.
20. Lange: Munch. Med. Wochenschr., 71: 63, 1924.
21. Strauss, H.: Zeit. f. d. ges. Neur. u. Psy., 87: 784, 1929.
22. Gruhle, H. W.: Zeit. f. d. ges. Neur. u. Psy., 154: 395, 1936.
23. Lennox & Cobb: Medicine, 7: 105, 1928.
24. Penfield, W.: Annals of Int. Med., 7: 303, 1933.
25. Penfield, W.: Arch. of Neur. & Psychiat., 40: 417, 1938.
26. McFarland, R. A.: J. Comp. Psychol., 24: 147, 1937.
27. Binder, H.: Schweiz. Arch. f. Neur. u. Psy., 35: 209, 1935 and 36: 17, 1935.
28. Gruhle, H. W.: in Handbuch für die Geisteskranken, Vol. 8, special section 4, p. 669, edited by Oswald Bumke, Springer, Berlin, 1930.

SENSITIVITY.*

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"Sensitivity" is a word of many connotations. The multiplicity of meanings and the vagueness of the term have made for confusion particularly in psychiatric usage. It seems therefore desirable to define sensitivity, and to scrutinize at least some of the expressions of and attitudes toward sensitivity as experienced by our patients.

In the psychiatric literature the word sensitivity is essentially used in three ways: (1) purely descriptively; (2) in relation to paranoid, paranoic and schizophrenic disorders (Kretschmer); (3) denoting a personality trait so predominant as to permit the classification of a group of "sensitive psychopaths." In these three usages, without close analysis, the patient's sensitivity is regarded as a suffering, as a shortcoming or liability. The literature does not do justice to that kind or those kinds of sensitivity which represent an asset rather than a liability of the individual.

Before attempting a differentiation along such lines, we shall briefly discuss the "essence of sensitivity" which we call *emotionally charged impressibility*. This is in our opinion a common denominator in all varieties of sensitivity; it is manifested in various ways in every expression of and attitude toward sensitivity.

Subsequently we shall remark on two main groups of sensitivity, each one of which includes two distinct varieties, namely:

1. Sensitivity experienced as a liability by the individual and so evaluated by the group:
   a. Vulnerability.
   b. Touchiness.
2. Sensitivity experienced as an asset by the individual and so evaluated by the group:
   c. Empathy.
   d. Symphoria.

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A. The Essence of Sensitivity.

Following the definitions in Warren's Dictionary of Psychology we may say that sensitivity as a quality connotes "the susceptibility of an organism to stimulation." There is often a quantitative implication defined as "the capacity to be affected by stimuli of relatively slight intensity and to receive sensations therefrom and to respond thereto." Sensitivity is distinguished from sensitiveness "a psychophysical disposition marked by a low threshold for various stimuli or situations, including those which give sensations, pleasure or unpleasantness, or which elicit certain emotions." We do not deny the wisdom of this distinction which might be very valuable if there were two distinct adjectives available; we hold, however, that in most instances sensitiveness as defined in Warren has just been called and will be continued to be called sensitivity. We shall use the latter term throughout this discussion.

We start with a concept of sensitivity which includes: (1) the biological susceptibility of the organism to be stimulated, to be affected by stimuli and to respond to them; (2) the psychological or experiential capability of the person to receive and to be affected by impressions and to express them with definite emphasis on the emotions.

Sensitivity in such broad terms is common to all human beings, although in obviously varying degrees. In psychiatric usage the word has so far been used with these implications: (1) a high degree of receptiveness for stimuli and impressions; (2) the tendency to be emotionally affected by any stimuli and impressions, and to be affected more easily and to a higher degree (as manifested in the response) than other members of the social-cultural group; (3) the capability to be emotionally affected by experiences which other members of the group regard as either slight or irrelevant. The emphasis lies throughout on the individual's susceptibility and impressibility, not on the stimuli which may hit him or the occurrences by which he may allow himself to be impressed. If he were not so impressible he would not respond as he does. If there were not this impressibility he could not be either "sensitized" or "desensitized."

We are not here, however, dealing with any random sort of impressibility. The sensitive are possessed of a considerable degree of emotionally charged impressibility—this, indeed, *is* sensitivity.

Our main questions are: (1) How do they experience their sensitivity? and (2) What do they do about it?

B. Varieties of Sensitivity.

1. Sensitivity experienced as a liability.

Vulnerable and touchy persons carry as a burden their "considerable degree of emotionally charged impressibility." They are conscious of it with a sense of being "different." This difference is recognized whenever they estimate their endowments and compare themselves with other members of the group. The difference under consideration plays a greater rôle in the vulnerable than in the touchy, so much so that it is reflected in the attitude of the group which finds the vulnerable more or less inacceptable but gets along fairly well with the touchy. The sensitivity of the vulnerable and the touchy is expressed in their responses to the real or fancied moods, opinions or actions of others. What other people feel, think, do, or—just as important—what these sensitive imagine that other people feel, think, do about them hurts them either under practically all circumstances, if they are vulnerable, or under certain circumstances, if they are touchy.

a. *Vulnerability.*

Vulnerability and insecurity are peculiarly linked together. The vulnerable person feels himself a misfit; he must retreat to some environment in which his actions and reactions will not fall prey to criticism, competition, or repudiation. At times he may be able to live so guarded and ordered an existence that he may gain a temporary sense of security, but his vulnerability is potentially present. It may lie dormant for a time, only to torment him anew when his restricted environment is invaded, when new situations demand adaptation, when a carefully cultivated routine is interrupted.

We shall not argue here the question whence comes this insecurity that manifests itself in vulnerability or vice versa, nor shall we speculate whether early experiences have "sensitized" an individual to become more or less vulnerable. Frustration, real or imaginary, is the common lot of mankind. The vulnerable person meets frustration on his own terms. Most things in life signify danger to

him. He lives in fear, anticipates fear and reacts as if all encounters must eventually produce their share of "malice and evil."

The vulnerable person is mistrusting. He expects and detects disapproval, ill-meaning and insult always and everywhere. The most casual glance, a gesture, a word spoken too loudly or too softly will be ominous to him. While he is quick in insinuating and assuming offense, he is slow in ridding himself of suspicion. He is almost painfully aware of himself and of his "being different" from other people. He is timid and will not venture willingly into anything unknown. His ill-defined, ever-present fears prohibit him from sharing with others. He is incapable of emotional communication although he may desperately crave it in his loneliness. He finds himself unable to merge with a group and freely to participate in group activities. He shuns competition and cannot tolerate criticism, but he in turn will criticize and judge readily. More often he feels safer to endure in silence with self-restraint and sullenness, retaliating, as it were, in daydreams, fancying how he might punish and destroy his alleged adversaries. He cannot countenance deviation from his code of moral and ethical values, but may be pathetically cognizant of his non-conformity with the mores and standards of his culture which he experiences as threatening. At times the pent-up emotions may break through in irritability or impulsive actions, even in panic-like outbursts; but these are shortlived and followed by feelings of misgiving and shame, by greater constraint and more intensified withdrawal.

Some individuals rationalize their vulnerability. It becomes to them exceptional refinement or gentility, a peculiar brand of understanding or kindness; they may label it idealism. They try to use it as an asset, to capitalize it. They are delicate, mimosa-like creatures misunderstood in a coarse world filled with vulgar and low people. For all the rationalization the effect remains the same. Vulnerability drives deeper into isolation which, it is true, in some instances offers prestige. Forced into social contact a vulnerable person may parade his vulnerability with the expectation that others *must* be kind and gentle to one who is so obviously unfit for the combat of life.

The constant ministrations to self-protection in a limited, narrowed field of operation, make eventually for a rigid, inflexible individual who performs his self-saving devices in routine fashion.

It is not surprising, then, to find among the vulnerable, anancastic persons whose meticulous, fussy, detail-loving endeavors, compulsively executed, are directed toward the creation of order—order in the tangible environment, order among things that stand in spacial relation, order in events in temporal relation. Such time-table-like existence may develop into rut-formation.

No stratum of society is entirely free of these vulnerable creatures. The majority exist neither disrupting nor honoring the community in which they live. Some are considered valuable because they are painstaking, careful workers who dare not complain. To the outer world they seem to accept the status quo without grumbling and seem to wish for little if any change because they somehow realize that they cannot tolerate change. However, there are a few vulnerables who transmute their daydreams into cultural values and contribute to society from their ivory towers. We find the vulnerable among artists, mystics, poets, philosophers and scholars. We see them withdrawn, seeking refuge in some prestige-giving isolation.

The attitude of the social group towards the vulnerable is in general one of annoyed tolerance. The group has no active interest in them and tends to leave them alone. Most attempts to draw them into the community and its activities reveal that they are difficult to deal with because they are so easily hurt and always ready to withdraw. Even the few creative vulnerables are not expected to participate in the social life of the group. They are considered cranks and recluses, at the same time their talents are benevolently appreciated although with reluctance. After their death the reverent biographer crowns them with laurel.

b. *Touchiness.*

We have shown that the vulnerable is a permanent liability to himself and to the community whenever it cares to bother about him at all. Vulnerability is a dominating and pervasive trait of the personality. The vulnerable's patterns are suffering passivity and withdrawal lest he be hurt. His medium is loneliness. The touchy variety of the sensitive contrasts with this picture in a number of aspects. Any kind of personality may be possessed of the trait touchiness. Touchiness itself, though obviously determined by a

"considerable degree of emotionally charged impressibility," does not make for passivity and withdrawal, but finds expression in activity and aggressiveness; it permits the individual to keep his contact with the group, if in a temporarily more or less disturbing manner. The vulnerable withdraws. The touchy kicks against the pricks. The sensitivity of the vulnerable person is integral, of the touchy spotted. Touchy people are Achillean. When their weaknesses are flicked on the raw their sensitivity breaks out in unmistakable emotional display. A touchy person when insulted may, rather pointedly and demonstratively, withdraw, but this is only to let off steam: everyone, himself included, knows that he will shortly return to the group. The group does not take touchiness very seriously, does not consider it as strange but understands it and bears with it, often with an amused smile. The touchy individual is accepted as a member of the group set apart only during touchy spells. In most instances he faces reality just as does the group with which he rightly identifies himself.

Any "spot," any part of the individual's anatomy and physiology, any component of his emotional-intellectual makeup and any social-cultural and racial factor referring to him, his family or clan or wider group may serve the touchy person as an Achilles' heel. He experiences his "spot" as an inferiority which gives him a certain, but not permeating or overwhelming feeling of insecurity. He feels slighted and assumes that the other person wants to impress his superiority upon him (wants to "rub it in"). This the touchy is not able or willing to accept; he protests, he demonstrates, he antagonizes, he shouts or swears or cries, he threatens, he becomes violent, he compensates, he over-compensates.

In the choice of his sensitive spot the touchy is dependent on the standards and attitudes of his group.* Among the lilliputians small stature is not a point of touchiness. In our culture reference to an organic defect, be it internal or external, is considered tactless,—it is tacitly assumed that an individual will feel touchy about his physical or functional abnormality. The touchiness of the bald-headed man has served as content of many a joke. The plastic surgeon finds a lucrative field of operation where often the demand

* It does not fall within the scope of this paper to discuss the phenomenon of collective touchiness observable in families and other social bodies, in small and large religious, national and racial groups.

springs from an individual's touchiness rather than from functional necessity. The nose serves as just one illustration.

The concept of organ-inferiority (Adler) and the compensatory reaction of individuals with such inferiorities is not denied in this discussion. However, it must be stressed that in our culture it is the individual's attitude that in a broader sense determines organ-inferiority. The same organ abnormality may be evaluated very differently by different individuals; to some it becomes a point of touchiness, to others it does not, and even those who will become concerned about a specific organ-inferiority, will experience this inferiority in different ways, only one of which is over-compensation.

Touchiness about physique is commonly recognized. Less frequently commented upon, but frequently observed, is touchiness about energy output, temperament, intelligence and character.

Touchiness, although its manifestations are sporadic, shortlived and restricted, may create in some instances a more or less generalized tension and uneasiness. A person may be touchy on many issues and about many situations and may experience these in varying degrees of intensity and with many kinds of responses.

Because touchiness is not a dominating personality trait in the sense vulnerability is, its expressions are much more numerous and variable according to the makeup of the individual who happens to be touchy. All degrees of aggressiveness from gentle irony to manslaughter may be expressions of touchiness. If hysterical features are present, touchiness will be vented in the dramatic manner moving between martyr-like endurance and thundering pathos. One touchy person will be satisfied by a humorous or sarcastic verbal comeback; another one will "get even" after thoughtful planning; he may write a "furious" letter which perhaps he will not mail. Short indulgence in daydreaming is sometimes seen in which the insulted touchy individual fancies himself as superior and comforts his hurt pride with imagined reprisals and ideas of revenge on a grand scale. Such temporary getting away from reality is quite different from the isolation and aloofness of the vulnerables. In fact the touchy gets away to come back; even in his episodic fancy he is not unmindful of his group. The vulnerable in his withdrawal practices his immanent tendency towards isolation.

Not a few touchy individuals try to counteract their touchiness and find "prophylactic" measures to forestall its manifestations.

Most common among these is a particular, fastidious, obsessive concern with the touchy spot, a concern which has a certain protective value. The individual, for instance, who is sensitive about his family background may busy himself intensively with genealogical investigations. The finding of a fairly prominent, if distant, relative may not only allay his touchiness, but prove an effective tool of defense should this touchy spot be hit again in the future. Society smiles at such "spotted" anancasms, considers such "quirks" as inoffensive pastimes. Anancasm is a tool to help the touchy person reach a more comfortable adjustment. The anancasms developed by the vulnerable tend to spread over and to take hold of his whole life; they may, indeed, become his very way of living. In his time-table-like existence he constructs and fortifies his own aloof reality in which life is tolerable for him, in which, however, he thoroughly alienates himself from the group.

2. SENSITIVITY EXPERIENCED AS AN ASSET.

People come to the physician because they are and feel sick. Disease is considered a liability as the word patient, sufferer, clearly indicates. It is understandable that physicians have learned primarily to think in terms of liabilities. Only secondarily appreciation of the patient's assets developed when it was recognized how differently people take and go through illnesses. The psychiatrist has only relatively recently begun more fully to evaluate and therapeutically to exploit his patient's assets. This is strikingly brought home when we ask the patient for his cooperation. Nobody can cooperate in any venture if he exhibits only his liabilities, but hides his assets or refuses to use them. The degree and kind of cooperation we expect and eventually get from a patient is determined and definitely limited by a number of factors among which is his sensitivity. His emotionally charged impressibility and his ability (or inability) to do something about it and with it are highly important. In the psychotherapeutic situation vulnerability or touchiness in either physician or patient may seriously jeopardize rapport. Vulnerability is a tremendous, frequently insurmountable, handicap; touchiness is a definite obstacle. On the other hand, patient or physician or both may possess that sensitivity which finds expres-

sion in empathy and symphoria.* The physician's ability, and through him the patient's ability, to recognize and understand their sensitivities, to offset as much as possible vulnerability and touchiness by empathy and symphoria, may foster a cooperative atmosphere and activity, essentials to efficient psychotherapy.

c. *Empathy.*

Any attitude which makes for subtle "understanding" and rapport with another individual by necessity includes the "essence of sensitivity." Empathy signifies the ability of a person to "feel himself into" another person, to recognize more or less quickly and with accuracy his moods, desires and needs, as it were, from within the other person. By empathy thus is meant the immediate and flexible orientation of one individual—the empathetic person—in the experiential constellation of another—the empathized. Such "feeling in" is achieved on the basis of a "considerable degree of emotionally charged impressibility."

Empathy is found as a personality trait in a great variety of personalities; it never is a dominating trait; one could not construct an "empathetic personality type." Empathy is under the control of the individual who can, so to speak, switch it on and off at will; empathy consequently is used as a tool—in many cases a perfectly conscious and planned effort. The empathetic attitude is actually one of curiosity. The empathetic person wonders what is going on in the other person. While he is able to find this out, he is not interested *in doing* anything for the other person. He satisfies his curiosity, which may or may not be a scientific one, and then either stores his newly gained knowledge away, or exploits the "collected material" for his own purposes which need not be utterly egotistic. This point should be stressed: the empathetic person enriches himself and his experiences regardless of any gain or loss for the empathized individual. One observes that this is a peculiarly one-sided relationship between two per-

* We have chosen the word symphoria, derived from symphorein (to bear together), because it conveys the notion of participating activity. Sympathy and symphoria are akin as they both imply participation. Sympathy, however, lacks the notion of activity and furthermore carries with it other connotations.

sons. Beyond the immediate interpersonal relationship, the empathetic person may draw upon his insight to express it in a work of art or in scientific writing. Here he gives and he may give very freely whereas in the interpersonal relationship where he practices his empathy he is only at the receiving end.

This peculiar one-sidedness of the empathetic attitude makes it understandable that a few of the aloof, withdrawing vulnerables are, as we mentioned, capable of creating works of cultural value in their ivory towers. They—by no means all the vulnerables—are able to feel into their fellowmen, to look into the emotional life of others without letting others see their own.

Among the many who "have" empathy and use it skillfully are certain hysterical personalities who are virtuosos in feeling into a person; exploiting their empathy to display the whole gamut of emotional expression. Some so-called hysterical swindlers belong here, probably also a few charlatans and perhaps an occasional medicine man or shaman.

No matter in which kind of person and in what setting empathy appears, it is definitely advantageous to the person possessed of it. It gives understanding of other people "just for the asking"; it is a mechanism that can be switched on and off *ad lib*. The empathetic person is aware of this advantage and derives from it a feeling of superiority and security. In numerous ways—as artist, as actor, as writer, as advertiser, as scientist, as public speaker, etc.—he brings his empathetically gained experiences home to the group which, pleased or displeased, cannot fail to appraise his empathy as an asset: here, after all, is a person who knows the ways of men and women and can tell us something about them.

Though highly impressible for the emotions of the other person, the empathetic does not—this in contrast to the symphoric—actually participate in them. He remains emotionally on his own, cool and detached—able to evaluate, to verbalize, to formulate. This from his own and from the group's point of view is just one more asset. It is, of course, the very basis of the particular one-sidedness in the empathetic contact: the empathetic person is contacting the other person but is not contacted by him. Association with the empathetic person may lead some to believe that they are participating in a profound and very effective mutual

contact, but this only shows the ready willingness of human beings to be fooled and to fool themselves. We all believe what we desire to believe.

A queer kinship seems to exist between the vulnerable who withdraws from contact yet craves it, and the empathetic who contacts without participation. Both are, as it were, theoreticians of human contact.

d. *Symphoria.*

The empathetic individual is contacting but not contacted; he is impressible for, but does not participate in, the emotions of another individual. In contrast: the symphoric individual is contacting and contacted; he participates in the emotions of the other, the symphorized individual.

Empathy signifies exploring, reconnoitering, contemplating, estimating, judging, "feeling into," but not "feeling with" the other person. Symphoria implies "feeling into" and "feeling and swing-with" the other person.

We cannot conceive of a symphoric type of personality but there are individuals, if not very many, in whom symphoria appears to be the most outstanding, we may say, the dominating personality trait. Indeed, the symphoric experience in its depth and breadth and the manifestations of symphoria depend largely upon the configuration of the whole personality; intelligence and expressive capacity play a remarkable rôle here. In an unintelligent person with poor verbalization and vocabulary an expression of symphoria may be restricted to a hearty handshake or a friendly glance—at times perhaps feigning sentimentality where actually symphoria is displayed.

In a few persons symphoria comes to very powerful expression. The symphoric are natural, their responses and expressions are genuine, unstudied and unaffected. They have no need for theatricals. This further increases their efficiency.

Whereas a vulnerable may unsuccessfully ponder about what he might do for his fellowmen, the symphoric by his very presence is a comfort and uplift. We are discussing the symphoric sensitive, not members of the hyperthymic-hypomanic group who waste their words and actions; the symphoric calms and satisfies with a glance, a gesture, a few words.

There is a similarity between vulnerability and symphoria as both varieties of sensitivity have a tendency to appear as dominating personality traits and both seem to be more highly emotionally charged than either touchiness or empathy. The most striking difference is noted in the aloofness of the vulnerable and the participation of the symphoric.

Emotional participation and communication are characteristic of the symphoric attitude. Wherever there is genuine contact, sympathy, participation and communication between human individuals, symphoria must be present—even if in some dilution. Symphoria is, so to say, the climax of a "considerable degree of emotionally charged impressibility" in the medium of human warmth.

These last remarks implicate the attitude of the group towards the symphoric; it is, indeed, in our western civilization, outspokenly positive. Symphoria ranks high on the scale of social values; we find it labeled human kindness, warmth, charity, benevolence, and so forth. Occasionally it is ridiculed. Its importance in the family is paramount. It is instrumental in the formation of groups except the militaristic group. The symphoric person wanted and welcomed, useful and helpful in most if not all situations cannot but experience his symphoria as an asset. There are not many chances for feelings of inferiority and insecurity in his outlook.

We have described the four varieties of sensitivity as if they were isolated phenomena. There may actually be considerable overlapping of them in any one person but usually one variety stands out most. Some vulnerables no doubt have a certain capacity to feel into and with others; but if vulnerability is the dominating trait, symphoria will find little if any expression. Touchiness is ubiquitous, hence also found among the vulnerable, the empathetic and the symphoric. That vulnerables can be empathetic has been mentioned; they may use empathy as a tool to "dissect the enemy" or to guard their own schemata and systems. That the symphoric person is implicitly empathetic we have discussed; some empathetics may tend toward experiencing certain people in a symphoric manner.

Individuals develop, so do their qualities and traits.

Sensitivity, in all of its varieties, may undergo noticeable changes as a person progresses through life's course. A dominating variety will remain essentially dominating throughout life. One gathers that vulnerability tends to increase towards early middle life and from then on changes little. Touchiness shows a predilection for the span of adolescence, but is well enough known earlier and later. Empathy and symphoria need the background of a somewhat mature person; prior to early middle age they are rarely completely developed; both are liable to involution with the approach of senium. Aging itself not infrequently is a touchy spot. Disease, any kind of disease, may for a time stress and overstress sensitive attitudes.

All four varieties of sensitivity are found in males and females. There may be a certain prejudice in associating the vulnerable more with the younger male and symphoria with the mature women, with the mother image. Episodic fluctuations, particularly of touchiness, but also of vulnerability occur during menstruation, pregnancy and the menopause.

Conclusion and Summary.

We have discussed four varieties of sensitivity which have the common denominator of a "considerable degree of emotionally charged impressibility." This we consider the essence of sensitivity. Sensitivity is experienced in different ways by the individual and by the group. Vulnerability is a dominating personality trait; the pattern of the vulnerable is withdrawal. The social group is disinterested in such non-participants. Touchiness is a spotted sort of sensitivity; it is manifested in episodic aggressiveness. The touchy is apt to project his hurt feelings. Although it is not a dominating personality trait, it is so common and familiar that the group tolerates it well. Empathy is characterized by cool curiosity; it permits the establishment of a one-sided relationship in which the empathetic contacts without being contacted himself. Empathy is socially accepted. Symphoria is sensitivity in the medium of human warmth; the communication and participation of the symphoric with his fellowmen determines the high esteem he enjoys among them.

Emotional impressibility rests on hereditary-constitutional grounds, which does not mean that it can be dealt with as a Mendelian character. It is shaped within the whole makeup of the individual and definitely modified—essentially in its expression—by the social group in which the emotionally impressible person grows and develops. Any variety of sensitivity has a bearing on the individual's attitude towards other people, towards the group. This attitude is fed (given content) by social-cultural forces, by living and experiencing with and in a group. The group may either foster or prohibit overt expression of any of the varieties of sensitivity. It may taboo any kind and number of cultural factors which serve as sources and contents for the expression of emotional impressibility. Hence we have to judge an individual's sensitivity according to the social-cultural background on which he learns to express or to suppress it. The individual somehow realizes how his group expects him to behave. If he does not or cannot conform to the group's expectation, he feels uneasy, insecure, anxious—such feelings are obviously expressed by the vulnerable and the touchy. On the other hand, the empathetic and the symphoric conform, their very sensitivity is helpful in their overt adjustment; they can and do experience it, each on his own terms, as an asset of their personality and as an asset for the group.

THE ATTITUDE OF NEUROLOGISTS, PSYCHIATRISTS AND PSYCHOLOGISTS TOWARDS PSYCHOANALYSIS.*

By ABRAHAM MYERSON, M. D.

Psychoanalysis is one of the oldest systems of concepts and of therapeutics in modern medicine. The beginning of Freud's work in psychoanalysis is, in fact, contemporaneous with the introduction of diphtheria antitoxin. His first paper appeared in 1893 and von Behring's publications culminated in 1893, although they started in 1890. Before von Behring's introduction of diphtheria antitoxin, the diphtheric exudate was treated by a host of methods. With the introduction of antitoxin, the other forms of treatment disappeared, and only one method remained in the field of therapy, the injection of diphtheria antitoxin. Before its superior potency, haphazard empirical guess-work therapeutics suffered the fate that the clearly inferior technique always experiences when the superior one is introduced into the world. Natural selection operated at once, and only one therapeutic measure for the treatment of diphtheria has remained.

In the 45 years that have elapsed since psychoanalysis was born into the family of scientific work, the Wassermann reaction has been introduced (1906). Arsphenamine appeared upon the scene in 1910 as the primary basis for the modern treatment of syphilis. In 1907 Wagner von Jauregg brought into medicine the idea that heat in the form of fever was an important therapeutic tool. In 1922 the young Canadian, Banting, with his co-workers added to the armamentarium of medicine insulin to combat diabetes. Thirty-three years after Freud started his reign as the head of psychoanalysis a group of American scientists, Minot, Murphy and Whipple, completely changed the fate of people suffering from pernicious anemia by introducing liver extract as its main therapeutic avenue. In 1913 Schick published his first paper on the tech-

* From the Division of Psychiatric Research, Boston State Hospital, Boston, Massachusetts, aided by grants from the Commonwealth of Massachusetts and the Rockefeller Foundation.

nique which bears his name, and thus started a campaign for the eradication of diphtheria, the value of which cannot be overestimated.

The skeptical, hard-boiled medical profession has completely accepted all these advances in diagnosis and therapy and without any real struggle. The general law of life has operated, namely, wherever a method has shown superior therapeutic power, it has won the day, just as in the field of transportation the horse and buggy gave way to the automobile, and the mechanical methods of heating and cooling won over more primitive techniques. Results count, and where the results are clear-cut, the technique which brings them wins the day.

The psychoanalysts, when this is brought to their attention, have a ready answer which is primarily the same one that they use when a patient fails to get well and which is the most beautiful instrument for successful argumentation ever devised by man, namely, the medical profession has *resistance* based on prejudice, unconscious fear and anxiety, and a general distaste and dislike for the grim and remorseless truths initiated by analysis. True, there are many people to whom the sexual factors and unconscious basis of psychoanalysis are offensive in a prejudiced unthinking way. But after 40 years of social inoculation and when generations of medical men have grown up with active psychoanalysts writing, talking and working, the professional prejudice would have been broken down if results were overwhelmingly clear and valuable.

The present study did *not* start as an effort to answer the question, "How far do those people who might be assumed to know the field of nervous and mental diseases and who can be listed in a wider sense as professional students of the mind and its diseases accept psychoanalysis?" It started as a reaction against the very extreme statement of a psychoanalyst to the effect "that practically all informed scientists accept psychoanalysis." To see whether or not this was the case, a questionnaire was drawn up, which at first was sent rather haphazardly to many entitled to be called leaders in psychology, neurology and psychiatry. As the answers became interesting, the scope of the inquiry was extended, so that finally four questions comprising the questionnaire were sent to a group of leading psychologists connected with universities, to the officers and every third member of the American Neurological Association, to the officers and every tenth member

of The American Psychiatric Association, and more or less haphazardly a group of psychoanalysts were canvassed on the assumption that their very membership in the psychoanalytic society necessitated only a small sample. Nevertheless, the proportion of psychoanalysts who were questioned was larger than the proportion questioned of members of The American Psychiatric Association, and in fact many of them had been questioned as members of the American Neurological and American Psychiatric Associations.

Beyond any doubt, the questions which were submitted are imperfect and do not permit of any complete answer. Many people pointed out the difficulties in their mind as they attempted to answer the questionnaire, and I have no hesitation in saying that the questionnaire could be improved and the whole technique of canvassing made more scientific. However, I have no intention of presenting a completely scientific answer to the question, "In how far has psychoanalysis been accepted and in how far do psychologists, neurologists and psychiatrists believe that important therapeutic results have been contributed by this technique," since this is impossible. Despite objections, most of the writers had no great difficulty in classifying themselves somewheres in the four categories or else placing themselves between one category and the succeeding one, and the results are interesting and important.

The first questionnaire sent out asked the recipient to classify himself in one of the following groups:

1. Those individuals who completely accept psychoanalysis.
2. Those who feel very favorably inclined towards it but do not wholly accept it and are, to a certain extent, skeptical.
3. Those who, in the main, tend to reject its tenets but feel that Freud has contributed indirectly to the human understanding.
4. Those who feel that his work has, on the whole, hindered the progress of the understanding of the mental diseases and the neuroses and reject him entirely.

The second questionnaire was modified somewhat and the recipients were asked to classify themselves as follows:

1. Those individuals who completely accept psychoanalysis. The term "completely" need not be used too literally. One may substitute "wholeheartedly" or "in general."
2. As above.
3. As above.
4. As above.

The most serious objections came from psychoanalysts who protested that nobody could accept completely a technique or point of view. Nevertheless nearly all of them finally registered themselves in Group 1, to the effect that they did completely accept psychoanalysis, by which I infer that certainly they may have points of difference with Freud and the Freudian doctrine, but that on the whole they accept the leading tenets and are optimistic about the therapeutic results. Similarly, people who were on the whole not friendly to the psychoanalytic movement expressed themselves as balking at a complete rejection and insisted that they belonged between the attitudes expressed by Groups 3 and 4. Still others found the second attitude difficult to accept, and some asked me to leave out the word "very" from the phrase "very favorably."

The *first* position may be considered as the extreme of acceptance and friendliness, as well as of optimism, concerning therapeutic results, although this is not directly questioned. The *second* position represents friendliness, but is sufficiently divergent from acceptance to embrace the individual who is not eligible to membership in the American Psychoanalytic Association. It is the position of a man who has been profoundly influenced by the psychoanalytic movement, accepts a great many of its terms, believes in the unconscious, and usually agrees that a longer or shorter process of self-revelation on the part of the patient is desirable, but who is an eclectic in the treatment of the neuroses. The *third* position is one of rejection but not of marked unfriendliness; such people do not accept the psychoanalytic theories or use its technique. They believe in psychotherapeutics of other types and follow physiologic measures of treatment as well, but they acknowledge the value of Freud historically and indirectly. The *fourth* position represents a completely hostile attitude. Not only is it implied that the psychoanalytic procedures and doctrines are invalid, but that the movement as a whole has done harm to psychiatry and to the research by which the general understanding of mental states, both normal and pathologic, is furthered. It is obvious, of course, that there are intermediate positions, as will be seen. These were assumed by many of those questioned.

Results.

Of the 428 questionnaires sent, 307 individuals replied, which on the whole is a good percentage. Only inference can be made as to why the others did not reply. Some explanation of this reticence appears in answers made to a later questionnaire in which the individuals were asked to answer as follows:

1. I am willing to be quoted by name in respect to my attitude on psychoanalysis as evidenced in my reply to the questionnaire.
2. I am willing to be quoted but not by name or identification.
3. I do not wish to be quoted at all.

Of these, a considerable number did not want their names given or to be identified, while others stated they did not wish to be quoted at all. Obviously, there was to some extent a distaste for or fear of the controversy implied in the whole procedure, so that it appears at once that a great deal of emotion is involved in any study of the psychoanalytic movement in a way which would undoubtedly not be found in a similar study, for example, which would involve the treatment of syphilis by arsphenamine or by fever therapy. This is confirmed by the fact that those who did not wish to be quoted belong mainly in the groups which, though friendly, do not completely accept and those which reject but are not unfriendly. The usual reasons why questionnaires are not answered probably also operated, because, first, there are persons who do not believe in questionnaires, and secondly, there are those who forget to answer.

Position of Members of the American Neurological Association.

Of the 97 members of the American Neurological Association who received questionnaires, 75 replied. These may be classed as follows: five belong in Group 1; eight classify themselves between 1 and 2; 23 stated they belong in Group 2; four classify themselves between Groups 2 and 3; 25 declared themselves to be in Group 3; three stated that between Groups 3 and 4 was a good enough allocation of their position; four totally rejected psychoanalysis and were hostile; three were definitely non-committal or equivocal.

In general (Chart 1), the American Neurological group shows a beautiful curve of replies, about an equal and small number who accept and completely reject; an equal number who are friendly and to a certain extent are linked up with the psychoanalytic movement as compared to those who are not unfriendly, but who reject psychoanalysis, conceding that Freud has contributed to the learning and procedures of psychiatry.

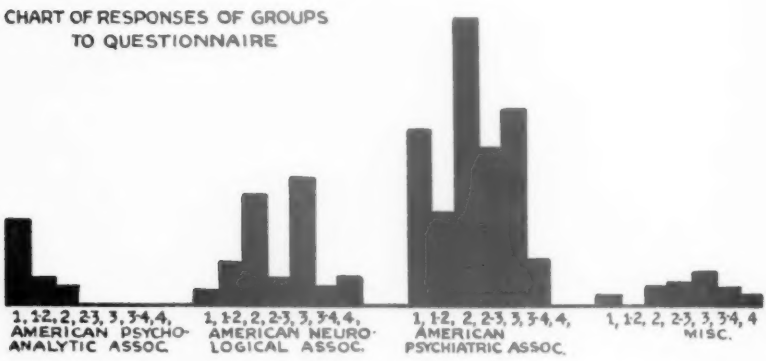


CHART 1.

POSITION OF MEMBERS OF THE AMERICAN PSYCHIATRIC ASSOCIATION.

Of the 266 members of The American Psychiatric Association canvassed on the basis stated above, 179 replied. Twenty-five classed themselves in Group 1; 15 between Groups 1 and 2; 54 in Group 2; 32 between Groups 2 and 3; 39 in Group 3 rejecting; 8 belong between this group and total rejection; while there was none who completely rejected psychoanalysis as having had some value. Six were non-committal.

It will be seen that the members of The American Psychiatric Association accept psychoanalysis in greater degree than do the members of the American Neurological Association; that if we place Group 2 as the middle of the series of positions, the American Psychiatric as judged by these replies is about equally divided, there being 94 on the friendly and more or less acceptance side of the fence and 79 on the rejecting side of the barrier.

Position of Members of the American Psychoanalytic Association.

Of the members of the American Psychoanalytic Association, 28 replies were received to 36 questionnaires sent. Of this group, 16 were in Group 1; 4 between Groups 1 and 2; 3 in Group 2; and 5 were skeptical or non-committal.

As would be expected, this group is largely on the acceptance side, although without too great a slavish adherence to psychoanalysis. The most heated letters sent me came from individuals in this group. Some took me to task; some took the position of instruction in the art of questionnairing; and others even felt impelled to lay down the elementary laws of scientific thought and procedure.

Miscellaneous Group.

Of a miscellaneous group, made up mainly of important members of the American Psychological Association and some physiologists interested in psychological research, 25 replies were received to 29 questionnaires sent. Two accepted psychoanalysis; 5 belonged in Group 2; 6 placed themselves between Groups 2 and 3; 7 placed themselves in Group 3; and 3 between Groups 3 and 4; 2 completely rejected and placed themselves in Group 4.

Thus, in this group of eminent men, having an almost 100 per cent inclusion in Who's Who in Science and in America, the general attitude is more on the rejection side than on the acceptance, and goes a little further in the direction indicated by the position of the American Neurological Association.

Citations.

Many exceedingly interesting letters were received in answer to the questionnaire. Space does not permit the citation of all of these. I cite, therefore, a few from each group of those who have permitted citation.

GROUP 1.

Those who placed themselves in Group 1 in my classification ranged in their answers from the statement made by Isador Coriat, "In reply to your inquiry, I place myself in category number one,

namely, those individuals who completely accept psychoanalysis" to those who are less terse and more cautious.

For example, Lucile Dooley states, "I am in class 1, as completely accepting psychoanalysis, if by psychoanalysis is meant a valid *method* of investigating the human personality. I do not accept all its present formulations as final, nor, I believe, does its founder do this. Additions, revisions and corrections are hoped for and earnestly sought. As a valuable instrument for research, and in some cases for therapy, I accept it. Further, I believe that the method is not entirely replaceable by any other as yet discovered method. And for the one who would use it a personal analysis is as indispensable as is a high powered microscope for the bacteriologist." Her letter which ends by thanking me "for the opportunity of responding to the questionnaire and thus aiding in a valuable project" may be placed side by side with the letter of Smith Ely Jelliffe who writes as follows:

"By experience and study I would place myself in Group 1—just as if you asked do I believe 'completely in the concept of evolution.' 'Completely' is an intangible phrase for any growing body of experimental science, such as 'psychoanalysis,' or 'evolution' or similar large aspects of operational concepts. Questionnaires of this type do not mean much to me. I think they are usually very stupid. But *'chacun a son gout,'* as the old lady said when she kissed the cow."

GROUP BETWEEN 1 AND 2.

Of those placing themselves between Groups 1 and 2, I cite Lawrence Kubie's letter as summarized by me and approved by him. Dr. Kubie, after a preliminary discussion as to the validity of the questionnaire, points out as follows: "For instance, I personally cannot accept either of your first two categories, although in a vague way they come nearer to my point of view than the other two. The first category is too blindly sectarian; the second one vague, muddled, and chicken-hearted in its ambivalence. Therefore, I think you need at least three more categories, to wit, (1) those who believe that the psychoanalytic technique, properly employed, uncovers significant dynamic psychological facts not obtainable in any other way at present; and (2) those who believe

that the psychoanalytic technique, judiciously employed, can in certain cases secure therapeutic results not obtainable in any other way at present; and (3) those who believe that the basic principles of psychoanalytic theory are sound, however much they may disagree with any details of theory. (These basic principles could easily be formulated for your questionnaire to lessen still further its ambiguity.) I will classify myself under all three of these categories. I cannot, with any justice to my own convictions, classify myself under any of the four categories which your questionnaire would impose."

I have taken the liberty, however, of classifying Dr. Kubie between Groups 1 and 2. His characterization of the second point of view classifies a good many men of The American Psychiatric Association as "vague, muddled and chicken-hearted" in their reaction to psychoanalysis.

I cite Louis Casamajor: "I do not particularly like the four groups you make of people who react towards psychoanalysis. I feel that they are not near enough. Personally I stand somewhere between one and two; probably closer to one than to two. While I doubt if I 'completely accept psychoanalysis' yet I am sure that my feelings toward it are much more than 'favorably inclined.' If you should have to put me in either one group or the other I would prefer to be in group one."

William Malamud, who is in favor of the research, states, "It is somewhat difficult to fit my own reactions into any one of the four groups that you mention, but I would feel inclined to place myself somewhere between groups one and two. By this I mean that my attitude is that psychoanalysis has contributed a great deal of knowledge to the fields of psychopathology and psychotherapy. At the same time there are certain features both in the theoretical concepts and in some of the practical technique that leave me skeptical or appear to me superfluous."

Tracy J. Putnam writes as follows, "I feel that none of your categories exactly fits my attitude toward psychoanalysis, which might fall between 1 and 2. I certainly do not agree with all that Freud has to say and feel that some of his followers are of a low order. On the other hand, I do feel that Freud has made a tremendous and theoretical and practical advance in psychology and psychiatry. It seems to me that one can well compare psychoanalysis

to neurological surgery. It has some brilliant successes to its credit, some gallant failures, some egregious mistakes and some charlatanism, but even the mistakes have contributed information, and it is inconceivable that we can ever do without it in the future."

GROUP 2.

Of the men in this group, Karl M. Bowman answers as follows, "It is not easy to classify myself in the manner you have suggested, as I am perhaps somewhat ambivalent. I would say that I feel Freud has made many important and valuable contributions to psychoanalysis, and that there are many of his formulations which I would essentially accept. On the other hand, there are many of his other formulations with which I definitely disagree. Again, if the term is used as psychoanalysis, one might have many objections to the attitudes and opinions of a large number of the psychoanalysts of this country, without that being particularly a reflection on Freud himself. I would think that I fit most nearly under your second group, but I do not feel that this is quite an exact statement of my attitude. Number three would seem to me to give less credit to Freud than I would feel inclined to give."

A very cogent statement is that of Clarence O. Cheney, "I feel that it (psychoanalysis) has contributed a good deal to psychiatry but I am not by any means willing to accept all of the theories or conclusions that have been formulated by Freud and some of his disciples who, I feel, are inclined to reason from the particular to the general and oftentimes seem not to be able to use common sense. I regret also that not a few of them seem to be interested only in the psyche of the patient and not in the patient himself and in his living in and adjusting to a community. I think it does not help psychoanalysts either to see so many persons who are analysts unable to adjust themselves in their lives very well and who, in fact, at times seem infantile. I grant, of course, that psychoanalysis in itself should not be blamed for this. I feel that there may be many persons, including social workers and other professional groups, who have become instilled with the idea that psychoanalysis is necessary whereas they are using time that might be more profitably spent."

Stanley Cobb expresses the following point of view, "I should think on the list of attitudes toward psychoanalysis you could put

me down as a number two. In fact, in almost anything that you ask me about, I think I would be a number two, because anybody who accepts anything whole is sure to be wrong. Therefore, I strive to remain a skeptic in all fields. I rather wish you had left out the word 'very.' Perhaps your paragraph would then express my attitude more exactly."

A typical attitude is expressed by Eugen Kahn, "Let me say that I seem to belong to your group two with some modification insofar as I feel favorably inclined towards Freudian analysis, cannot wholly accept all of it, and am skeptical to quite some degree."

Nolan D. C. Lewis takes the following position, "I usually think of psychoanalysis in terms of at least three of its aspects—first, a good deal of it exists as a body of doctrine, metapsychology or philosophy, if you will; second, another aspect is the application of the technique as a research method which can be used for the purpose of deeper dissection of mental content; and third, its psychotherapeutic possibilities. I still think there are certain types of cases that respond better to psychoanalysis if well done, by an expert, than to any other variety of psychotherapy. In other words, in some cases, it is the most valuable approach. I have never thought of psychoanalysis in terms of any possibility of its replacing psychiatry or that the terms psychoanalysis and psychiatry are in any way synonymous. It is only a part of the body of knowledge included in psychiatry, but I do believe that the method and findings justify additional clinical researches and evaluations."

While William L. Russell thinks he fits best in Group 2, he makes the following important statement in regard to those who should practise psychoanalysis, "I believe, however, that I should say that on the other side I believe that only those who are well qualified in general psychiatry should undertake to practise psychoanalysis. I am under the impression also that where psychoanalysis dominates in a psychiatric organization the young physicians entering the service do not get a proper foundation for a sound development in psychiatry. They become imbued with the idea that the main task is to work out a theoretical formulation on psychoanalytic lines of the problems presented by the patient. There is a tendency to neglect the more obvious factors in the understanding and the treatment of the patient. There is also a tendency to neglect the physical. In fact, Freud has written that a person does not

need medical training to practise psychoanalysis successfully. This view is, of course, far from what we have been teaching and practising in psychiatry as a branch of medicine."

Israel Strauss feels favorably inclined towards psychoanalysis, but adds this sting to the tail of his remarks, "but more skeptical of some of those who practise it."

GROUP BETWEEN 2 AND 3.

In this group particularly, a large number of individuals did not wish to be quoted either by name or at all.

One important psychiatrist of the elderly group, who does not wish to be quoted by name, states, "Indeed I am quite sure that I stand somewhere between the 2nd and 3rd (group). I certainly can credit psychoanalysis with more than those in your third group. Probably my chief difficulty lies in its direct therapeutic use, particularly in the practice of psychiatry. As far as I am able to observe, while it gives one an insight into mechanisms, it signally fails to produce beneficial results."

GROUP 3.

Of this group, Gordon Holmes states, "I think you can place me in the third group, that is, I believe Freud has contributed to our understanding of the human mind, although I have never been convinced that many of his tenets are acceptable. I am also of the opinion that except in a small proportion of cases psychoanalysis has failed as a line of treatment, and that in the hands of uncritical enthusiasts and of those without adequate experience of clinical work it has often produced more harm than good."

Selecting Group 3 in preference to the others, Samuel D. Ingham states, "I think it is probably true that my own conception of the motivation of human behavior may on the whole be better than it would have been if Freud had not written the Scenario of Psychoanalysis. I do feel that he has made important contributions to the understanding of human nature. I also feel that most of his would-be followers have been woefully misled. I am inclined, too, to think that enthusiasm in regard to a psychoanalytic viewpoint has tended to inhibit progress in psychology on the basis of a more strictly biological approach."

Andrew H. Woods' letter speaks for itself, "For your own interest I should say that the word psychoanalysis ought not be spelled with a capital P as if it were a new denomination or school of medicine. Taking all the florid literature produced by Freud, but added to enormously by his followers, perhaps it has to be considered as a new school of thought. I look upon the technique for re-discovering experiences that have emotional value, that is the technique of guided memory associations, as extremely valuable. It is, of course, nothing new, but has been organized into a much more useful form under Freud's stimulation. The careful study of dream symbolism and the symbolism of psychoneurosis appears to me another contribution of great value. The idea of catharsis is to me nothing more than confession and has always been useful. I feel sure that many of the so-called cures through psychoanalysis are nothing more than relief gained through Christian Science and other dramatic performances by self-confident operators."

GROUP BETWEEN 3 AND 4.

The following is an abstract of the letter of a very well known neurologist, who does not wish to be quoted by name, "I would say, offhand, that less than five per cent of the patients in my office were cases where the major causation of the condition was such as to include them in the Freudian group. By this I mean that the other ninety-five per cent of the cases were amenable to other forms of therapy, and the therapeutic results were to be obtained in a much shorter period of time and with more lasting effect. Of the five per cent of the cases, these were treated, and very successfully, with the Freudian technique. . . . . In reply to question four, I think that psychoanalysis has been the greatest block to the study and understanding of mental disease, and by this I mean insanity, that has happened since the time of Rush. All doctors in all institutions for the care of the insane that I have been in touch with in the United States were so saturated with the Freudian concept that real investigation of mental diseases was almost entirely excluded. There was some little work here and there but in an entirely unsympathetic atmosphere. . . . ." He ends up by stating, "From the standpoint of the professional psychoanalyst, I, not having been psychoanalyzed, have no right to the above opinions, and they are, therefore, of little or no value."

Since the above is a typical answer, I am not quoting further from the group standing between 3 and 4.

GROUP 4.

The outstanding personality in this group is Bernard Sachs. He underlines Group 4, to wit, he is of those who feel that Freud's work has, on the whole, hindered the progress of the understanding of the mental diseases and the neuroses and *reject him entirely*. In a footnote he states, "Strongly for No. 4 with a little concession as to the introduction of a few useful concepts." Thus, he is willing to admit that Freud has indirectly and to a limited extent enriched human knowledge.

AUTHOR'S PERSONAL OPINION.

I take it that the instigator of this questionnaire has a right to express himself more fully on the matter than any of his subjects, so that his "cultural compulsives," which is a polite phrase for prejudices, may directly appear. I also find it somewhat difficult to confine myself to the categories of classification. Roughly, I can state that I belong mainly in Group 3 with a flow towards Group 2 and an equal flow towards Group 4.

I am definitely of the opinion that Freud is a great man and entitled to be called one of the great men of his times. Greatness, I believe, does not reside in the truth or non-truth of what the great man believes, since the truth is an inaccessible ideal of human striving. The effect of Freud on his generation has been enormous and it is probably impossible for any contemporary to say whether that has been good or non-good, since these are viewed from the standpoint of the individual's trends, desires and wishes, being in the main subjective.

The concrete values of Freud's work seem to me to be as follows, and they are, in the main, indirect results of his work:

(1) Freud has been one of the men who has taken the obscenity out of sex and made it possible to study its phenomena objectively. While he has had predecessors and co-workers, such as Krafft-Ebing, Moll, Havelock Ellis and others, no one has been so ruthless as he in the approach to the matter. Homosexuality, autoeroticism, and the various manifestations of heterosexuality have been em-

phasized as authentic and competing directions of human sexuality and have been brought fully to the social consciousness by his work and, to a lesser extent, by his followers.

(2) He has emphasized struggle within the personality more emphatically and more candidly than any predecessor or contemporary. Inner struggle as such has been well recognized by the theologians, the philosophers, the psychiatrists, and is deeply imbedded in the consciousness of the average man. Whether his analysis of the struggle is the correct one or not, even those who reject his point of view have had to pay more attention to the details of human life and to the *concealed* human difficulties because of the pressure of his ideas and his work. Whether we agree or not as to the facts of his discovery, research into the human mind has been stimulated by his activities. In my opinion, this is an indirect result since I do not agree with the findings. All in all, therefore, this bold voyager into the realm of human attitudes and mental diseases has exercised a great influence directly useful in the opinion of his followers, indirectly of great value in the opinion of a humble psychiatrist who, nevertheless, rejects the authenticity of the phenomena described.

This rejection includes Freud's concept of the consciousness, of the censor, and of the sharp separation of unconscious processes from the conscious activities of the individual. I do not believe the unconscious is an organized personality or is a place where complexes, forgotten experiences, so to speak, roam around looking for chances to express themselves, by eluding the vigilant censor or the preconscious in neuroses, dreams, slips of the tongue or any of these phenomena. To me, the unconscious is the sum total of those drives, instincts and activities which the viscera would naturally bring into action. The social structure, through the forebrain, tries to limit by injunctions of morality, fitness and decorum the activities, let us say, of the male genitalia; but these organs and their linked-up glands and hormones do not often easily submit to culture, morality, decorum and fitness. The forbidden activities are called crime. The struggle between the visceral drives and the forebrain and society, which may be concealed or diverted, can easily be brought into consciousness and, in fact, is a component of the consciousness, whether acknowledged or not.

(3) I reject entirely the so-called free association technique. I submit that you can take ten words of a time-table and get at any hidden struggle of the individual and reach as many mental situations and complexes as you can by the words of the dream. *In other words, the patent content of the dream has nothing to do with what is called its latent content or to the matter which is disclosed.*

Moreover, the associations *are conditioned* by the examination and study itself. A beautiful young psychoanalyst of the female sex would arouse other associations than the austere Freud would or could. The very fact that psychoanalytic doctrines have infiltrated the thinking of the community and the fact that the patient seeks a psychoanalyst alters the associations in a very significant manner.

(4) The doctrine of infantile sexuality is completely against the facts of patent type. There are no sexual acts corresponding to the postulated sexual attitudes. Moreover, *the infant does not have hormones in his urine or has so little as to be almost negligible. Hormones appear in puberty in a great flood corresponding to the overt behavior.* Throughout the writings of Freud and his followers, *sensuality* is continually identified with *sexuality,* as in the classic statement made by Freud, that when one sees the infant after nursing at the breast, relaxing with satisfaction, it brings to mind and apparently is identical with the relaxed lover after a sexual relationship.

(5) The whole concept of symbolism seems to me an exercise in ingenuity and without the slightest proof. Straight things, round things, enclosed things—all these are too common, too universal, in fact, to be symbols of the phallus, the lingham, the vagina, and any other part, male or female. Vigorous objects, like bulls and horses, can be symbols for everything under the sun, as well as the father. It is an arbitrary selection which makes them symbols in the Freudian sense and gives them the value accredited them by psychoanalysis.

The psychoanalysts have a very ingenious subterfuge for escaping criticism. So long as you have not been psychoanalyzed, you cannot judge the results of psychoanalysis. But I am not a surgeon and yet I can judge the results of surgery. I can tell when my patient has had a successful operation for brain tumor, even though

I could not bore a hole straight in the skull. I can even judge when a chemical problem has been attacked properly, although I am no chemist. The general criteria of science can be utilized by a non-psychoanalyst with validity in judging both the analytic idealogy and its results.

And when one sees the analysts passing judgment on the quick and the dead with as complete assurance in the one case as in the other, one must come to the conclusion that a clinical psychoanalysis is largely superfluous. If one can speak as freely and as authoritatively of Leonardo da Vinci and of Moses as of a little four-year-old boy subjected to an analysis, then surely it is not necessary to do an analysis at all in the technical sense. By extending themselves too far, the psychoanalysts have proven quite clearly that their clinical technique is unnecessary.

When one runs across such biological absurdities as that the child is the symbol of the lost penis, and then sees mother love operating with vigor throughout the whole animal scale and realizes that the lioness probably has no particular complexes due to the operation of the super-ego, one can only reject the interpretation of human mother love as given by Freud and his followers. . . . . When one reads in Freud's "Discontents of Civilization" that woman has become the guardian of the hearth-fire because she is anatomically so constituted that she cannot put out the fire with a stream of urine, one wonders why there has been any acceptance of such doctrines.

And so on, and so on, and so on!

An omission of serious type is conspicuous throughout the work of Freud and his followers. The *present moment* and the *present situation* are entirely neglected as practically having no etiologic bearing. So far as Freud and his followers are concerned, all causation is entirely a matter of the very earliest period of life. The rest is of little significance. Moreover, one would never know from Freud that patients live in an economic world, have a struggle for existence; that the society in which they live is clumsily adapted to their individual needs and, in fact, often maladapted to the human being and his mental health. The long lag between sexual maturity and the legitimate and proper satisfaction of the sexual impulses would seem to me of huge importance, and the other strains of mankind are given, practically speaking, no weight or importance

by him. . . . . The arduous preparation for life which we call education and which is often a crucifixion of all the natural desires of the child has no weight so far as psychoanalysis is concerned. . . . . The struggle to develop constant purposes in an organism which is built up around shifting polarities of expression and which is poorly designed for the coordinated life of civilization apparently has no importance. . . . . The strain of the competitive impulses and their effect on ego-evaluation, and this independently of any sexual relationship whatsoever, receives a bow of acknowledgement but nothing more. In a word, the important strains of existence, the discrepancies between the biologic nature of man and the social nature, and that independently of any unconsciousness, or sex, or what you will of complexes, is passed over almost disdainfully.

Psychoanalysis is reactionary. This sounds like "tu quoque" in a world where the psychoanalysts accuse the non-believers of just exactly this. Essentially analysis harks back to the ancient separation of mind and body, even though analysts continually give lip homage to the relationship of mind and body. But this is only lip homage. Practically, it is considered bad form and poor psychoanalysis if the patient with a neurosis under analysis gets sedatives for sleep, tonics for his appetite, exercises and physiotherapy to increase his muscular endurance and general sense of well-being. I do not know whether there is any ex cathedra prohibition of the care of the whole individual which is so important a part of present day medical belief, but in practice the psychoanalyst constantly operates as if the patient's complexes existed in a sort of physiological vacuum; as if somehow the analysis would be injured, if side by side with it the hygiene of the individual was changed so as to improve his general health, and if the well established procedures by which the body is made more vigorous would contaminate the purity of the psychoanalytic procedures. This is true reactionism. It is against the attitude of modern scientific thought, which states that the separation of mind and body is an artifact harking back to primitive and theological thought.

I state definitely that as a therapeutic system, psychoanalysis has failed to prove its worth. First of all, it has not conquered the field as is the case with any other successful therapeutic approach, as I have indicated in the first part of this paper. There is more

reason to extol in the case of the psychoses the pharmacological measures and the physiological stimulations than psychoanalysis. The neuroses are "cured" by Christian Science, osteopathy, chiropractice, nux vomica and bromides, benzedrine sulfate, change of scene, a blow on the head, and psychoanalysis, which probably means that none of these has yet established its real worth in the matter, and surely that psychoanalysis is no specific. Moreover, since many neuroses are self-limited, anyone who spends two years with a patient gets credit for the operation of nature.

NITROGEN INHALATION THERAPY FOR SCHIZOPHRENIA.

Preliminary Report on Technique.*

By F. A. D. ALEXANDER and H. E. HIMWICH,
Albany, N. Y.

This investigation is the result of the cooperative effort of a large group. Dr. Basile Lipetz is the psychiatrist in charge of the patients and has followed their progress throughout the treatments. To Dr. S. J. Martin we are indebted for the electrocardiographic studies. We would also like to thank Mr. Joseph Fazekas and Drs. Harvey Slocum, Hubert Doudna, Benjamen Etsten and Philip Gleason for their valuable contributions in this investigation.

Rationale and Advantages.

Newer treatments for schizophrenia have attracted much interest and attention during the past few years. Two methods of so-called "shock treatment," insulin [1] and metrazol [2] have proved to be of therapeutic value. Studies of the physiological changes occurring during these treatments have revealed that in both cases there results a marked decrease in cerebral metabolism. The diminished cerebral oxygen consumption,[3] decrease in frequency of cortical rhythm,[4, 5] and the appearance of neurological disturbances are all indicators of a depressed cerebral metabolism.[6]

Despite the fact that both insulin and metrazol treatments are efficacious, objections to their use have been raised. The insulin treatment is time consuming (five hours per day, five days per week), demands constant nursing and medical care and the number of patients that can be so treated is therefore limited. To ensure the most satisfactory results with insulin the hypoglycemia may be maintained for five hours and this procedure entails the

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risk of producing irreversible changes in the brain.[7] Further, a certain percentage of schizophrenic patients do not respond favorably to insulin shock therapy.

The value of metrazol has been seriously curtailed by the high incidence of fractures and dislocations resulting from the severe convulsions.[8, 9] The patients' fear of this form of treatment also lessens its therapeutic value. Metrazol has the added disadvantage of being a "fixed dosage drug" whose effects cannot be terminated at will but must run their course after injection.

If the beneficial effects of metrazol and insulin therapy are dependent upon diminished cerebral metabolism a method of producing such diminution, which could avoid the objections mentioned above, would seem a preferable procedure. To produce such an effect we have subjected patients with schizophrenia to short periods of anoxia produced by the inhalation of nitrogen.[10] By this method the degree of cerebral anoxia can be controlled constantly and the treatment can be terminated instantly if untoward reactions should occur. The severe convulsions characterizing both metrazol and insulin treatments do not appear during the nitrogen therapy. There is no post-treatment confusion and in less than five minutes after cessation of the treatment the patients are able to walk about. No restraints are used. The patients are cooperative and have no fear of the treatment. The average duration of these treatments in our series has been about five minutes. To obtain and record data, we have usually maintained a group of four physicians or housemen to carry out the procedure. Patients have been treated frequently, however, with the attention of only one physician and one nurse. Several of our patients were ambulatory for a part of the period during which they received the therapy.

It is a well-established fact that, as a result of progressive decrease in cerebral metabolism, neurological signs indicating depression and release of the various levels of the central nervous system make their appearance. With anoxia, as with hypoglycemia, the neurological signs indicate a depression first of the higher centers, cortex and cerebellum and subsequently of the lower centers, the basal ganglia and hypothalamus, then the mid-brain and finally the medulla becomes involved.[11] It has been established that for the optimum results from insulin treatment the hypoglycemia should be maintained until there is evidence of mid-brain release, namely,

torsion and extensor spasms.[12] The sequence of changes observed in the nitrogen therapy is the same as with insulin except that the duration of the changes is limited to seconds rather than hours. We have taken the same criteria for termination of the nitrogen treatment as are now accepted for insulin. *Anoxemia beyond this point would involve the vital centers of the medulla and is emphatically warned against because it is not compatable with safety.*

The arterial blood oxygen saturation at the end of the convulsion in metrazol shock is usually about 40 per cent.[13] The average saturation for nitrogen therapy is lower, about 20 per cent.

Typical changes of oxygen content and hemoglobin saturation of the arterial blood during the nitrogen treatment are presented in Table I.

TABLE I.

Effect of Progressive Anoxia on Hemoglobin Saturation of Patient Receiving Nitrogen Inhalation Treatment.

Condition.	Oxygen content vol. per cent.	Oxygen capacity.	Hemoglobin sat. per cent.
Onset of unconsciousness.....	4.99	18.24	22
Before termination with oxygen.....	2.46	18.24	13

Results.

Since June, 1938, over 300 treatments have been administered to 13 patients at the Albany Hospital without serious complications. In five other hospitals the therapy has been administered over 500 times. Table II represents an analysis of the clinical results in the series treated in Albany as supplied by Dr. Basile Lipetz from whose service the clinical material was obtained.

Apparatus.

The apparatus is that ordinarily used in gaseous inhalation anesthesia. It consists of a tight fitting face mask, a canister of soda lime and five-liter breathing bag connected in series. To the face mask, through a small nipple oxygen or nitrogen is admitted. Nitrogen is supplied from a large tank of commercial gas. Oxygen is delivered from an ordinary gas machine capable of releasing a

TABLE II.

Pt.	Sex.	Age.	Diagnosis.	Duration of illness.	No. of treatments.	Condition on discharge.
1.	M.	22	Paranoid.	6 weeks.	46	Full remission.
2.	M.	26	Paranoid.	2 months.	30	Full remission.
3.	F.	19	Hebephrenia.	1 year chronic 1 month acute.	45-10	Full remission. Mild relapse.
4.	M.	35	Paranoid.	1 year.	21	Improved.
5.	F.	34	Hebephrenia.	2 months.	11	Full remission.
6.	F.	23	Paranoid with catatonic features.	1 year.	18	Improved. Better adjusted.
7.	F.	31	Paranoid.	5 years. 3 years. 1 year.	19	Definitely improved. Better adjusted.
8.	F.	35	Manic-depressive manic stage. Schizoid tendencies.	10-12 years (recurrent).	14	Unimproved.
9.	F.	20	Schizophrenia simplex, hypochondriacal.	3 years	15	Somewhat improved. Left of own accord.
10.	F.	34	Depressive psychosis with schizoid tendencies.	9 months	29	Greatly improved.
11.	F.	18	Hebephrenia.	1 year	17	Improvement at first and then no further improvement.
12.	F.	29	Schizophrenia simplex.	2 years	22	Somewhat improved.

rapid flow of the gas instantly. The lead tubes from the supplies of oxygen and nitrogen are connected by a Y piece, a few inches from the face mask. An exhalation valve at the end of the breathing bag permits the gases to spill over into the air. Fig. I represents the apparatus unassembled and Fig. 2 as it is actually employed.

Method.

The treatments are administered in a room close to the psychiatric wards. The patients walk to the room accompanied by a nurse. They are then assisted in climbing upon an ordinary treatment table with a regular full size pillow, covered with a sheet or blanket, and their shoes are removed. No mechanical restraints are used at any time. It is rarely necessary to restrain the patients manually and then only for a short period at the height of the anoxia. When the patient is made comfortable on the table, blood pressure and pulse readings are taken till they reach control levels. This is usually accomplished within a few minutes.

Preceding the first treatment the method is explained to the patient before the mask is applied. It is a practice for the operator to reassure the patient as is done before inhalation anesthesia is induced. With the patient's head turned slightly to the side the mask is applied with about five liters of oxygen running into it. The mask is then adjusted till there is a tight fit and no leak in or out of the mask about the face. When the breathing bag is full the oxygen is shut off at the gas machine so that its addition to the mask is discontinued. The tank valve on the nitrogen tank is then opened and the control valves adjusted until about six liters per minute of nitrogen are flowing into the mask. This rate is continued until the treatment is terminated. As the nitrogen flows into the apparatus it gradually increases in percentage in the mixture at the expense of oxygen.

When the anoxia is sufficiently acute that it is desired to terminate the treatment, the nitrogen control valve is closed and oxygen admitted rapidly to the mixture. These maneuvers take less than three seconds and it is possible to terminate the treatment almost instantly. The physiological processes rapidly return to normal. The mask is usually removed from the patient's face about one minute after oxygen is administered. The patient is asked how

he feels, is allowed to lie quietly for about three minutes and then permitted to step down from the table and walk back to the ward. It is advised that the ambulatory patients rest for at least one-half hour after the treatment before leaving the hospital.

Treatments are given thrice weekly on alternate days at about 1.30 p. m. and luncheon is with-held till treatment is finished. For our practical purposes this time is most convenient; for others a treatment before breakfast might be preferable.

LANDMARKS—SIGNS OF INCREASING ANOXIA.

While the signs of anoxia may vary slightly in individual patients from day to day, and between different patients to a greater extent, they are usually constant. Fig. 3 represents a record of a typical treatment demonstrating the chronological order of onset of signs.

Neuro-Muscular Changes.

Shortly after the administration of nitrogen and preceding unconsciousness, there may be a transient period of restlessness during which the patient may move his hands, legs or head voluntarily. The first neuro-muscular change observed is twitching about the face, usually at the corners of the mouth. This is followed quickly by clonic contractions of the muscles of the extremities. The period of clonic contractions lasts usually about 20-30 seconds and may be followed by torsion extensor spasms, tonic-tetanic spasms, and extensor spasms such as opisthotonos. When these muscular changes have appeared the bout is arbitrarily terminated.

Consciousness is lost at about the time that neuro-muscular changes appear and is regained immediately when oxygen is introduced. The spasms relax and the patient assumes a normal supine position almost immediately. Frequently clonic contractions and twitchings about the face are observed transiently in the recovery period as the depressive effects of anoxia rapidly regress through the phylogenetic layers of the brain. There is no post-treatment soreness or stiffness and no neuro-muscular changes severe enough to injure or embarrass the patient even though he lay on a hard treatment table. It is not necessary to employ mouth gags to protect the lips and tongue. There is no post-treatment confusion.

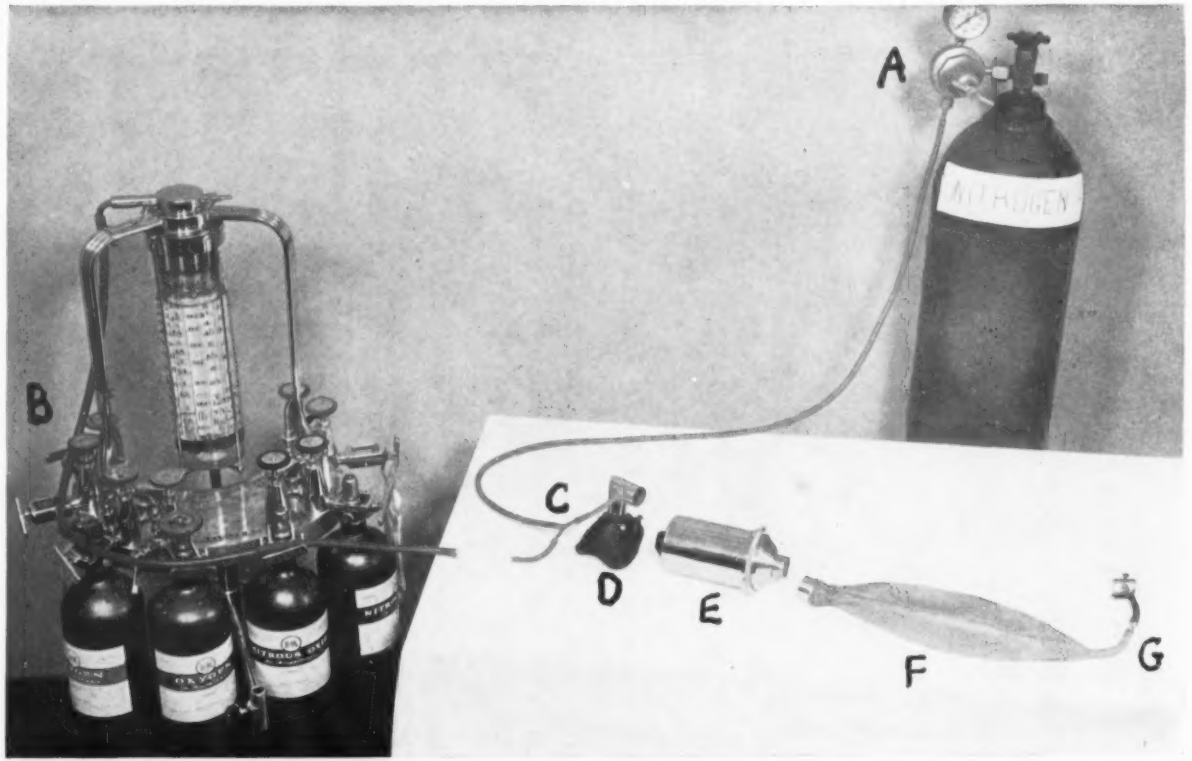


FIG. 1.—Apparatus unassembled. *A*, Tank of commercial nitrogen with reducing valve. *B*, Oxygen supply from anesthesia gas machine. *C*, Y-connection piece where gases are admitted to the mask. *D*, Face mask. *E*, Canister of soda lime. *F*, Five-litre breathing bag. *G*, Exhalation valve.

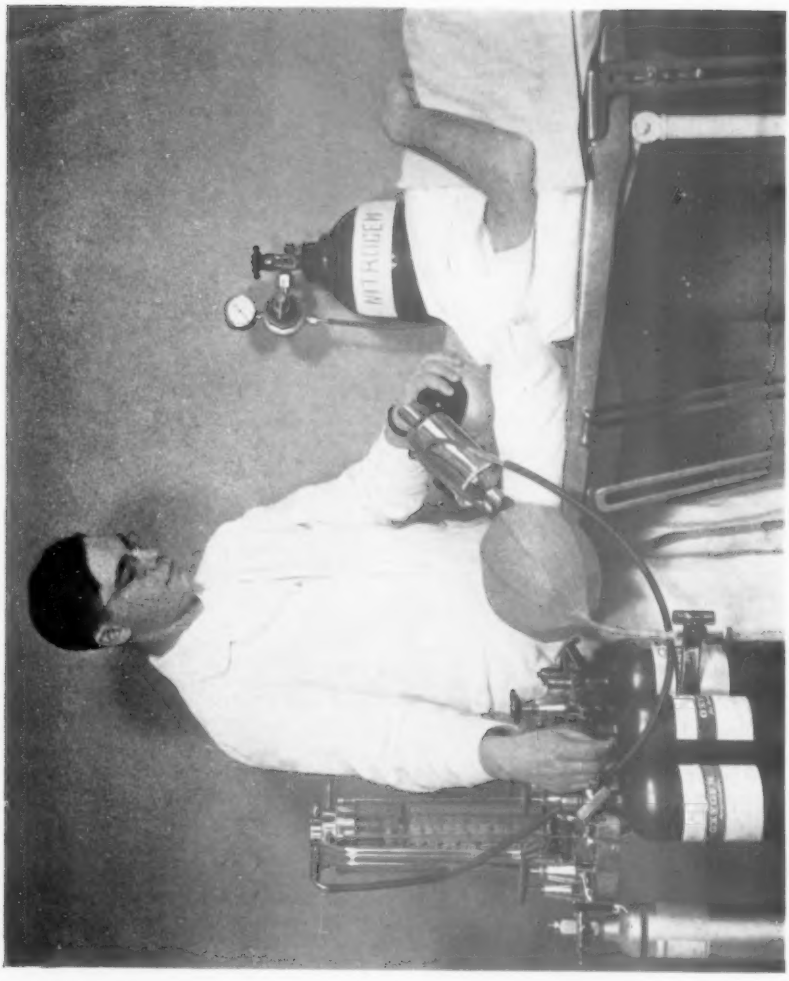


FIG. 2.—Apparatus assembled and in use.

Respiratory Changes.

Respiratory stimulation is the most consistent change observed during these treatments. It appears as the first sign of onsetting anoxia, usually just before the heart rate begins to increase. The

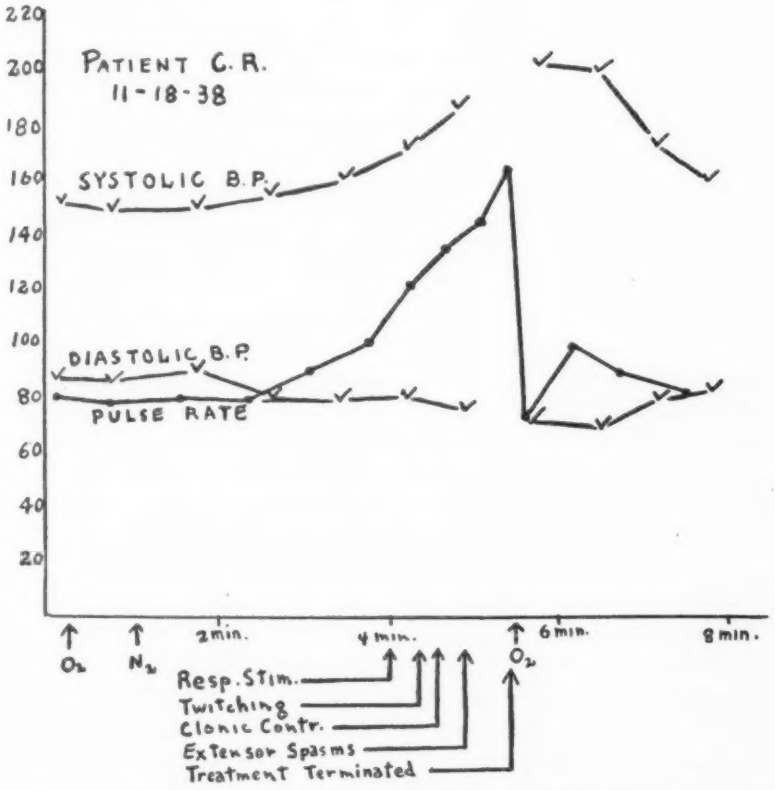


Fig. 3.—Graphic record of circulatory changes during a typical treatment. Chronological order of appearance and duration of neuro-muscular signs designated at bottom of chart. At the height of the anoxia it was impossible to read the blood pressure because of mechanical reasons. Unconsciousness occurred at about the point where twitchings appear.

change is usually first in rate and then in volume until the minute volume is greatly increased. After the beginning of the neuro-muscular changes respiration becomes arrhythmic though still augmented. At the height of the bout there may be considerable

respiratory obstruction. At this time inspiration is usually short and unobstructed, expiration prolonged and obstructed. There is frequently phonation. In patients with thick, short necks the respiratory obstruction may be severe. Cyanosis is more pronounced in these patients and the veins of the neck are dilated and engorged. We have observed only one case in this series in which respiratory arrest occurred before the treatment was terminated and one case in which obstruction was sufficiently severe to decrease respiratory exchange seriously. It must be emphasized that the controllability of the progressive degree of anoxia produced presupposes that a patent airway is maintained and that tidal exchange is not interfered with greatly by respiratory obstruction. If severe obstruction should occur the degree of anoxemia would be augmented and the expedition with which oxygen admission to the mixture terminates the anoxia, seriously retarded.

When oxygen is admitted terminating the treatment, apnea usually occurs immediately. Its duration varies but spontaneous respiratory efforts are usually resumed in less than 12 seconds. This period of apnea is probably due to a carotid sinus mechanism and is likely to be more pronounced where acute anoxia is suddenly terminated by inflation with oxygen during narcosis or anesthesia.

Circulatory Changes.

About two or three minutes after the beginning of nitrogen administration the pulse rate begins to increase and becomes progressively faster until the treatment is terminated. This change in rate is usually constant; that is, once it has begun to change it increases in each successive 30 seconds by a constant amount and appears as a straight line on the chart. At the height of the anoxia it is often impossible to palpate the radial pulse because of the intense muscular contraction at the wrist. Electrocardiographic tracings at that time may be difficult to obtain for obvious reasons. When the pulse rate reaches or exceeds 160 we arbitrarily terminate the treatment. Introduction of oxygen is usually followed immediately by a sudden fall in pulse rate to or below normal. Occasionally there is a transient bradycardia. This bradycardia, relative or actual, is followed by a mild tachycardia lasting about a minute or two with a subsequent return to normal. We have frequently

observed a sinus arrhythmia at the time of the bradycardia. If it is present normally in the patient it is likely to be accentuated.

Blood pressure changes are not as consistent as pulse changes and tend to vary considerably between different patients. About one or one and a half minutes after tachycardia begins, the systolic blood pressure begins to increase. Usually it increases progressively until the anoxia is acute and the neuro-muscular changes severe. During the height of the treatment it is often impossible to read the blood pressure because of the spasm of the muscles. Following administration of oxygen the systolic blood pressure usually continues to rise. In some cases it rises abruptly to 200 mm. or over. After a short period it falls gradually to normal but lags behind the pulse rate. The systolic blood pressure is often highest at the time of the post-termination bradycardia.

The diastolic blood pressure varies greatly among different patients. It may rise slightly, remain unchanged or fall markedly. It frequently begins to fall at about the time the systolic pressure begins to rise and may approach zero before the treatment is terminated, due perhaps to a relative aortic regurgitation. In such cases, however, the diastolic pressure returns to a normal level gradually (three minutes) after the treatment is ended.

Electrocardiographic tracings (lead II) taken during the nitrogen treatment of schizophrenia revealed the following changes in order:

1. Initial tachycardia of sinus origin.
2. T-wave changes and occasional premature auricular systoles.
3. Towards end of treatment (if anoxia severe enough) S-T deviations, sudden slowing of sinus pacemaker to normal rate and, in one case, bradycardia. Occasional premature ventricular extrasystoles.

Other Changes.

At the greatest depth of anoxia, wide dilatation of the pupils occurs with no reaction to light. Cyanosis develops usually after respiratory stimulation and tachycardia are in evidence. It apparently depends somewhat on the degree of respiratory obstruction and is most severe when respiratory obstruction is greatest and when the neuro-muscular activity is most intense. A normal color is regained almost immediately when oxygen is introduced.

There is always increased salivation at the height of the treatment, rarely increased mucous production. Urinary incontinence occurred eight times, six in the same patient.

Complications.

Several technical complications may arise which are not of serious consequence but tend to interfere with the efficiency of the treatment. The soda lime in the canister should be always fresh and adequate. We believe that the freedom from a sensation of suffocation which the patients experience is due largely to an efficient absorption of the carbon dioxide from the exhaled air. There should be no obstruction to respiration from failure to have the bag well distended. Two of our patients occasionally took large inspirations which almost depleted the bag. Sudden obstruction to inspiration because of insufficient gas might cause the patient to become alarmed. It is absolutely necessary to obtain a tight fit of the face mask about the nose and mouth and maintain it. In excited patients constant care must be taken to keep the fit tight. A momentary lapse allows the mixture to be diluted with air and necessitates beginning the treatment over again.

The patient should be made as comfortable as possible before treatment is begun so that he is not distracted or disturbed. It should be insured that the mouth is free of gum, bridge work, food or other foreign material.

The apparatus should be checked before each treatment to make sure that it works efficiently. An ample supply of oxygen is an absolute necessity.

The most important essential for the management of complications is the presence of an operator who is familiar with the use of gas equipment and who can recognize the signs of untoward reaction and the criteria for termination of the treatment. In our experience the persistence of respiratory effort even with the severe anoxemia is a comforting fact. In some cases we have merely removed the mask at the height of the anoxia to determine whether such would be a safe procedure in the event of an emergency. Though return to normal was slower than when pure oxygen was insufflated there was no evidence of danger.

Nitrous Oxide.

In an effort to ascertain whether nitrous oxide offered any advantage over nitrogen in this form of therapy, we have used it in an exactly comparable manner in 20 cases. Not only do the signs and symptoms differ quantitatively, if not qualitatively, but we feel that nitrous oxide is not satisfactory because of the post-treatment confusion and nausea which occur and because we have observed ominous electrocardiographic changes indicating serious cardiac arrhythmias when this agent has been employed.

Discussion.

The dangers of producing irreversible changes in the brain by complicating anesthesia with acute anoxia are well known.[13, 14, 15] This treatment is designed to produce a definite depression of cerebral metabolism and yet avoid irreversible changes. It has been shown that the older parts of the brain phylogenetically are more resistant to anoxia than the cortex and basal ganglia.[16] It is further known that central nervous system cells are most likely to be irreparably damaged if anoxia is relatively prolonged.[17] It seems, therefore, that a very short period of acute anoxia will be most likely to produce desired therapeutic effects with the minimum of danger to the patient. If the criteria presented are adhered to and the depression of cerebral metabolism not carried to the vestibular nuclei so that the treatment is stopped when extensor spasms appear, danger of involvement of the vital medullary centers and unsuccessful resuscitation should be avoided.

It must be emphasized that technical inability to protect against respiratory obstruction may plunge subacute anoxia into the acute stage involving the medulla. It is advised, therefore, that only those who have had experience in anesthesia perform these treatments.

All patients should receive a thorough physical examination before the therapy is instituted. Pre-existing cardio-circulatory pathology should, at least for the present, preclude treatment. Mild upper respiratory infection is not a contra-indication to the therapy. In view of the known dangers of anoxia occurring during anesthesia and the possibility of altering the signs it is felt that sedative medication in association with the treatment should be avoided.

Although it is believed that the heart is relatively resistant to anoxemia, any evidence of cardio-circulatory dysfunction during the treatments should be an indication to proceed with extreme caution.

Conclusion.

Encouraging results from a year's experience in the treatment of schizophrenia in a small group of patients by nitrogen inhalation therapy have been noted.

The methods and technique employed have been described.

The landmarks of treatment and the signs and symptoms of acute anoxia observed during these treatments have been discussed.

As a result of our experience with this form of therapy we believe that it can offer most promising and safest results if it is conducted by physicians well trained in the use of gas anesthesia apparatus, cognizant of the signs and symptoms of acute oxygen want, and capable of instituting efficient resuscitating measures in the event of untoward reaction.

It appears that the most satisfactory results will obtain if the anoxia produced is severe and the time of the treatment limited to six minutes.

The use of nitrous oxide as a substitute for nitrogen or the employment of depressant drug sedation in conjunction with nitrogen inhalation therapy are contraindicated.

BIBLIOGRAPHY.

1. Sakel, M.: Neue Behandlungsmethode der Schizophrenia, Moritz Perles, Wien and Leipzig, 1935. See English translation. Nervous and Mental Disease Monograph, No. 62, New York, 1937.
2. von Meduna, L.: Z. f. d. ges. Neur. u. Psychiat., 152: 235, 1935.
3. Himwich, H. E., Bowman, K. M., Wortis, J., and Fazekas, J. F.: J. Nerv. and Ment. Dis., 89: 273, 1939.
4. Hoagland, H., Rubin, M. A., and Cameron, D. E.: Am. J. Physiol., 120: 559, 1937.
5. Himwich, H. E., Hadidian, Z., Fazekas, J. F., and Hoagland, H.: Am. J. Physiol., 125: 578, 1939.
6. Himwich, H. E., Frostig, J. P., Fazekas, J. F., and Hadidian, Z.: Am. J. Psychiat. (in press).
7. Rossman, I. M., and Cline, W. B.: Am. J. Psychiat., 94: 1323, 1938.
8. Friedman, M. M., Harris, M. M., and Horowitz, W. A.: J. A. M. A., 112: 1684, 1939.

9. Bennett, B. T., Jr., and Fitzpatrick, C. P.: J. A. M. A., 112: 2240, 1939.
10. Himwich, H. E., Alexander, F. A. D., and Lipetz, Basile: Proc. Soc. Exper. Biol. and Med., 39: 367, 1938.
11. Heymans, C., Bouckaert, J. J., Jourday, F., Nowak, S. J. G., and Farber, S.: Arch. Neurol. and Psychiat., 38: 304, 1937.
12. Frostig, J. P.: Am. J. Psychiat., 94, Suppl., 220, 1938.
13. Lowenberg, K., Waggoner, R., and Zbinden, T.: Ann. Surg., 104: 801, 1936.
14. Courville, C. B.: Medicine, 15: 129, 1936.
15. Steegman, Albert T.: Arch. Neurol. and Psychiat., 41: 955, 1939.
16. Dixon, T. F., and Meyer, A.: J. Biochem., 30: 1577, 1936.
17. Harreveld, A. V., and Marmont, George: J. Neurophysiol., 2: 101, 1939.

PHYSIOLOGICAL STUDIES OF EXPERIMENTAL INSULIN AND METRAZOL SHOCK.

II. Vascular and Respiratory Responses During Metrazol Convulsions.*

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Introduction.

The beneficial effects in the treatment of schizophrenia either by means of metrazol shock, with an associated hyperglycæmia, or by insulin shock, with a coincident hypoglycæmia, are well known. Although the percentage of so-called "cures" is somewhat lower now than it was even a year ago, the number of shock-treated patients who may be considered as having been benefited by such treatment represents a relatively large proportion of the schizophrenics. Even if these patients are improved sufficiently to allow them to leave hospital for a period of only six months to a year the institutional, state and provincial economy is greatly improved.

From the results of the experiments reported in this paper, and in papers by other workers, it is apparent that metrazol shock produces a sequence of rather serious events. No violent convulsions, of course, need occur in order to obtain similar beneficial effects in the patients treated by the insulin shock method. Unfortunately, though, insulin shock is much more difficult to study experimentally than is metrazol shock because the action of insulin is slower and depends upon so many physiological factors.

Although comparable experimental studies in connection with insulin shock have not been completed, it would appear that the physiological factors involved in these two types of shock therapy are not specifically related. What then is the mechanism which results in the almost dramatic improvement shown by some of the schizophrenics consequent to shock treatment? Since no specific common or related factors in the two types of treatment have been

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recognized to date and if further work does not reveal such common agents then the beneficial results must be attributed to some psychological influence. Perhaps when the results of our clinical investigation (which involves some 50 different, simultaneous physiological and biochemical determinations done at regular intervals in insulin and metrazol-treated schizophrenics as well as in untreated schizophrenics under similar controlled conditions) are completed some common factors may be brought to light. Then, too, a better understanding of the *modus operandi* of the treatments may lead to more rational types of therapy.

The possibilities of an early solution of the schizophrenia problem are obviously remote but not at all hopeless. Its ultimate disposition, however, is going to continue to require much experimental and clinical research from many different and perhaps completely unrelated aspects. In an effort to facilitate such work we feel that the periodical presentation of progress reports of the results of our experimental work is warranted.

In 1938 workers from this laboratory presented a preliminary report [1] on some experimental studies of insulin and metrazol shock which were carried out as part of a general investigation on schizophrenia. Several aspects of the work then reported in progress have since been discontinued while other aspects, the results of which will be reported in a series of papers, have received increased attention.

Although a great deal of work has been reported on the pharmacological action of metrazol (see review by Hildebrandt [2]) most of it has been done on anæsthetized animals and for the purpose of studying particular isolated actions. Few, if any attempts have been made to study the effect of metrazol on the intact, unanæsthetized animal and to correlate the various physiological and biochemical changes which occur simultaneously throughout the convulsion period.

The intravenous injection of metrazol, in effective convulsive doses, produces a typical series of physiological and biochemical changes characteristically associated with the various specific stages of the convulsion. These changes were studied throughout a total of 60 induced convulsions in a series of 45 normal conscious rabbits.

The purpose of this paper is to present the results of these experiments.

Experimental.

A. Stages of convulsion.

When an effective convulsive dose of metrazol (0.3-0.4 cc. per kilo of a 4 per cent solution, in normal saline) is injected intravenously, as rapidly as possible, a latent period of about six seconds elapses before any changes are observed in the animal. Following the latent period the animal shows evidence of great excitement and quickly loses consciousness. This stage of excitement, lasting about 10 seconds, is followed by the tonic phase which is characterized by extreme spastic extensor rigidity lasting about 15 seconds. The animal passes from this condition into a clonic stage characterized by synchronous running movements. The synchronous movements are later replaced by asynchronous running movements, at which time vigorous biting and chewing is observed. This combined clonic stage lasts from 1-4 minutes and gradually gives way to a stage in which gross random movements predominate. This gross activity becomes weaker and after 2-3 minutes the animal appears to be in a stage of exhaustion. The stage of exhaustion is of indefinite duration but usually lasts about 20 minutes. Recovery follows and the animal appears quite normal in from 30 minutes to one hour.

These phases and their time relationships are indicated in Fig. 1, *a*.

B. Systemic blood pressure, respiration and heart-rate.

Under local anæsthesia (1 per cent novocaine) cannulæ were inserted in the carotid artery and trachea for the purpose of recording kymographically the systemic blood pressure (B. P.) and respiration (Resp.), respectively. The blood pressure and respirations remained normal during the latent period of an induced convulsion. At the onset of the excitement phase the B. P. rose abruptly from the normal of about 120 mm. Hg. to 200 mm. or more and was maintained during this period (Fig. 1, *b*). A correspondingly great increase, both in rate and depth, of respiration occurred in the excitement stage (Fig. 1, *d*). At the beginning of the tonic phase the respirations ceased and complete apnœa persisted until well into the clonic phase. At the same time the B. P. decreased slightly and was maintained at this lower level through-

out the tonic phase, only to increase again with the onset of the clonic stage.

The heart-rate, recorded by means of a tonometer, increased only after the B. P. had reached its maximum during the stage of excitement. (The probability is that this initial increase in heart-rate is a response to the release of adrenalin). The heart-rate remained rapid throughout the tonic and clonic phases (Fig. 1, *c*).

Early in the clonic phase respirations began to reappear coinci-

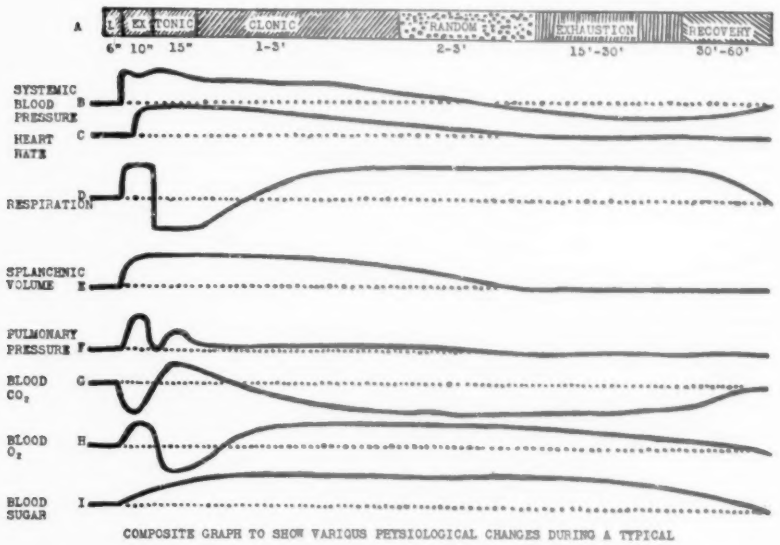


FIG. 1.—Metrazol Convulsion.

dent with the second rise in B. P. which occurred at this stage. The respirations gradually increased in rate and depth until intense hypernœa had developed late in the clonic phase. Coincident with the reappearance of respiratory movements the elevated systemic B. P. began to decrease gradually, reaching a normal level early in the random phase. The decrease in B. P. persisted and became subnormal later in the random phase and only returned to normal again late in the recovery stage.

The heart-rate returned to normal during the period of random movements. The respirations remained increased in rate and depth until the onset of recovery.

C. SPLANCHNIC VOLUME AND PULMONARY BLOOD PRESSURE.

At the height of the B. P. rise during the tonic phase, blanching of the ears and cyanosis of the mucous membranes were noted. Since these conditions occurred simultaneously with the increase in B. P. it appeared that such an increase was due to peripheral vasoconstriction. It was considered possible that a compensatory accumulation of blood might have occurred in the splanchnic area or in the right heart and pulmonary bed. It was necessary then to determine the changes which occurred both in the pulmonary blood pressure and in the splanchnic volume.

Under local anæsthesia a pulmonary cannula was inserted into the upper part of the right ventricle and directed well into the pulmonary artery. The pressure was recorded in mm. of Hg.

Plethysmographic recordings were made of the volume of several loops of small intestine. Constriction or dilatation of the splanchnic vessels was thus reflected in a change in the recorded volume.

Coincident with the sudden rise in systemic blood pressure and the appearance of peripheral vasoconstriction an equally sudden increase in splanchnic volume took place (Fig. 1, *e*). This increase was maintained until the systemic B. P. started to fall and then it, too, slowly decreased, returning to normal during the period of exhaustion. The splanchnic dilatation was also observed directly in the form of engorgement of the vessels and increased motility of the gut.

The peripheral vasoconstriction and increased systemic blood pressure were reflected, not only in the dilatation of the splanchnic vessels but also in a tremendous increase in the pulmonary blood pressure. Since there was an increase in the splanchnic volume it was assumed that the increase in pulmonary B. P. was not the result of an increased venous return to the right heart. The peripheral vasoconstriction, of central origin, increasing the peripheral resistance, resulted in an increased systemic B. P. With the associated increase in heart-rate it would be correspondingly more difficult for the left heart to expel its contents adequately. This, in turn, would render it more difficult for the pulmonary arterial blood to enter the left heart and a subsequent increase in pulmonary B. P., as recorded from the pulmonary artery, would result. Thus

the increase in pulmonary B. P., as evidenced in the stage of excitement, may have been the result of mechanical factors in the cardiovascular system. On the other hand, there may have been an actual constriction of the pulmonary vessels coincident with the peripheral vasoconstriction.

In any event, the pulmonary B. P. increased suddenly during the excitement phase, decreased just as rapidly at the onset of the tonic phase only to rise again during the period of apnœa in the tonic phase. With the re-establishment of respirations, early in the clonic phase, the pulmonary B. P. decreased significantly and then slowly returned to normal during the period of random movements.

D. CEREBRAL BLOOD FLOW.

Since changes in cerebral circulation are not entirely dependent on changes in systemic circulation (Wolff [3]) it is necessary to study the blood flow through the brain directly. Indirect methods of investigating the changes in cerebral blood flow, such as by recording the flow through the internal carotid artery by means of a thermostromuhr, are open to some criticism and almost invariably are influenced by the changes in systemic blood pressure and blood flow. The data obtained by this indirect method [4] have been reinterpreted and the experimental technique modified and improved in an endeavor to obtain a true evaluation of the changes in cerebral blood flow as a result of insulin and metrazol treatment. Such a study, by direct means, is at present in progress and will be reported at a later time.

E. INFLUENCE OF VARIOUS DRUGS ON METRAZOL CONVULSIONS.

1. *Curare.*—In view of the great changes in systemic B. P., respirations, pulmonary B. P., etc., it was necessary to repeat the above observations in curarized rabbits in order to rule out the possibility that such effects were the result of extreme muscular activity during the convulsion.

In a series of five curarized rabbits, maintained under artificial respiration, systemic B. P., pulmonary B. P. and heart-rate records were obtained following the intravenous injection of a convulsive dose of metrazol. No convulsive movements were observed. (In a preliminary trial no convulsive movements were observed even

after the administration of five times the optimal convulsive dose of metrazol).

As usual, the systemic B. P. increased after the ordinary latent period. After some 30 minutes the B. P. had decreased slightly to about 180 mm. Hg. and at the end of about 45 minutes had returned to normal. At no time did the B. P. fall below normal as seen in the non-curarized rabbits.

The heart-rate changes observed in these curarized animals were essentially the same as in the non-curarized ones.

The pulmonary B. P. increased suddenly coincident with the rise in systemic B. P. but unlike the changes observed in the non-curarized animals this increase was not as marked and was maintained for a period of about 15 minutes. This might possibly be accounted for by the absence of any period of apnœa, since artificial respiration was of necessity used throughout the experiments.

In general, then, it may be stated that the cardiovascular changes observed during an ordinary metrazol convulsion were not the result of the great muscular activity which is a feature of the convulsion.

2. *Atropine.*—It was found that the minimal lethal dose of metrazol was not greatly in excess of the optimal convulsive dose. In atropinized animals (0.2 mg. atropine per kilo) it was possible to inject as much as four or five times the optimal amount without fatal results. Typical convulsions, however, were produced by the injection of even the normal convulsive dose.

The systemic B. P. increased suddenly in the stage of excitement but was less marked than in non-atropinized rabbits. This increase was maintained for some 30 minutes, while in non-atropinized animals the increase lasted only about four minutes. Actually the systemic B. P. changes were quite similar to those observed in the curarized animals.

The heart-rate showed changes more marked than those in non-atropinized animals. A pronounced tachycardia commenced during the excitement phase and continued well into the random period, after which time the rate slowly returned to normal.

The changes in respiration were similar to those seen in the normal rabbits both in their character and in their time-relation with the convulsive phases.

Pulmonary B. P. changes were not nearly as marked as in the non-atropinized series.

3. *Nicotine.*—Nicotine solution, 1 : 1000, was injected in small, single doses into a series of rabbits until no further increase in B. P. was produced by subsequent injections. At this time a convulsive dose of metrazol was administered. The resulting convulsions were typical in every respect. The physiological and biochemical changes associated with the various phases of the convulsions were essentially the same as those observed in animals which had not been subjected to previous nicotine injections.

4. *Barium Chloride.*—All of the previous experiments were performed on animals with normal blood pressure and with normally reactive vascular systems. It was deemed advisable to study the effects of metrazol shock in a series of animals in which a vasoconstriction, with a resulting increase in systemic blood pressure, had been produced prior to the injection of metrazol. This condition was obtained by the intravenous injection of barium chloride (10-12 mg.). When the B. P. had become constant at about 150 mm. Hg., metrazol in the usual convulsive dose was injected. Typical clinical phases of the convulsion were observed. The B. P. did not reach the levels seen in rabbits which had not received the barium chloride, increasing only to 170-180 mm. Hg.

Changes in respiration were greater than in rabbits which had not received barium chloride. The period of apnœa was considerably shortened and there was a greater increase in the rate and depth of respiration. The pulmonary pressure changes, although of greater magnitude, were essentially similar to those seen in animals without barium chloride. In a large percentage of experiments (70 per cent) acute pulmonary œdema supervened during the terminal random movements and the animal died suddenly.

5. *Adrenalin.*—The synergism of adrenalin and metrazol was studied in a series of five normal conscious rabbits. An intravenous injection of adrenalin (0.5 cc., 1 : 10,000 solution) was followed 10 seconds later by an injection of the usual effective convulsive doses of metrazol. The B. P. response to metrazol and adrenalin appeared to be additive since the systemic B. P. reached, on an average, 310 mm. Hg. The heart-rate increased shortly (6-8 seconds) after the adrenalin was injected, became very rapid and irregular at the onset of the phase of metrazol excitement, and

remained that way until the systemic pressure had returned to normal. The pulmonary pressure showed a very rapid rise to 60-70 mm. Hg. and remained high, then gradually fell to normal during the period of random movements. In this series of experiments four of the five rabbits died within five minutes of the injection of metrazol, from acute pulmonary œdema.

The rapid development of pulmonary œdema following the administration of adrenalin and metrazol was similar to the type of pulmonary œdema seen in the barium chloride-metrazol treated rabbits. In the barium chloride-metrazol treated animals the systemic blood pressure increased only to 170-180 mm. Hg., much less than with metrazol alone, while the B. P. in the adrenalin-metrazol treated animals increased to over 300 mm. Hg. The pulmonary pressure changes in both these series were greater than in animals treated with metrazol alone. Since both barium chloride and adrenalin produce constriction of the pulmonary bed it seems reasonable to assume that the additive vasoconstrictor effect of metrazol is responsible for the development of the pulmonary œdema in these instances. This was further tested in a series of 12 rabbits, to which varying amounts of adrenalin were given 5-6 seconds prior to an injection of metrazol.

It was first established that small quantities of adrenalin (0.2 cc. per kilo, 1 : 10,000 solution) enhanced the effect of a normal convulsive dose of metrazol (0.3 cc. per kilo of a 4 per cent solution) to such an extent that all the animals died of pulmonary œdema. Convulsions and fatal pulmonary œdema were also produced by using sub-convulsive doses of metrazol (0.2 cc. per kilo) after adrenalin administration. With the same amount of adrenalin typical convulsions, without fatal pulmonary œdema, were produced with as little as 0.05 cc. metrazol per kilo (4 per cent solution). Death from pulmonary œdema also resulted from the administration of 0.1 cc. of adrenalin (1 : 10,000) per kilo, and 0.2 cc. per kilo of metrazol (4 per cent solution).

This synergism between adrenalin and metrazol, which is very apparent in the rabbit, may or may not be observed in other species of animals. If such a condition is found to exist in the cat, dog, etc., it should be realized that this same type of synergism might also occur in human cases with elevated blood pressures or in individuals in whom the release of excess adrenalin might con-

ceivably take place. In such individuals metrazol shock therapy might be contraindicated.

F. CHANGES IN BLOOD GASES.

Investigations of the changes occurring in blood CO_2 and O_2 were undertaken to test the hypothesis that cerebral anoxia might

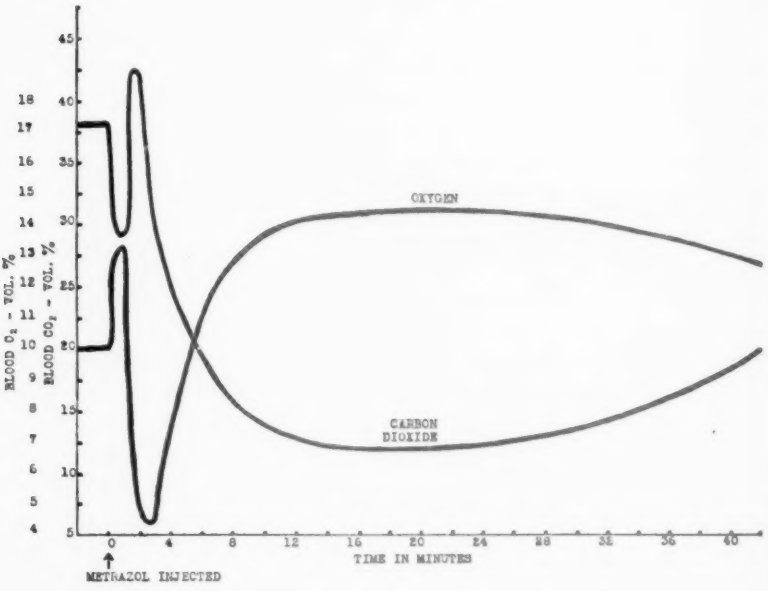


FIG. 2.—Metrazol Convulsion. Changes in Blood Gas in a Normal Conscious Rabbit.

be the mechanism underlying the therapeutic effect of metrazol convulsions in schizophrenic patients. A blood-pressure cannula was inserted in one carotid artery. A "T" cannula was introduced into the opposite carotid artery, so that samples of the blood supplied to the brain might be obtained at various stages of the metrazol convulsion. Constant and typical changes in the blood CO_2 and O_2 were found to occur (Fig. 2).

The CO_2 content of arterial blood decreased suddenly during the phase of excitement only to increase beyond normal during the period of apnœa in the tonic phase (Figs. 1 and 2). With the

re-establishment of respiration in the clonic phase (Fig. 1) the arterial blood CO_2 fell rapidly to very low levels (10-15 vol. per

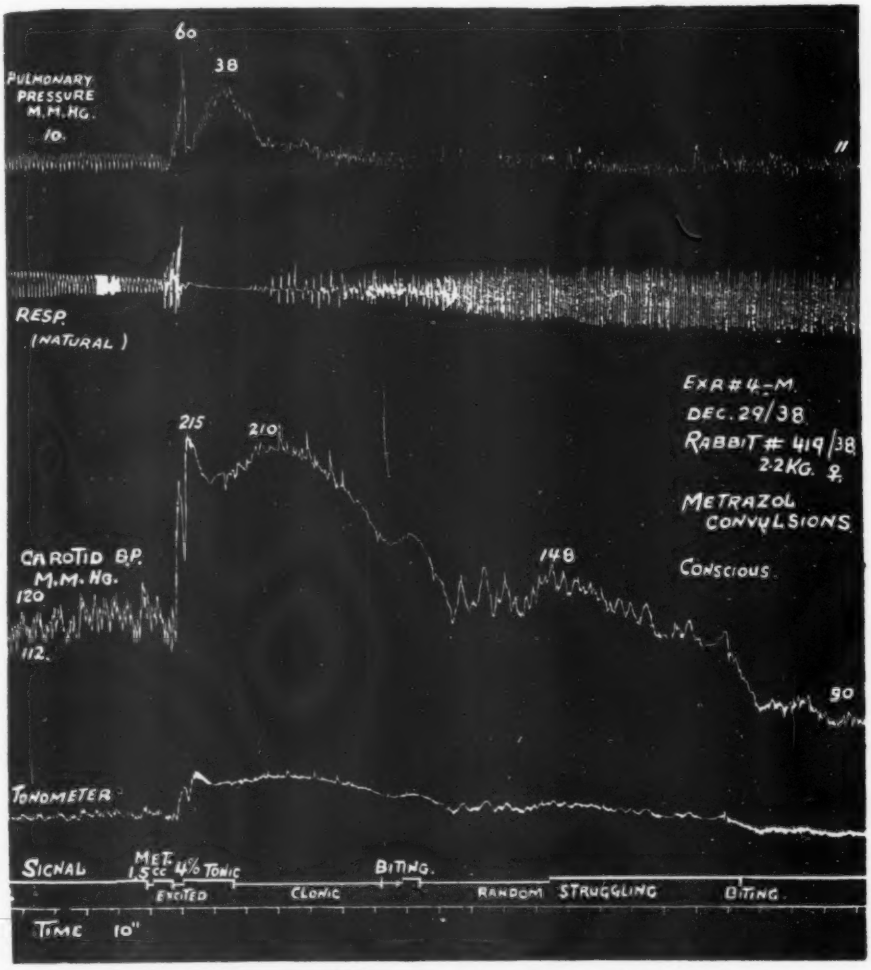


FIG. 3.—Composite Kymograph Record to Show Changes in Blood Pressure and Respiration During a Typical Metrazol Shock in a Normal Conscious Rabbit.

cent). This hyperpnœic acapnia resulted from the interaction of two factors: (1) stimulation of the respiratory centre by the metrazol resulting in an increase in the rate and depth of respiration (Fig. 3) with a "blowing off" of CO_2, and (2) the hypercarbia

and anoxia of the previous apnœic period. The arterial CO_2 reached its lowest level 15-20 minutes after the convulsion and returned to normal in approximately one hour (period of recovery, Fig. 1).

The O_2 content of arterial blood increased almost always during the phase of excitement and was associated with the increased respiratory activity in this phase (Figs. 1 and 2). During the tonic phase of the convulsion the O_2 content fell to subnormal levels. Since there was no respiratory activity during this phase cyanosis was evident in all instances. As respirations became re-established during the clonic phase of the convulsion the O_2 content of the arterial blood rose and with the development of hyperpnœa the O_2 continued to increase, reached a maximum of 16-18 vol. per cent in about 10 minutes and gradually returned to normal after 1-1½ hours.

The graphs showing the changes of the CO_2 and O_2 during a metrazol convulsion (Figs. 1 and 2) are almost perfect mirror images of each other.

G. BLOOD SUGAR CHANGES.

Determinations of true blood glucose were made on 0.3 cc. of arterial blood deproteinized by the method of Herbert and Bourne [5] and using a modified Shafer-Hartman reagent (Harding and Downs [6]).

Almost immediately after the injection of metrazol the blood glucose began to increase (Fig. 1) and reached its maximum value within a few minutes. This marked hyperglycæmia (175-300 mg. per cent) persisted throughout the later phases of the convulsion and gradually decreased to normal levels in from one to two hours following the injection of the metrazol.

Discussion.

The results of the experiments reported in this paper amplify and in some respects modify those presented in our preliminary report.[1] When the problem had been studied in more detail and from different aspects, the interpretation of two observations previously reported has had to be changed. In one instance the difficulty of obtaining blood samples from the ear veins during the

tonic phase of the metrazol convulsion was taken as evidence of a tremendous fall in blood pressure, whereas it has now been found that that difficulty was due to a very marked peripheral vasoconstriction, which was attended by an increase in the systemic blood pressure.

In the second, a misinterpretation in the earlier report may have occurred. Leibel and Hall [1, 4] concluded that a marked decrease in cerebral blood flow occurred during insulin and metrazol shock. The blood flow, in their experiments, was recorded by means of thermostromuhrs placed around the carotid artery and the meningeal branch of the jugular vein. Although decreases in blood flow through those vessels were recorded, the influence of vasoconstriction of the peripheral branches of the carotid artery may have been sufficient to produce such a decrease independent of any changes in the blood flow through the brain substance.

Thus the necessity of studying this problem from as many aspects as possible is apparent and the desirability of obtaining the results, not of isolated pharmacological actions but of many simultaneously recorded physiological and biochemical events has become increasingly evident.

The difficulty of recording such events in the conscious animal is great. However, the difficulties of obtaining continuous recordings of systemic and pulmonary blood pressures, respiration, heart-rate and gut volume, and at the same time securing samples of blood for blood sugar and gas analysis during an induced metrazol convulsion are obviously even greater. Such simultaneous observations have been made, however, in a sufficiently large series of animals to give us an idea of what actually is happening during such a convulsion.

Fig. 1 is a composite graph showing the various changes which occur during a typical metrazol convulsion in a normal conscious rabbit. From a study of this graph it will be seen that the physiological and biochemical changes are related, rather specifically, to the phases of the convulsion.

No changes occur during the latent period but with the onset of the stage of excitement the systemic blood pressure increases abruptly and the respirations increase in rate and depth. This hyperncea is sufficient to "blow off" considerable CO_2, which is reflected by a marked decrease in the blood CO_2, and to increase

oxygenation as indicated by the increase in blood O_2. At the same time an increase in the splanchnic volume and a simultaneous increase in pulmonary blood pressure take place. This increase in pulmonary blood pressure may be due to a constriction of the pulmonary blood vessels or may be the result of secondary mechanical factors or both. As the pulmonary blood pressure reaches its maximum the heart-rate increases and only returns to normal towards the end of the phase of random movements. It will be noted also that the splanchnic volume follows the same course.

The respiratory movements change abruptly with the onset of the tonic phase of the convulsion. The previous hyperpnœa is replaced suddenly by a period of complete apnœa. Associated with this condition is a temporary return to normal of the elevated pulmonary blood pressure. The most pronounced effect of the apnœa is to be seen in a great increase in blood CO_2 and an equally significant decrease in the blood O_2.

At the beginning of the clonic phase respirations re-commence and within a short time increase again to an hyperpnœic state. These changes in the respiratory movements are reflected once more in the level of the blood oxygen and carbon dioxide. It will be noticed also that after the active convulsive movements have ceased and the animal is in the stage of exhaustion, the splanchnic volume, heart-rate and pulmonary blood pressure return to normal while the systemic blood pressure decreases to a subnormal level. The hyperpnœa which persists throughout this period is replaced by normal breathing late in the recovery phase, at which time the blood O_2 and CO_2 likewise return to normal.

The hyperglycæmia is maintained throughout the later phases of the convulsion. The changes which occur in the electrical activity of the brain during typical metrazol convulsions will be described in a later paper.

Certain conclusions of possible clinical significance may be drawn from the observations made in the above experiments. Although there have been relatively few fatalities as a result of metrazol shock treatment the experimental results here recorded would suggest that this type of therapy should be used with some degree of caution: the patients so treated should be selected as regards age and freedom from cardiovascular or pulmonary disease.

The sustained increase in blood pressure might be disastrous in cases having aneurysms or areas of cerebral softening, from whatever cause. Likewise, the subsequent fall in blood pressure might be of serious concern in patients with narrowed coronary arteries, in which cases a decrease in coronary artery blood flow might cause serious impairment of cardiac function. The tachycardia in some instances might lead to further cardiac irregularities.

Cognizance should also be taken of the possibility of a synergistic action between metrazol and other drugs and also of the effect of metrazol in cases with moderate elevation of the blood pressure.

Further, the studies of the changes in the electrical activity of the brain during such shock reveal that profound alterations occur within the central nervous system. We have not found any pathological changes in the brains of 12 rabbits which received some 30 daily injections of convulsive doses of metrazol. There is still the possibility that in man, and particularly in schizophrenics, pathological changes might occur as a result of metrazol convulsions.

Summary.

Some of the physiological and biochemical changes characteristically associated with the various specific stages of the convulsions produced by the intravenous injection of metrazol have been studied in unanæsthetized rabbits.

The changes in the heart-rate, systemic and pulmonary blood pressure, splanchnic volume, respiration and blood gases are of sudden onset and of marked degree.

The severity of these changes is such that the possibility of a hazard being associated with the clinical use of metrazol must be borne in mind.

BIBLIOGRAPHY.

1. Hall, G. E.: Am. J. Psychiat., 95: 553, 1938.
2. Hildebrandt, F.: Handbuch der Exper. Pharmak., 5: 151, 1937.
3. Wolff, H. G.: Physiol. Reviews, 16: 545, 1936.
4. Leibel, B., and Hall, G. E.: Proc. Soc. Exp. Biol. and Med., 38: 894, 1938.
5. Herbert, F. K., and Bourne, M. C.: Biochem. J., 24: 299, 1930.
6. Harding, V. J., and Downs, C. E.: Can. Chem. Met., 16: 12, 1932.

THE RESULTS OF SHOCK THERAPY IN THE TREATMENT OF AFFECTIVE DISORDERS.*

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The extension of shock therapy to the affective psychoses has been by no means as widespread as in the content disorders such as schizophrenia, paranoia and compulsion states. The reports by Steinburg and Nierenberg in the Elgin Papers and that of R. H. and G. A. Young and that of Friedman and Meduna in the *Journal of American Medical Association* for February 11, 1939, are practically the only reports in the literature dealing with the treatment of these disorders. The fact that a large percentage of the patients so afflicted recover spontaneously creates the doubt of the justification for such severe treatment as jaws are dislocated, femurs broken, vertebræ are crushed, and even death may occur following convulsions induced by metrazol. Nevertheless, the difficulty of handling the case of acute mania and the suffering of the patients with the depressed and mixed forms of manic-depressive psychoses would justify any procedure that would shorten the course of the disease.

The first case of manic-depressive psychosis was treated with metrazol convulsions at the University of Virginia Hospital in September, 1937. The results obtained indicated that other persons so afflicted should be similarly treated. In October, 1938, 21 cases of the manic-depressive group so treated were reported.

At the present time results of a follow up study on 37 cases diagnosed as with manic-depressive psychosis or involutional melancholia are reported. No case that has not been discharged from the hospital at least five months is included in the report.

Metrazol shocks were found to have a marked effect on the symptoms of the affective psychoses.

Often one or two convulsions produced an elation in a formerly depressed individual, or relieved a very agitated one to such an

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Ill., May 8-12, 1939.

extent that he could eat and sleep. This sudden and often miraculous change, on the other hand, would in some instances soon fade if the treatments were not continued, to be followed by all the original symptoms which might be exaggerated, making the resulting state worse than that existing previous to the treatments. It was soon discovered also that this state of mild elation and well-being was sometimes upset by subsequent shocks; also, it was found that patients often reached a state of partial improvement from which no amount of shock could lift them.

In treating the affective disorders (in distinction to the treatment of the schizophrenias) it is necessary to deal with an older group—a group in which the muscles and joints are less pliable and the cardiovascular system more likely to be diseased. Also, it was found that when a certain point was reached occupational therapy, hydrotherapy and psychotherapy offered more than further shocks. Therefore, it was decided that an optimum point could be reached, where metrazol injections could be stopped and the newborn approach to the patient cultivated. Thus the body and brain of the patient might escape further strain, while the results would be even more gratifying.

These facts led to the employment of a somewhat new technique in the handling of these disorders. First, a complete cardiovascular study was made in order to estimate the status of the heart, but especially of the peripheral vessels. Then, if there were no other contra-indications, shocks were given every other day for a week. Following this, if there was improvement, two or three days were allowed to elapse while the patient's amnesia and post-metrazol confusion disappeared, allowing an estimate of symptomatic results to be made. Then, if deemed necessary two or three more shocks were given and, again, a rest period allowed. During these short periods of observation the patient was sent out, made to exercise and to mingle with others as in normal life. By feeling along in this manner, advantage could be taken of each forward step, so that it was seldom necessary to give over eight or 10 convulsions to produce the desired result. The treatment ends when the patient has reached a state of well-being, with insight and freedom from former agitation, or if it is found after 15 convulsions that there is no improvement. Only nine of the cases reported had as many as 10 convulsions.

This method has several disadvantages. The longer interval between treatments allows the patient to dread convulsions more. Yet, it is possible that this very dread aids in the therapy. The pauses allow the patients to appreciate their back pains and other aches, but this tends to shift their attention from their former preoccupations, permitting the doctor, through therapy, to get in better touch with the patient. Most cases show improvement after a few successful treatments. The results presented must be considered from the point of view of the general hospital and not one especially adapted to treating the mentally sick, as all of these patients were in the public wards or in private rooms of a general hospital which has no psychiatric ward.

TABLE I.

	At discharge.		
Results.	No.	Per cent.	
Greatly improved	17	46	89
Improved	12	32	
Slightly improved	4	11	
Unimproved	4	11	

In Table I the condition of the patients at discharge is presented. The results are given as greatly improved, improved, slightly improved and unimproved. These terms are vague, connoting diverse meanings, but seem to us better than remission, partial remission, etc. Seventeen of the 37 were considered greatly improved or in complete remission; 12 were thought definitely improved; four were slightly improved and four unimproved. If percentages are used it is found that 46 per cent were considered greatly improved; 32 per cent improved and 11 per cent slightly improved, making a grand total of 89 per cent improved. This result seems unduly high, yet convulsions certainly have a profound effect on the affective symptoms in practically every case that submits to more than two convulsions. On the other hand, it is also true that what is sometimes interpreted as improvement may be only a change in the picture.

Table II gives a somewhat different picture. This shows the results collected after at least five months have passed. Fifteen cases are still greatly improved and, except for an occasional back pain, are apparently well. Nine are improved, but, although able

to do their work, have pains and periods of depression. Four are slightly improved and nine have relapsed and are all in state hospitals or other institutions. It is interesting to note that two patients who ran away and were discharged unimproved are now among those apparently well. Although the follow-up is not so gratifying, yet 41 per cent are apparently well and 76 per cent improved—a result better than the reports on such cases treated by other methods.

Although this series is small, it was thought worth while to subdivide the group; first, into those with involutional melancholia, and those with manic-depressive psychosis, and then to subdivide the latter into the manic phase, the depressed phase, the mixed form and those with a marked schizophrenic coloring. The involutional group are 19 in number; the manic-depressive group includes three manic and seven depressed patients, while there were four in mixed depressions and four with manic-depressive reactions with schizophrenic coloring.

TABLE II.

Results.	At follow up. No.	Per cent.	
Greatly improved	15	41	76
Improved	9	24	
Slightly improved	4	11	
Unimproved	9	24	

Treatment with metrazol produced convulsions of persons over 45 years of age, as are the great majority of those showing involutional changes, has many obvious risks; yet, of all the affective disorders the chronicity and severity of the agitated depression occurring at the involutional period more clearly justifies any treatment that will definitely shorten their course. Careful study, especially of the cardiovascular apparatus, previous to shock, accompanied by manual restraint during convulsions, has enabled us, in the treatment of this group, to escape with no major injuries. X-ray after treatment has shown cupping of the bodies of the vertebræ and the patients have complained of back pain. Yet, this complication seemed to affect the patient's ultimate recovery very slightly.

Table III shows the results obtained in the cases with involutional melancholia. The age of the patients varied from 39 to 62. There were five men and 14 women; the average duration of illness before treatment was 13 months. The number of shocks varied from three to 25, but the average number was eight. The results at time of discharge indicate that nine, or 48 per cent, were greatly improved; four, or 21 per cent, were definitely improved; three, or 15 per cent, slightly improved, and three, or 15 per cent, showed no improvement. After a follow-up of at least six months it was found, however, that 14 of the 19 were doing their work and going about socially, one was socially adjusted but had not returned to her profession as a school teacher, while four were unimproved or

TABLE III.

INVOLUTIONAL MELANCHOLIA.

Age varied from 39 to 62—average 49.
Duration of disease before treatment 1 to 24 months—average 13 months.
Number of convulsions varied from 3 to 25—average 8.

Results.	At discharge.		At follow up.	
	No.	Per cent.	No.	Per cent.
Greatly improved	9	48	14	78
Improved	4	21	1	..
Slightly improved	3	15	..	..
Relapse	3	15	4	21

worse. Among 15 holding their improvement for at least six months, six still have pains in the back, which, however, apparently do not cause them great concern. Thus it appears that the affective disorder in 15 of the 19 patients, or in 78 per cent, had been greatly meliorated and, although approximately half of these still have body pains caused at least in part by the treatment, the patients themselves and the patients' activities establish the treatment as a success.

In the manic-depressive group there were three with the manic and eight with a depressive reaction. The follow-up shows that five of the 11 are still improved, while the remainder have had a relapse, usually returning to a depressed state. There is, then, only 45 per cent improvement in this group at the end of six months, a rate of improvement that might have occurred without treatment. However, shock therapy does enable one to handle a patient in

acute mania in a general hospital, as the patient becomes much quieter after two or three treatments.

The other two groups, that of mixed depression and that of those patients who had a history of previous attacks but on this

TABLE IV.

MIXED TYPE OF DEPRESSION.

History no.	Age.	Duration before admission, months.	No. of convulsions.	Condition at discharge.	Length of follow-up, months.	Present status.
142237	41	3	6	Greatly improved	7	Greatly improved
139049	45	5	9	Improved	9	Still improving; working. Has back pains.
140809	44	6	6	Improved	9	Working; social. Still has depressed spells.
128316	30	12	7	Improved	6	Greatly improved

TABLE V.

MANIC DEPRESSIVE WITH SCHIZOPHRENIC TRENDS.

History no.	Age.	Duration before admission, months.	No. of convulsions.	Condition at discharge.	Length of follow-up, months.	Present status.
122467	26	1	5	Greatly improved	5	Greatly improved
136107	30	3	31	Improved	10	Slightly improved
86248	32	24	17	Greatly improved	17	Greatly improved
139672	41	2	23	Improved	7	Greatly improved

admission, besides their symptoms of manic-depressive psychosis, showed others usually demonstrated in schizophrenia, are too small to warrant any conclusions. Those with the mixed form have all improved and seem to offer hope for further success in this type of disorder. Three of the four in the other group are apparently socially adjusted, but it was necessary to treat them with a greater number of convulsions to obtain this result. (See Tables IV and V.)

CONCLUSIONS.

A follow-up study six months after discharge from the hospital of 37 patients who had shown affective disorders shows 41 per cent as greatly improved and 76 per cent definitely improved. Of 19 cases diagnosed involutional melancholia, 15, or 78 per cent, have been able to make a social and business adjustment. Moreover, this study has shown that metrazol convulsions given with care can be used in patients in the fifth decade of life. We found also that a few convulsions when used in connection with other forms of therapy may produce satisfactory results in patients suffering from the mixed forms of depression, especially with the agitated depression accompanying the period of involution.

A COMPARATIVE STUDY OF HYPOGLYCEMIC SHOCK TREATMENT AND CONTROL OBSERVATION IN SCHIZOPHRENIA.*

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The purpose of this communication is to report the results of a comparative study of hypoglycemic shock treatment and control observation in schizophrenia. So far we have failed to find reports dealing with control investigations.

In choosing patients for treatment or control observation attempts were made to match the material according to sex, age and duration of psychosis. No definite limits were set for the duration of psychosis in order to compare the results in various duration groups. The technique was carried out along the lines given by Sakel. The control patients were given intra-muscular injections of physiologic salt solution at the same time when the treated patients were receiving insulin. They were kept in bed during the morning hours on the same ward where the treatment was carried out.

This report is based on the results of treatment of 100 patients, 50 men and 50 women and on the results of observation of 69 control cases, 36 men and 33 women, and of 15 other patients, eight men and seven women, who improved spontaneously while undergoing the various preliminary tests and examinations. The reason for the larger number of the treated patients is the fact that some of the unimproved control cases were later submitted to a course of treatment. The patients were followed up eight months to two years after termination of treatment or control observation.

General Survey of Results of Treatment and Control Observation.

Of the 100 treated patients 36 (36 per cent) showed various degrees of improvement while 61 (61 per cent) remained unim-

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proved (Table 1). One patient died during a protracted coma while two other patients died of pulmonary complications caused by the treatment. Of the improved cases 33 were sent home or placed in boarding homes. Seventeen of the improved patients (47 per cent) have relapsed and returned to the hospital.

Of the 69 control patients 15 (21.7 per cent) showed various degrees of improvement and 54 (78.3 per cent) remained unimproved. Thirteen of the improved patients were sent home or placed in boarding homes. Of the improved patients five (33.3 per cent) relapsed. Comparing the results in both groups we find a somewhat smaller percentage of improved cases in the control group. However, if we add to the control group the 15 patients who spontaneously improved during the period of the preliminary tests and examinations the percentage in the treated and untreated groups become equal. In this combined group of 84 control and spontaneously improved patients 30 (35.7 per cent) showed various degrees of improvement while 54 (64.3 per cent) remained unimproved. Twenty-eight of the improved patients were sent home or placed in boarding homes. Of the 30 improved patients eight (24 per cent) relapsed.

We shall not consider the groups by sex separately as there were only slight differences.

Survey of Results in Relation to Duration of Psychosis.

In our attempt to correlate the results of treatment or control observation with the duration of psychosis we use the same criteria for recovery, much improvement and improvement as other investigators have done. Patients who died during treatment were included in the unimproved group (Table 2).

There were no recoveries among the eight treated patients with psychosis of less than six months' duration. One patient (12.5 per cent) was much improved, six patients (75.0 per cent) improved and one patient (12.5 per cent) failed to improve.

Among the 27 treated patients with psychosis of from 6 to 18 months two (7.4 per cent) recovered, four (14.8 per cent) were much improved, nine (33.3 per cent) were improved and 12 (44.5 per cent) failed to improve.

None among the 65 patients with psychosis over 18 months duration recovered and none showed much improvement. Fourteen

TABLE 1.

GENERAL SURVEY.

Treated.		Controls.		Control and spontaneously improved.	
Improved	36 (36%)	Improved	15 (21.7%)	Improved	30 (35.7%)
Unimproved	61 (61%)	Unimproved	54 (78.3%)	Unimproved	54 (64.3%)
Dead	3 (3%)				
Total	100 (100%)	Total	69 (100 %)	Total	84 (100 %)
Released	33	Released	13	Released	28
Relapsed	17 (47%)	Relapsed	5 (33.3%)	Relapsed	8 (26.6%)

TABLE 2.

TYPES OF RESULTS IN RELATION TO DURATION OF PSYCHOSIS.

	Treated, 100.				Controls, 69.				Controls and spontaneously improved. 84.			
	Recov.	Much impr.	Impr.	Unimpr.	Recov.	Much impr.	Impr.	Unimpr.	Recov.	Much impr.	Impr.	Unimpr.
Less than 6 mos.	0	1	6	1	0	0	3	1	1	4	8	1
6 to 18 mos.	2	4	9	12	0	2	4	14	0	2	5	14
Over 18 mos.	0	0	14	51	0	2	4	39	0	4	6	39
Totals	2	5	29	64	0	4	11	54	1	10	19	54

patients (21.5 per cent) improved and 51 (78.5 per cent) remained unchanged.

Of the four control patients with psychosis of less than six months duration none recovered and none improved. Three patients (75 per cent) were improved and one (25 per cent) failed to improve.

Among the 20 control patients with psychosis of from 6 to 18 months duration none recovered, two (10 per cent) were much improved, four (20 per cent) improved and 14 (70 per cent) failed to improve.

Of the 45 control patients with psychosis over 18 months none recovered, two (4.4 per cent) showed much improvement, four (8.8 per cent) were improved and the remaining 39 (86.8 per cent) did not show any change.

Among the 14 combined control and spontaneously improved patients with psychosis of less than six months one (7.2 per cent) recovered, four (28.5 per cent) were much improved, eight (57.0 per cent) were improved and one (7.2 per cent) failed to improve.

Of the 21 combined control and spontaneously improved patients with psychosis from six to 18 months duration, none recovered, two (9.5 per cent) were much improved, five (23.8 per cent) improved and 14 (66.6 per cent) did not improve.

Among the 49 combined control and spontaneously improved patients with psychosis of over 18 months duration none recovered, four (8.1 per cent) were much improved, six (12.2 per cent) improved and the rest of the 39 (79.7 per cent) failed to improve.

To sum up it can be stated that both treated and control patients with shorter duration of psychosis showed the highest percentage of various degrees of improvement combined. The patients considered as "recovered" constituted the smallest part while those considered as "improved" made up the bulk of the group.

Relapses in Relation to Duration of Psychosis.

Since a number of both control and treated patients relapsed the question arose whether there is any relation between the duration of psychosis and the occurrence of relapses (Table 3). Among the 17 relapsed treated patients two had a psychosis of less than six months, six patients had a psychosis of between 6 and 18

months' duration and nine patients had a psychosis of over 18 months' duration.

Of the five relapsed control patients one had a psychosis of less than six months, two from 6 to 18 months and two over 18 months' duration.

Among the eight relapsed combined control and spontaneously improved patients three had a psychosis of less than six months, two between 6 and 18 months and three over 18 months' duration. These figures show a greater occurrence of relapses among patients with longer duration of psychosis.

TABLE 3.

RELAPSES IN RELATION TO DURATION OF PSYCHOSIS.

	Treated.	Controls.	Controls and spontaneously improved.
Less than 6 months	2	1	3
6 to 18 months	6	2	2
Over 18 months	9	2	3
Totals	17	5	8

DURATION OF REMISSIONS IN RELATION TO DURATION OF PSYCHOSIS.

Of the two relapsed treated patients with a psychosis of less than six months' duration one remained in the community about 11 months and the other less than six months (Table 4). Among the six relapsed treated patients with a psychosis of from 6 to 18 months one remained outside 11 months, another nine months, two seven months and two less than six months. Of the nine relapsed treated patients with psychosis of over 18 months two remained in the community about 11 months, one nine months, two seven months and four less than six months.

Among the three relapsed control and spontaneously improved patients with psychosis of less than six months one remained outside nine months and two others less than six months. Of the two patients with psychosis of between 6 and 18 months one remained in the community about 11 months and the other seven months. Of the three patients with psychosis over 18 months' duration one

remained in the community 11 months, one nine months and one less than six months.

There are 18 treated patients still in the community (Table 5). Of four patients with a psychosis of less than six months three have been out of the hospital for periods ranging from a year to two years and the fourth 11 months. Among 10 patients with a psychosis of 6 to 18 months' duration seven have been out for periods of a year to two years and three for periods ranging from 8 to 10

TABLE 4.

RELAPSES AFTER TREATMENT.

Duration of psychosis.	Duration of remission.				
	1 yr. to 2 yrs.	10 mos. to 12 mos.	8 mos. to 10 mos.	6 mos. to 8 mos.	Less than 6 mos.
Less than 6 mos.....	0	1	0	0	1
6 to 18 mos.....	0	1	1	2	2
Over 18 mos.....	0	2	1	2	4
Totals	0	4	2	4	7

RELAPSES OF CONTROL AND SPONTANEOUSLY IMPROVED PATIENTS.

Duration of psychosis.	Duration of remission.				
Less than 6 mos.....	0	0	1	0	2
6 to 18 mos.....	0	1	0	1	0
Over 18 mos.....	0	1	1	0	1
Totals	0	2	2	1	3

months. Of four patients with psychosis of over 18 months' duration one has been out nine months, another seven months and two less than six months.

Among 20 still improved control and spontaneously improved patients 10 had a psychosis of less than six months. Eight of them have been out of the hospital for periods ranging between a year and two years, one 11 months and one less than six months. Four patients with a psychosis between 6 and 18 months have been out for periods of a year to two years. Of six patients with psychosis of over 18 months' duration two have been in the community from a year to two years, one 11 months, one nine months, one seven and one less than six months.

Considering these figures it can be stated that both the treated and control patients with shorter duration of psychosis have the longest remissions as measured by their residence outside of the hospital.

TABLE 5.

TREATED PATIENTS STILL IN COMMUNITY.

Duration of psychosis.	Duration of remission.				
	1 yr. to 2 yrs.	10 mos. to 12 mos.	8 mos. to 10 mos.	6 mos. to 8 mos.	Less than 6 mos.
Less than 6 mos.....	3	1	0	0	0
6 to 18 mos.....	7	0	3	0	0
Over 18 mos.....	0	0	1	1	2
Totals	10	1	4	1	2

CONTROL AND SPONTANEOUSLY IMPROVED PATIENTS STILL IN COMMUNITY.

Duration of psychosis.	Duration of remission.				
Less than 6 mos.....	8	1	0	0	1
6 to 18 mos.....	4	0	0	0	0
Over 18 mos.....	2	1	1	1	1
Totals	14	2	1	1	2

SUMMARY OF CONCLUSIONS.

A group of 100 patients of the predominantly chronic type of schizophrenia, equally divided by sex, underwent a course of hypoglycemic shock treatment while 69 patients—36 men and 33 women—were observed as controls. Fifteen other patients, eight men and seven women have improved spontaneously while in preparation for treatment. The report is based on the state of the patients eight months to two years after termination of treatment or control observation.

Various degrees of improvement took place in 36 per cent of the treated patients, a lower percentage than generally reported.

The percentage of improvement in the control group was 21.7.

The percentage of improvement in the combined control and spontaneously improved group was 35.7 or about the same as in the treated group.

Relapses took place in 47 per cent of the treated patients, in 33.3 per cent of control cases and in 24 per cent of the combined control and spontaneously improved patients.

The treatment was the cause of death in 3 per cent of cases.

"Recovery" occurred in only slightly over 7 per cent among treated patients with psychosis of not more than 18 months' duration.

The same rate of "recovery" took place among the combined control and spontaneously improved patients with psychosis of less than six months' duration.

The percentage of "improved" ranged from 75.0 to 21.5 in the treated group, from 75.0 to 8.8 in the control group and from 57.0 to 12.2 in the combined control and spontaneously improved group, in proportion to the duration of the psychosis.

"Lack of improvement" ranged from 12.5 per cent to 78.5 per cent in the treated group, from 25.0 per cent to 86.8 per cent in the control group and from 7.2 per cent to 79.7 per cent in the combined control and spontaneously improved group, in proportion to the duration of the psychosis.

The duration of the psychosis in the improved treated, improved control and in the spontaneously improved patients was shorter than in the unimproved patients.

The highest percentage of relapses occurred among patients with psychosis of longer duration.

Both treated and control patients with psychosis of shorter duration had the longest lasting remissions.

GLUCOSE AND INSULIN TOLERANCE: RELATION TO INSULIN SHOCK TREATMENT.*

By MAURICE KAPLAN, M. D., and A. A. LOW, M. D., Chicago, Ill.

Introduction.

The present paper purports to study: (1) the carbohydrate metabolism in psychotic patients as reflected in the glucose and insulin tolerance tests; (2) possible correlations between the results of these tests and various aspects of insulin therapy, specifically the coma dose, the pre-coma length and the blood sugar during treatment.

Twenty-six patients were used for the first part of this study. Twenty-three of the cases were classified as dementia præcox and three as manic-depressive psychoses. Each patient was given an insulin tolerance and glucose tolerance test on different days. Ten of the patients with dementia præcox were then given insulin shock therapy. Most of the remaining patients also received insulin therapy, but since this was preceded by other treatments, such as metrazol or fever, they were not included in the second part of this study.

Various authors have estimated the amount of sugar in the blood during fasting and at one-half, one, two and three hours after the administration of sugar. Curves have been analyzed with respect to the successive rise and fall of the glucose values and the length of time required for a return to the fasting level. Pollock and Boshes [1] quoted Mosenthal's classification of curves as normal when the rise was between 120 and 160 mg., high when the rise was a peak of 160 mg., prolonged when the curve failed to drop below 120 mg. at the end of the second hour, high prolonged when the curve rose above 160 mg. and failed to return to 120 mg. at the end of the second hour, and low when the curve did not rise above 120 mg. The consensus of opinion, they said, is that a normal curve is one that starts at a fasting level below 120 mg.,

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rises to a peak not above 160 mg. and returns to the fasting level in less than two hours. There is a great variability of curves in normal subjects. In 286 normal subjects John [2] found 25.8 per cent with "diabetic" curves. The significance of the variability of curves in normal subjects is still a matter of speculation.

Kasanin,[3] in a study of 33 cases of dementia præcox, obtained normal curves in 11, the remainder showing abnormal curves. After a review of the literature, he found that the average curve for patients of this type is well within normal limits, though the percentage of abnormal curves is higher than in healthy subjects. He did not find any characteristic curve for patients with dementia præcox. Lorenz,[4] studying cases of dementia præcox and other mental disorders, found that the reaction of mentally ill patients to glucose feeding is generally within normal limits.

In the study here reported each patient was given one glucose tolerance test. One patient had two tests. Glucose was given orally, the solution consisting of 100 grams of glucose with 78 cc. of lemon juice and 182 cc. of water. It was administered in the morning after a fasting specimen of blood had been drawn. Subsequent samples of blood were drawn from the cubital vein at one-half, one, two and three hours after administration. In making the determinations, the Folin-Wu filtrate [5] was used in the Somogyi modification [6] of the Shaffer-Hartman method.[7] The standards set by Mosenthal were used in evaluating the results. The curves were considered normal if the blood sugar level had returned to below 120 mg. by the end of the second hour.

The fasting level was found to range from 54.1 to 121.7 mg., with an average value of 90.9 mg. The peak of the rise in blood sugar ranged from 76.6 to 249.6 mg., the maximum rise from the fasting level ranging from 33.8 to 142.1 mg. The peak of the rise was reached in one-half hour in eight cases, by the first hour in 12 cases, at the end of the second hour in five cases, and not until the third hour in one case. In three cases the curve did not reach 120 mg. In one of these cases the test was repeated, but a low curve was again obtained. In 14 cases a high curve was obtained, the blood sugar rising above 160 mg. In nine of these, the blood sugar had not yet fallen to 120 mg. or less by the end of the second hour. In one case the blood sugar had fallen to 114.2 mg. by the second hour, but at the third hour had again risen to 137.6 mg.

In four of the nine cases the blood sugar was still above 120 mg. at the end of the third hour. Of the remaining nine cases, in which the blood sugar rose to between 120 and 160 mg., six showed a decline to below 120 mg. by the second hour, two reached this level by the third hour, and in one case the curve was still rising at the third hour. The average curve for all the cases showed a rise to 151.6 mg. at the first hour, with a return to below 120 mg. at the third hour.

It is apparent that in this series of cases there is a preponderance of abnormal curves, only six falling within the normal range. The largest number of curves were high, with the high prolonged variety predominating.

Insulin Tolerance Test.

Few studies of the insulin tolerance test in the psychoses have been reported. To my knowledge, the insulin tolerance test has not been studied in the "functional" psychoses. However, sufficient work has been done with normal subjects to permit the assumption of a norm in response. The insulin was injected subcutaneously, intravenously or intramuscularly. It was given in varying amounts, both irrespective of and proportional to the body weight.

McCormick, MacLeod, Noble and O'Brien, as quoted by Pollock and Boshes,[1] found that the extent of reduction of the sugar content does not follow the dose per kilogram or correspond to the actual level of the blood sugar before the injection. Neither does the mode of injection, intramuscular or intravenous, make any significant change in the steepness of the curve. Bodansky and Simpson,[8] using the intravenous route, found that the lowest level of blood sugar occurred about 18 minutes after the injection. The original level was regained within 75 minutes. The fall was twice as great in under-nourished subjects as in obese persons. Sendrail [9] found in 16 normal persons that the drop in blood sugar averaged 24 mg. and that the return to the fasting level occurred within an average of 65 minutes and not once with an interval of over 90 minutes. Meyerthaler and Kleineidam [10] injected 7 units of insulin intravenously in normal subjects and found that the blood sugar fell to the lowest point in 19 minutes but returned to the original level after 60 minutes.

The preceding studies of normal persons suggest that, whether the insulin is given subcutaneously or intravenously, the greatest fall is noted within the first half-hour and the original level is reached in less than three hours.

In the present investigation insulin tolerance tests were made of the same 26 patients who were subjected to the glucose tolerance tests. The dose was uniformly 10 units, given subcutaneously after a fasting blood specimen had been drawn. Samples of blood were then drawn from the cubital vein at one-half, one, two and three hour intervals. The method used for the determinations was the same as in the glucose tolerance test.

The fall in blood sugar ranged from 12.5 to 66.6 mg., with an average of 40.4 mg. (Table 1). In three of the patients the maximum fall was reached within a half-hour. The original fasting level was regained by the second hour in one of them. In one instance the curve rose to within 11 mg. of the original fasting level, and in the third case the curve remained only slightly above the lowest level reached. In eight cases the maximum drop was reached by the first hour. In only one of these did the blood sugar return to the original fasting level by the third hour, and in two more it returned to within 10 mg. of that point. In seven cases the lowest point of the curve was reached at the end of the second hour. In only one case was the original level attained by the third hour. In the remaining eight cases the maximum fall in blood sugar was reached by the third hour.

These results indicate a significant difference in the response to the insulin tolerance test in these patients as compared with normal subjects. This difference is chiefly in the prolongation of the period required for the return to the fasting level. Since in the fasting animal the blood sugar level is restored by the action of adrenalin in mobilizing glycogen from the liver, one would suspect a defect in some phase of this mechanism.

A comparison of the glucose and insulin tolerance curves in the same patients did not reveal any significant correlations. A high curve in the glucose tolerance test may be associated with a deep or shallow curve in the insulin tolerance test, or a low sugar curve may be associated with a deep insulin curve.

TABLE 1.

COMPARISON OF MAXIMAL VALUES IN THE GLUCOSE AND INSULIN TOLERANCE TESTS.

	Glucose tolerance.			Insulin tolerance.		
Patient.	Maximum rise in B. S., mgm.	Hours.	Return to normal level.	Maximum fall in B. S.	Hours.	Return to fasting level.
A. T.	112.7	1	Third Hour	63.6	3	..
E. C.	73.2	1	Second hour	47.4	½	..
A. D.	137.6	1	.....	66.0	1	3
C. K.	36.1	½	Low curve	45.6	1	..
S. C.	49.4	½	Low curve	18.0	½	2
L. M.	36.8	2	Low curve	54.8	1	..
V. B.	41.3	1	Second hour	37.4	3	..
E. Z.	87.3	½	Third hour	35.5	2	..
B. D.	141.0	1	.....	30.0	½	..
L. W.	61.6	1	Third hour	26.3	1	..
A. W.	132.3	2	.....	33.9	3	..
E. D.	68.1	3	.....	35.0	3	..
J. N.	111.0	½	Third hour	49.9	3	..
E. S.	65.9	1	Second hour	38.2	2	..
H. R.	87.1	1	Second hour	41.3	3	..
R. R.	36.4	2	Third hour	29.3	3	..
E. B.	47.0	1	Second hour	26.9	1	..
B. L.	123.4	2	Third hour	39.1	2	..
Z. L.	138.5	½	First hour	29.3	1	..
V. W.	142.1	½	Second hour	12.5	2	..
G. G.	90.6	½	.....	49.7	3	..
T. W.	50.6	1	Second hour	37.6	1	..
R. F.	41.6	1	Second hour	55.4	2	3
A. B.	57.5	1	Third hour	58.6	1	..
M. C.	33.8	2	Third hour	47.4	2	..
M. H.	76.0	½	.....	44.0	2	..
Average	79.9	..	.....	40.4	..	..

RELATION OF GLUCOSE AND INSULIN TOLERANCE TESTS TO THE AVERAGE COMA DOSE.

Comparisons were made between the highest blood sugar value in the glucose tolerance test, the lowest blood sugar value in the insulin test and the average coma dose required in the 10 treated cases. That the number of cases is insufficient for valid inference is fully understood.

Treatment was usually begun with an initial dose of 20 or 30 units. The dose was increased daily by from 10 to 20 units until coma was obtained. Reduction of dosage is a well-known technique and needs no mention. Coma was arbitrarily set as that point in

TABLE 2.

THE RELATIONSHIP OF THE AVERAGE COMA DOSE TO THE GLUCOSE TOLERANCE TEST.

Patient.	Average coma dose.	Fasting.	½ hour.	1 hour.	2 hours.	3 hours.
L. W.	32 u.	107.5	131.5	169.1	152.8	88.7
E. Z.	86 u.	112.7	200.0	187.9	123.3	97.7
V. B.	89 u.	90.2	98.4	131.5	90.2	75.0
L. M.	90 u.	80.4	81.6	79.1	117.2	112.0
B. D.	109 u.	106.0	203.0	247.0	177.0	147.0
S. C.	161 u.	64.8	114.2	105.2	80.4	64.0
A. D.	176 u.	112.0	203.0	249.6	210.5	157.8
A. T.	180 u.	84.9	161.6	197.6	130.0	119.5
C. K.	252 u.	80.4	116.5	103.0	105.2	100.8
E. C.	272 u.	97.9	122.5	171.1	106.7	87.3

TABLE 3.

THE RELATIONSHIP OF THE AVERAGE COMA DOSE TO THE INSULIN TOLERANCE TEST.

Patient.	Average coma dose.	Fasting.	½ hour.	1 hour.	2 hours.	3 hours.
L W.	32 u.	78.9	63.9	52.6	54.1	52.6
E. Z.	86 u.	82.3	53.3	48.1	46.8	48.1
V. B.	89 u.	97.7	67.1	67.8	63.3	60.3
L. M.	90 u.	127.7	86.3	72.9	81.2	93.2
B. D.	109 u.	86.0	56.0	56.0	75.0	75.0
S. C.	161 u.	100.7	82.7	96.2	105.2	101.1
A. D.	176 u.	108.0	91.0	42.0	68.0	108.0
A. T.	180 u.	101.5	54.1	56.8	41.8	37.9
C. K.	252 u.	111.0	93.7	65.4	65.4	72.9
E. C.	272 u.	84.9	37.5	42.8	51.1	45.0

the hypoglycemic reaction at which the patient failed to respond to pin pricks and auditory stimuli.

No correlation was found between the highest sugar level in the glucose tolerance test, the lowest level in the insulin test and the average coma dose (Tables 2 and 3). In one case the highest sugar level in the glucose tolerance test was 116.5 mg. and coma was

obtained only after the use of an average coma dose of 252 units. In another case in which the blood sugar in the glucose tolerance test reached 200 mg., an average dose of 86 units was sufficient to produce coma. In this case the dose was gradually reduced to 70 units and coma was still produced. The same was true in regard to the insulin tolerance test. One case which reached a level of 42.8 mg. in the insulin tolerance test required an average dose of

TABLE 4.

BLOOD SUGAR LEVELS DURING HYPOGLYCEMIA: RELATIONSHIP TO PRE-COMA LENGTH.

Patient.	Fasting.	1st hour.	2d hour.	3d hour.	4th hour.	½ hour after glucose.	Pre-coma length on day of blood examination. Hours.	Minutes.
A. T.	61.0	47.3	31.9	19.0	18.0	40.6	3	13
E. C.	105.2	37.5	35.3	17.2	15.7	118.0	3	35
A. D.	95.8	20.3	24.0	21.0	31.5	72.1	2	50
C. K.	103.0	45.1	41.3	22.5	22.5	148.0	3	..
S. C.	120.0	37.5	27.8	45.1	36.8	106.7	3	40
L. M.	71.5	33.8	20.3	24.8	27.0	62.4	2	30
V. B.	82.7	47.3	24.0	24.8	16.5	97.7	3	5
E. Z.	80.1	21.8	21.8	32.3	20.3	96.2	4	40
B. D.	95.4	65.4	61.6	37.8	15.0	....	2	54
L W.	92.7	30.0	20.3	20.3	....	92.7	2	30
Average ...	96.4	37.5	30.0	26.1	25.2	98.6	..	..

272 units, while another that reached a level of only 60.3 required 89 units. On the other hand, a case that reached a level of 52.6 mg. in the insulin tolerance test required only 32 units.

THE RELATION OF THE BLOOD SUGAR LEVEL TO THE ONSET OF COMA.

Low content of blood sugar is apparently in itself not the sole factor in producing coma. According to Freudenberg,[11] the earlier, the more rapid and the greater the fall in the actual blood sugar the earlier do the symptoms of hypoglycemia appear. In his patients, coma began only when the blood sugar values had been below 30 mg. per cent for at least one hour and 20 minutes. From the results obtained in the 10 cases studied here (Table 4),

Freudenberg's estimation of the time necessary for the maintenance of low blood sugar is, if anything, conservative and by no means constant. In three of our cases a blood sugar content below 30 was reached during the first hour. In two of these, coma was reached in less than three hours after the injection of insulin, and in one, coma was attained after four hours and 40 minutes. In four cases this minimum fall in the blood sugar was reached at the end of the second hour. In all except one, coma was reached only after more than three hours of treatment. In the one exception, coma was reached in two hours and 30 minutes, but it should be noted that the sugar reading at the end of the first hour was 33 mg. per cent. One exception to the general principle was seen in that one patient apparently reached coma before the blood sugar dropped below 30 mg. per cent. It may, therefore, be concluded that the figure of 30 mg. per cent is arbitrary and that the chief factor is the length of time a person is allowed to remain with a significant fall in blood sugar. Cases have been reported, however, in which the patients failed to go into coma even when the sugar level had fallen to a point where it could not be determined and was allowed to remain at this point for three or more hours. It would seem, therefore, that besides the rapidity of fall and the length of time a significantly low blood sugar is maintained, some other factor is involved.

Conclusions.

1. Though the average blood sugar curve in 26 patients given a glucose tolerance test was within normal limits, only six of the individual curves were within normal limits.

2. The blood sugar curve obtained in the insulin tolerance test in these 26 patients was characterized by a marked prolongation of the time required for a return to the fasting level.

3. There was no correlation between the insulin and glucose tolerance tests and the average coma dose.

4. The onset of coma was dependent not only on a significant fall in the blood sugar level, but also on the length of time the low level was maintained.

BIBLIOGRAPHY.

1. Pollock, L. J., and Boshes, B.: Carbohydrate metabolism in epilepsy. Arch. of Int. Med., 59: 1000-1023 (June), 1937.
2. John, H. J.: A study of eleven hundred glucose tolerance tests. M. J. and Rec., 131: 287, 351, and 398, 1930.
3. Kasanin, J.: Blood sugar curve in mental disease (schizophrenia). Arch. Neurol. and Psychiat., 16: 414 (Oct.), 1926.
4. Lorenz, W. F.: Sugar tolerance in dementia præcox and other mental disorders. Arch. Neurol. and Psychiat., 8: 184 (Aug.), 1922.
5. Folin, O., and Wu, H.: A system of blood analysis. J. Biol. Chem., 38: 81, 1919.
6. Somogyi, M.: Reagent for the copper iodometric determination of very small amounts of sugar. J. Biol. Chem., 117: 771, 1937.
7. Schaffer and Hartman: The iodometric determination of copper and its use in sugar analysis. J. Biol. Chem., 45: 365, 1920-21.
8. Bodansky, A., and Simpson, S.: Blood sugar of normal human subjects after the intravenous injection of insulin. Quart. J. Exper. Physiol., 17: 57, 1927.
9. Sendrail, M.: L'épreuve de tolérance a l'insuline. Ann. de med., 27: 289, 1930.
10. Meyerthaler, F., and Kleineidam, E.: Blood sugar following the intravenous administration of insulin. Arch. f. Exper. Path. u. Pharmakol., 178: 315-319, 1935.
11. Freudenberg: The behavior of the actual blood sugar during insulin shock. Am. J. Psychiat., May, 1938.

METRAZOL REACTION IN PATIENTS WITH ARTERIAL HYPOTENSION.*

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AND
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A 41-year-old female was admitted in April, 1938, to this sanatorium. Since 1933 she had been suffering from a paranoid schizophrenia with remissions and exacerbations. On admission her blood pressure was 90 systolic and 74 diastolic, pulse rate was 80, otherwise her physical condition was normal. During the following seven weeks she had 19 convulsions, produced by metrazol injections, in amounts ranging from 3 cc. to 7 cc. By the end of the treatment she had made a satisfactory social recovery which has continued until now (four months).

Each metrazol convulsion was followed by a reaction which we have not seen described in the literature, and which we ourselves have only encountered once before. After the usual tonic and clonic stages and after breathing had been resumed again, her skin became ashen in color. The pulse did not appear after relaxation from the convulsion. Then respiration ceased and the patient lay completely motionless and atonic. From the end of the convulsion a period of 20 to 73 seconds elapsed after which the patient ceased breathing. This period of apnea lasted from 19 to 25 seconds. In all instances artificial respiration was applied. During this whole time, that is from 40 to 105 seconds, the pulse was not palpable. Occasionally faint, irregular heart sounds could be heard. The carotid pulse could be perceived a few seconds before the radial. After her recovery from the collapse the blood pressure was usually a few points higher than before. During the entire treatment the systolic blood pressure ranged between 68 and 100.

A second patient who showed the same reaction (lack of pulse and apnea) also had low blood pressures ranging from 90 to 118.

Our material suggests strongly that the post-convulsive syndrome of cessation of pulse beat and respiration was caused by the poor vasomotor condition, as evidenced by the low blood pressures in our two cases. Although the picture is an alarming one, it is possible to carry the treatment to completion.

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CASE OF SUBCLINICAL PELLAGRA—PSYCHOTIC TYPE—TREATED SUCCESSFULLY WITH CORAMINE (NICOTINIC ACID).

By MAX H. WEINBERG, M. D., Pittsburgh, Pa.

The recent advance in the treatment of pellagra with nicotinic acid may prove of great help in some hitherto obscure mental cases. Furthermore, the therapy, if successful, may help in establishing the diagnosis. Recently the author had such an experience, which is thought worth while to report.

Mrs. G. R., 58, housewife. Admitted to St. Francis Hospital Nov. 29, 1938.

On admission, the relatives stated that for the past month, the patient refused to eat, that she mumbled to herself and talked to imaginary people. She also became suspicious of every one around her and was very agitated. She expressed the fear that her friends and neighbors were going to harm her.

The significant facts in the past medical history were that she had had serious stomach trouble 20 years before and that she was treated for a similar condition in another hospital a few months before.

Her father died at the age of 58 with stomach trouble and nervousness.

Physical examination revealed tortuous temporal and brachial arteries and a blowing systolic murmur at the apex which was transmitted to the aortic area. The blood pressure was 220/158, and there was marked trophic disturbance over both legs and feet.

Neurologically, the patient presented a doubtful Babinski sign on the left, tremor of the hands and enlarged thyroid. Temperature never exceeded 100, and the pulse never was over 110.

Blood chemistry and B.M.R. were essentially negative. The W.B.C. on 12/18/38, when the patient was very much disturbed, were 18,700.

Mentally, the patient was confused, mumbling constantly, but could be veered back into a relevant conversation with the examiner, although this did not last very long. She harped on the fact that she is noisy, that "everybody knows it," and that "all is lost," and then started again to talk unintelligently. The patient had definite visual and auditory hallucinations and mild persecutory delusions. She had some insight into her condition and feared that she was "crazy."

The condition became progressively worse. She perseverated in a manner which almost amounted to "word-salad" of the præcox type. Later the perseveration became obsessive in character and fixed on but one Yiddish word "Katchke" (meaning duck). This word would be interspersed several

times in each sentence. The patient's confusion grew more pronounced. Nourishment was absolutely refused.

On 12/20/38, about three weeks after admission, the patient was found in almost complete collapse. Coramine was ordered as a cardiac stimulant. Altogether she was given four ampules, 12 cc., of coramine in three days.

The results were dramatic. In 24 hours the patient showed remarkable improvement, and within about a week she was restored to normal. She took care of all her own needs and ate without supervision. She was discharged Jan. 7, 1939, fully recovered.

The diagnosis of subclinical type of pellagra was made in retrospect as a result of the patient's quick and dramatic response to coramine (diethylamide of nicotinic acid) therapy.

DISCUSSION.

The writer is frank to admit that the case puzzled him greatly, and that the diagnosis was not arrived at until the successful outcome following the administration of coramine, which was given as a cardiac stimulant when the patient was in utter collapse. The patient's recovery was as sudden as it was dramatic, and is believed to be attributable to the use of the nicotinic acid.

In retrospect it was not difficult to establish the diagnosis. The anorexia and the constant refusal of food caused a subclinical type of pellagra. At first the mental symptoms were rather mild; but as the condition progressed, she became more and more confused, refused nourishment altogether, and presented the picture of apparent deterioration. On the day when coramine was resorted to, the patient was practically moribund.

Other writers who treated many cases of frank pellagra use practically the same glowing terms in describing their successful therapeutic results with nicotinic acid. Smith, Ruffin and Smith [1] say: "The results of this treatment were dramatic**; and in their summary they state: "A patient who had endemic pellagra with anorexia, dermatitis, sebaceous gland changes and dementia made a dramatic recovery after the administration of nicotinic acid in doses of 60 mg. daily for 12 days." Spies [2] and his co-workers reported on 73 cases of pellagra and in addition on 199 cases with subclinical pellagra. In discussing their observations, they say: "Within the first 24 hours the picture of apathy and lassitude gave way to one of wide-awake interest."

As is usual with new remedies, the question of dosage becomes a bone of contention and this medication proves no exception. In the discussion that followed the presentation of his paper by Spies, the difference of opinion was rather marked regarding the amount of the medication to be used. Spies speaks of 500 mg. a day in divided doses, and coramine 4 to 20 cc. daily. On the other hand, Ruffin,[3] in discussing Spies' paper, says: "I have found that small doses of nicotinic acid (1.5 mg. per kilogram of body weight, from 70 to 100 mgm. daily) result in prompt recovery and are as affective as doses 10 times as large in the average case of acute pellagra." Cleckley, Sydenstricker and Geeslin [3] generally used 100 to 150 mg. of nicotinic acid daily for only several days. The author used the smallest dose possible, only 12 cc. of the coramine in four days, and it proved sufficient to restore the patient to health. It would seem that the stand that Ruffin takes, namely that small doses of nicotinic acid are just as effective as large doses, is correct, and should be followed. The dose can always be increased if smaller doses are found inadequate.

Cleckley and his co-workers in what is the most recent article on the subject of nicotinic acid in atypical psychotic states point out that the acid can be used as a therapeutic test. After discussing the excellent results they obtained in 19 atypical psychotic cases, they say: "We have, however, been impressed by the dramatic improvement obtained in this series of cases and we are convinced that a therapeutic test with nicotinic acid is justifiable in cases of unexplained hebetude or unconsciousness." The writer can only add from his experience in this case that nicotinic acid may be used safely as a therapeutic test in such cases of obscure psychosis especially when one or more symptoms closely related to pellagra, such as anorexia, gastric disturbance, dermatitis, etc., are present.

CONCLUSIONS.

1. A case of atypical psychosis is presented.

2. Diagnosis of subclinical type of pellagra—mental type—is made retrospectively after coramine (nicotinic acid) therapy resulted in a dramatic recovery.

BIBLIOGRAPHY.

1. Smith, D. T., Ruffin, J. M., and Smith, S. G.: Pellagra successfully treated with nicotinic acid. A case report. J. A. M. A., 109: 2054-2055, Dec. 18, 1937.
2. Spies, T. D., Bean, W. B., and Stone, R. E.: The treatment of subclinical and classical pellagra. J. A. M. A., 111: 584-592, Aug. 13, 1938.
3. Cleckley, H. M., Sydenstricker, V. P., and Goesslin, L. E.: Nicotinic acid in the treatment of atypical psychotic states. J. A. M. A., 112: 2107-2110, May 27, 1939.

RECONSTRUCTION DREAMS.

By MAX LEVIN, M. D., Baltimore, Md.

This paper deals with those dreams in which a succession of events culminates, as if by a marvellous coincidence, in a stimulus coinciding with an external stimulus of like nature which wakes the sleeper. In a celebrated example a man dreamed he was taking part in the French Revolution; he was arrested, tried and sentenced to the guillotine; he was then led to the scene of the execution, his head was laid on the block, and he felt the impact of the knife on his neck, at precisely which moment he was wakened by a piece of plaster which dropped from the ceiling and struck him in the neck at the exact point struck by the knife in the dream.

The following example is from my own experience. I dreamed I was watching a baseball game. I saw the pitcher "winding up" and saw the ball leave his hand and travel toward the batter. I then saw the batter swing his bat, and heard a loud report as bat met ball. Precisely at that moment I was wakened by a sharp report made when the window shade, because of a defective spring, flew up and struck the top of the sash.

In this paper I shall propose an explanation of such dreams, which, as far as I know, have never been given a name. Later the reader will see why I speak of them as "reconstruction dreams."

Previous writers have paid little attention to reconstruction dreams, regarding them as curiosities of no very great significance. My purpose here is to show that, insignificant as they may be from the semeiological standpoint, they throw light on a most important problem in the physiology of the highest cerebral centers. I shall try to show that certain views I have recently set forth on the pathogenesis of hallucinations—views derived from Hughlings Jackson's theory of the nature of the physical basis of imagery—illuminate the mechanism of these otherwise hardly explicable dreams.

Adopting Jackson's terminology, I shall speak of vivid and faint images (a vivid image occurring when, for example, we see an

object, a faint image when we merely think of or visualize that object). The expression "substrate of an image" will be used to designate the pathways activated when we have a particular image. For example, in the sphere of visual imagery, the "substrate of the vivid image 'brick'" refers to the pathways activated when we *see* a brick; the "substrate of the faint image 'brick'" refers to those activated when we merely *think* of a brick. The substrate of any given *vivid* image is spread out over all levels of the nervous system, lowest as well as highest. The substrate of the corresponding *faint* image is confined to the highest levels; it consists of that part of the vivid image substrate which lies within the highest cerebral centers (Jackson,[1] Vol. II, pp. 69-70).

Consideration of the content of hallucinations seems to suggest that the *hallucination* (vivid image) of a person, asleep or psychotic, corresponds in content to the *thought* or *phantasy* (faint image) he would have had if at the moment in question he had been awake and sane. Examples were given in a previous paper,[5] of which the following is given here in illustration. MacCurdy,[7] in his study of war neuroses, reported that the dreams of soldiers at the front "have a purely war content, the setting of which is, as a rule, the section of the line in which the patient has last been, or that section in which he may have been subjected to most severe strain. The exact nature of the injuries which seem imminent in the dreams naturally varies with the type of fighting in which the man has been engaged. The enemy is throwing bombs at him, which explode at his feet, he is about to be bayoneted, he is shot down in an aeroplane or shells are raining upon him." Here we see with crystal clearness the relation between diurnal phantasy and nocturnal hallucination. The soldier during the day *thinks* of the shells, etc.; in his dreams he *sees* them.

Why should the sleeper see what during the day he merely thinks of? The answer has been discussed in previous papers [2, 3, 4] and here it will be stated only briefly. Referring to Jackson's views, cited in the last paragraph but one, we may say that when we have a vivid image there is activation of all levels of the nervous system, from lowest to highest. When, on the other hand, we have a faint image, activation is confined to the highest centers; at such a moment the highest centers are functioning independently of lower centers. Now one measure of the evolution of a nervous center

is its ability to function independently of the rest of the nervous system. Thus an infant just learning to toddle is unable to walk save when he puts his mind to it. His lower "walking centers" are not well enough organized to be able to function automatically, but "require the assistance of" the highest centers. As the child grows older and more practiced, the walking centers become better and better organized until at last they can function without assistance from above; he now can walk even when his mind is on something else. To take the opposite kind of example, a two year old child lacks the ability to think silently; the thoughts which pass through his mind are reflected in a continual chatter of words. Here it is the *highest* centers which are unable to function independently of *lower* centers. In time the child reaches the point at which he can, when necessary, think without speaking. At this point there is almost complete independence of the highest centers (not entirely complete, because even in silent thought there are rudimentary movements of the larynx). To return to the problem of imagery, one of the characteristics of the adult is that he can have faint images with ease, *i. e.*, he is capable of representation. The child, by contrast, is capable of perception but relatively incapable of representation. Thus in respect to imagery, as in respect to speech, we find that growth implies the evolution of the highest centers from a stage at which they can function only in association with lower centers to one at which they can, when necessary, function in isolation. When the highest centers have reached this advanced stage of evolution, they may, as in sleep and in the psychoses, return to the more primitive stage. Hence a stimulus which, in the alert adult, would activate the substrate of a *faint* image (a substrate lying entirely within the confines of the highest cerebral centers) will, in the sleeper, activate the substrate of the corresponding *vivid* image (a substrate spread out over all levels of the nervous system). For example, a soldier in the trenches, constantly thinking of the dangers that beset him, visualizes the enemy advancing with a bayonet; in other words, the external situation constitutes a stimulus to the substrates of certain faint images, and these substrates are in a state of more or less constant excitation. When he falls asleep he continues to react to the situation; the substrates of those images continue to be stimulated, but now they can no longer function as the substrates of *faint* images (since

the highest cerebral centers, reduced during sleep to a primitive condition, can no longer function independently of lower centers); there is concomitant activation of lower centers as well, and what was during the day the substrate of a faint image is now, during sleep, but the topmost part of the substrate of a vivid image.*

Reconstruction dreams may now be considered in the light of the foregoing views. A stimulus may cause one to think of certain past events. For example, a detective walking in the woods comes upon the body of a man with a crushed skull; there are footprints, a blunt blood-stained instrument and an empty wallet near the body, etc. Seeing these signs and objects, he pictures to himself a succession of events that will account for their presence: he "reconstructs" the crime. There arises in his mind a series of images corresponding to the events of the crime as he imagines them. These images are faint rather than vivid: the detective *visualizes* the events of the crime, but does not *see* them. In order that he may have faint images, his highest cerebral centers must be in optimum condition. If they were reduced to a condition sufficiently primitive, he would have, not faint, but vivid images. Thus if he were delirious at the moment of coming upon the scene, he might *see* the events of the crime, instead of visualizing them.

Let us see how this applies to my own dream, given earlier in this article. When I am awake, the sharp flick of a window shade may remind me of the report of a baseball bat as it meets the ball. When so reminded, I will visualize the baseball field, the players, the pitcher winding up and delivering the ball, and the batter swinging and meeting it (faint images). But when I am asleep the same stimulus will activate the substrates of the corresponding vivid images: I will *see* the pitcher throwing the ball and the batter swinging his bat.

How may one explain the illusion of antecedence, whereby the dream images seem to antecede the stimulus which wakes the sleeper? This question, involving as it does the appreciation of

* Since the substrate of a vivid image is more extensive than that of a faint image, the occurrence of a hallucination in response to a stimulus which under other circumstances would elicit only a phantasy may be regarded as an example of a "mass reflex." Elsewhere [4,6] I have tried to show that the concept of the mass reflex is important for the highest cerebral centers no less than for lower centers.

time, raises a knotty problem in the physiology of the highest cerebral centers, to which a final answer is not yet possible. To restate the question: since the vivid images of the reconstruction dream are evoked simultaneously (being all evoked in a flash by the stimulus which wakes the sleeper), why do they seem to be experienced serially? As a partial answer, it may be said that (with unimportant exceptions) vivid images can be experienced in no other way. In this respect vivid and faint images differ. A series of faint images may be experienced simultaneously even though they belong in a succession; in other words, events falling into a sequence may nevertheless, by a stretch of the imagination, be imagined simultaneously. An illustration of this was reported by the composer Mozart. Mozart used to compose his music entirely in his mind, and did not start to write a composition down until he had thought it out completely. Referring to his mental grasp of the composition, he said,[8] "Nor do I hear in my imagination the parts (of the composition) *successively,* but I hear them, as it were, all together." Obviously this is possible only with faint, and not with vivid images. One may *think* of two colors simultaneously, but one cannot *see* two colors occupying the same space at the same moment. Thus, besides visualizing this page solid white and visualizing it solid black, one can, by an effort of the imagination, experience both images more or less simultaneously, but it would be impossible to *see* the page both solid white and solid black at the same moment. This difference between faint and vivid imagery is matched, in the sphere of motility, by a similar difference between imagined and actual movement. One may, by an effort of the imagination, think of saying "John" and "Smith" simultaneously, but actually to say them simultaneously is impossible. Or, to take a closer example, the steps of a dance may be imagined simultaneously, but executed only consecutively.

In summary, then, the dreamer of a reconstruction dream may be compared with a detective who, stumbling upon the scene of a crime, "reconstructs" a succession of events that will account for the evidence that meets his eye. A stimulus, which incidentally wakes the sleeper, arouses in his mind a series of images, in the same way that a sharp loud noise may remind one of the impact of bat and ball and may make one think of a baseball scene. Were the sleeper awake at the moment in question, the images would be

faint ("representation"); since he is asleep, they must be vivid ("perception"). Vivid images, unlike faint images, can be experienced consecutively but not simultaneously, and thus arises the paradox of a succession of images seemingly culminating in the sleep-dispelling stimulus but actually evoked by that stimulus.

BIBLIOGRAPHY.

1. Jackson, J. Hughlings: Selected writings. Hodder and Stoughton, London, 1931-1932.
2. Levin, M.: Hughlings Jackson's views on mentation; their value for the psychiatrist. Arch. Neurol. and Psychiat., 30: 848, 1933.
3. ———: On the causation of mental symptoms: an inquiry into the psychiatric application of Hughlings Jackson's views on the causation of nervous symptoms, etc. J. Ment. Sci., 82: 1, 1936.
4. ———: Degrees of automatic action: some psychiatric applications of Hughlings Jackson's concept of "reduction to a more automatic condition." J. Neurol. and Psychopathol., 17: 153, 1936.
5. ———: The pathogenesis of hallucinations and delusions. Arch. Neurol. and Psychiat., 37: 839, 1937.
6. ———: Misunderstanding of the pathogenesis of schizophrenia, arising from the concept of "splitting." Am. J. Psychiat., 94: 877, 1938.
7. MacCurdy, J. T.: War neuroses. Psychiatric Bulletin, 2: 243, 1917.
8. Terry, C. S., editor: Forkel's Life of Johann Sebastian Bach. Harcourt, Brace and Howe, New York, 1920, p. 97.

AMNESIA: A STATISTICAL STUDY.*

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Amnesia or "loss of memory" is frequently used as a term for a hysterical condition in which a person is unaware of his own identity. Each year numerous patients are brought to the psychiatric division of Bellevue Hospital after wandering aimlessly around the streets and giving vague answers when questioned by the police. Examination discloses that neither their names nor addresses can be obtained from them. Many of these cases are ultimately diagnosed as senile dementia, alcoholism, schizophrenia or cerebral concussion; others, however, are classified as hysterical reactions.

Few statistical studies of hysterical amnesia are available, the most noteworthy perhaps being that of Abeles and Schilder.[1] A review of a large number of cases makes apparent certain trends which the present survey is intended to clarify. The material selected comprises 71 cases of hysterical amnesia admitted to the psychiatric division of Bellevue Hospital in 1937. Forty-one patients were male, thirty female; the age distribution ranged from 13 to 66 years; seven in the second decade, twenty-five in the third, twenty-four in the fourth, eleven in the fifth, three in the sixth and one in the seventh.

A survey of the cases brings out features of resemblance which suggest that they readily fall into groups distinguished by certain common traits. One such group is that of the person "disappointed in love." Thus, nine of the 21 unmarried patients developed amnesia immediately after an unfortunate incident in love; seven of these were female, two male; all but one were 26 years of age or less.

Case 1.—A 19-year-old girl walked up to a policeman and asked for help. She did not know her name or address and could give no information about herself. She seemed bewildered and could recall only that she had been walking for a long time and suddenly realized that she did not know who she was or where she was going. In the hospital her demeanor was tired and sad; she spoke little but responded to questions. Her memory began to re-

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turn by degrees almost as soon as she reached the ward. Her address was found in her purse. She admitted that it was familiar but did not know if it was her own. Relatives were notified and it was learned that the patient had been unhappy for weeks because of a love affair and had quarrelled bitterly with the man in question a few hours before being brought to the hospital. The patient recovered her memory almost completely within 24 hours, but she did not recognize this man when he called to see her.

It is noteworthy that amnesic patients who have been married greatly outnumber those that have not, the proportion being 50:21; of the married group, 14 have been divorced or separated. The histories of the other 36 married patients show that marital difficulties occur very frequently and have often reached an acute stage just before the onset of the episode. Such cases likewise form a distinct group.

CASE 2.—A 31-year-old female went up to a policeman and asked him to take her home as she did not know where she lived. In the hospital, she remarked when her wedding ring was pointed out to her, "I must be married." Under the influence of amytal, she told of her marital unhappiness and love for another man. She had finally determined to ask her husband for a divorce and had an appointment to discuss the matter with him. After waiting in vain for two hours for her husband to appear, she wandered off and lost her memory.

Acute financial worries preceding an attack of amnesia are more likely to occur in men, especially in heads of families. Often the patient has been resorting to alcoholism to forget the situation. Of the 71 cases, alcoholism is found in the past histories of 17; the preponderance of alcoholism among men may partially account for the greater percentage of male patients with amnesia.

CASE 3.—A 38-year-old male, married and father of four children, was brought to the hospital in an amnesic state. He had only three cents in his pocket. The patient expressed the belief that he had been worrying about business. "I remember something went wrong." The amnesia cleared up spontaneously and the patient was able to tell of his concern over his impending bankruptcy. In recent months he had been drinking alcohol excessively.

Acute emotional problems could be definitely established in 43 of the 71 cases and were probably present in a greater number. Where acute disturbances were lacking, the primary etiological agent sometimes appeared to be other than psychogenic, as in a group of eight cases which included epilepsy, head injury and post-operative conditions. In such instances, the amnesia showed a resemblance to the confusional states of organic disorders and sometimes recurred periodically.

CASE 4.—A 32-year-old male stopped his automobile in traffic and told a policeman that he did not know who he was or where he lived. By the time he was brought to the hospital, he had completely recovered his memory. He had experienced several such episodes previously, each ushered in with a spell of dizziness. Eight months ago he had bitten his tongue in the course of a "seizure" of some type.

In a considerable number of cases, the question of malingering was raised. Amnesic episodes which last for more than two or three days; the unaccountable disappearance of considerable sums of money; obvious reasons to fear returning home; these are factors which contribute to the suspicion of malingering. Married men constitute the majority of this group.

CASE 5.—A 29-year-old man was brought to the hospital by a policeman who found him wandering aimlessly on Broadway. He complained of a headache but could give no information about himself. Identifying papers were found and the family summoned. After a conversation with his wife and father, he declared that their voices were familiar. Under hypnosis, he told of financial difficulties and quarrels with his wife. To appease her, he wrote a check but was not in a position to cash it. In the meantime, he tried to borrow money but failed in his last attempt on the day of admission to the hospital. He then tore up the check and "lost his memory." According to information from his wife, he had often lied to her in the past and had once concealed for months the fact that he had lost his job, borrowing money from friends to bring home as his "salary." On the day of the amnesic episode, he left a note stating that he would not return until he had cashed the check. In an interview with the father, it was learned that the patient had a similar "nervous breakdown" on a previous occasion.

The differential diagnosis between hysteria and malingering is always difficult; the emotional problems and inadequacies of character of the typical hysterical personality are commonly found in the supposed malingerer. Among our cases, the patient himself never conceded that he had feigned amnesia. The testimony of relatives is not conclusive; they are often sceptical of the amnesia but may be vindictive or interested in concealing the emotional problems of the patient and are themselves inclined to be on the defensive.

A characteristic feature of hysterical amnesia is the apparent futility of the disorder. The patient solves none of his fundamental problems except perhaps to relieve immediate emotional tension or to gain the sympathy of harsh relatives. In this respect, amnesia may show a functional resemblance to fainting but is perhaps even more analogous to the plight of small children who have run

away from their parents and are unable to give information about themselves. Abeles and Schilder remarked that "amnesia is a weak attempt of a weak personality to escape conflicts. It is the fear of being punished by the family, by the father and mother or their representatives, which leads to an escape which does not harm the person too much."

Amnesia is necessarily a mental state which develops while the patient is alone and away from home. Almost invariably the attack occurs while he is walking on the street or riding in some conveyance: in New York, usually the subway. Psychologically, the attitude of the patient represents either a flight from or toward an object. The patient may say that he was looking for a familiar face and may even ask people on the street if they know him. Two characteristic findings in the history of the cases are significant: the patient almost always has been living with close relatives and almost invariably turns up in one of the most populous sections of the city. Another important fact for consideration is that the amnesic picture does not clear up spontaneously when the patient is alone but only after he has been taken into custody.

The great majority of patients included in the present survey were amnesic for not more than a day or two before admission to the hospital. After admission, memory usually returned spontaneously within 48 hours. In more persistent cases, hypnosis is less likely to be helpful than amytal, which is exceedingly useful in obtaining information as to the identity of the patient and in ascertaining details of his activities during the amnesic interval. Although the patient may have marks of identification in his possession, even his name and address, these appear to have but little meaning for him. A woman will remark spontaneously, "I have a wedding ring, I suppose I am married," or "I have a baby's picture in my purse, I don't know if it is my baby." Sometimes memory returns when a relative arrives to identify the patient; at other times the patient will show no sign of recognition but does not object to being discharged in the custody of the "stranger."

The behavior of most patients on admission is depressed and agitated. They may repeat, "I want to go home, I want to go home," or ask, "Please tell me who I am?" Some are very calm and indifferent; others are in a dazed condition. Most patients profess to remember nothing until a few hours previously when they sud-

denly found themselves wandering about the street. Sometimes they have fragmentary recollections, couched in vague terms such as, "I work near a park," "I think I do outdoor work, my hands are rough." They may recall their first names only. Loss of memory may be so complete that the patient cannot tell in which city he is and cannot give the name of the President of the United States. Others reveal superior intelligence and knowledge, solve algebraic equations and perform complicated tasks. Some patients have vague associations which have a bearing on events immediately preceding the onset of amnesia. A girl (Case 1) whose amnesia followed a telephone conversation with her sweetheart, declared that she remembered "something about a telephone" and was visibly upset whenever she heard a bell ring in the wards. There are patients with an uneasy feeling, usually substantiated later, that they were "supposed to meet someone." One girl could recall only "something about a river." Actually she had been on the point of throwing herself into the river, then recoiled and became amnesic. Significant false recollections are reported, as when the mother of a newly born infant states that she has two children when actually there are three. Similarly, a married woman who finally recalls her identity may be able to give only her maiden name.

CASE 6.—A 25-year-old female was admitted in an agitated and depressed state. She said that she had been riding in the subway and "suddenly saw black," felt a sharp pain in her head and could not recall where she was going. All that she could remember was that she had just returned from a trip to Porto Rico and was living in a downtown hotel. She made her way to the hotel and asked if she were registered there. When she found that she was not known in the hotel, she wandered off. Examination in the hospital revealed that the patient knew her maiden name and all events until 1932, five years before, when she returned from Porto Rico and registered at the downtown hotel. Soon thereafter she met and married her husband. When her husband arrived at the hospital to take her home, she did not recognize him, but consented to leave with him, saying, "He must be my husband because I heard him mention my brother's name to you."

A patient will frequently describe his last recollection before the onset of amnesia in such terms as, "everything went blank, there was a fog before my eyes." Two cases in our series had hysterical amaurosis. Similarly, a patient will describe his return of memory as "a fog lifting from me." In several instances, it was reported that memory returned after a dream in which some person, perhaps

the mother, called the patient by name. After memory has returned, the amnesia interval is often forgotten but may be recalled with the aid of hypnosis or amytal.

An important feature in our cases was the frequency of the complaint of headache. Reliable histories of recent head trauma were seldom obtained but occasionally a patient advanced the suggestion that he had been hit over the head and robbed. Such elaborations may expose the person to the suspicion of malingering, but a similar tendency to make accusations, frequently unfounded, was observed by Russell [2] during post-concussion confusion states. In connection with the false accusations, it should be emphasized that few of our patients could have been engaged in sexual or criminal activities during the amnesic interval.

SUMMARY.

1. Seventy-one cases of hysterical amnesia are reviewed; 41 were male, 30 female; the age distribution showed that most cases occur in the third and fourth decades of life.

2. Marital rifts and unfortunate love affairs are the most frequently recurring factors in the past histories. Alcoholism and financial difficulties also play important rôles. The various cases present characteristic features which are linked with the specific underlying problems.

3. Atypical cases of "hysterical amnesia" often appear to depend primarily on other than psychogenic factors, such as epilepsy, head injuries, etc.

4. The question of malingering is discussed and the typical features of the amnesic episode reviewed.

5. The frequency of headaches is emphasized and the significance of this complaint for a subsequent elaboration into false accusations is discussed.

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BIBLIOGRAPHY.

1. Abeles, M., and Schilder, P.: Psychogenic loss of personal identity; amnesia. Arch. Neur. and Psychiatr., 34: 587-604, September 1935.
2. Russell, W. R.: Amnesia following head injuries. Lancet, 2: 762-3, October 5, 1935.

THE LAURENCE-MOON-BIEDL SYNDROME.

Report of a Case.

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Although the Laurence-Moon-Biedl syndrome was first described 73 years ago and a considerable literature about this subject has appeared the world over, yet the etiology of this symptom complex is still a debatable question and some writers even doubt the advisability of regarding this peculiar combination of signs as a clinical entity. This confusion may be cleared up as more and more cases are recorded and careful studies are made of the complete classical syndrome of obesity, genital dystrophy, retinitis pigmentosa, mental deficiency, polydactylism and familial occurrence, and especially of the numerous variants of the true syndrome.

Many interesting ideas have been suggested in an attempt to explain the etiology of this condition. Early students of the subject considered a lesion of the pituitary gland responsible. The main objection to this theory was the absence in many cases of any evidence that could be interpreted as pointing to a lesion of this gland. Raab's theory that a high and massive dorsum sellæ led to a locking off of secretions from the pars intermedia with a resulting dysfunction of the hypothalamus has been discarded by the great majority of present writers because an abnormal sellæ is not present in all cases reported and because the latest studies of Wislocki and King [1] have disproven the theory of pituitary drainage into the hypothalamus. Biedl suggested that the syndrome is a familial form of adiposogenital dystrophy of cerebral rather than of pituitary origin and this idea is now generally accepted.

In more recent times the tendency has been to explain the pathogenesis of this syndrome on the basis of an hereditary developmental defect. Ornsteen,[2] for embryologic reasons; at-

[1] Wislocki, J. B., and King, L. S.: The permeability of the hypophesis and hypothalamus to vital dyes. Am. J. Anat., 58:421 (March 15) 1936.

[2] Ornsteen, A. K.: A contribution to the pathogenesis and heredity of the Laurence Moon Biedl syndrome. Report of three cases in one family. Am. J. M. Sc., 183:256 (Feb.) 1932.

tributes the first elements of the syndrome to a genetic defect of the prosencephalon, and the other symptoms, occasionally associated with the syndrome, such as atresia ani, polydactylism, skull deformities and others, to a chance coupling of genotypic unit characters. Jenkins and Poncher [3] state that this theory "furnishes a working hypothesis for adiposogenital dystrophy, mental deficiency and retinitis pigmentosa, but does not offer an adequate explanation for the other defects." They suggest that the syndrome is due to the linkage of two or more genes bearing unit characters; one for obesity and adiposogenital dystrophy, one for retinitis pigmentosa, and one for polydactylism They speculate as to whether the mental deficiency is due to one of these genes or is dependent on a linked gene. Rathmell and Burns [4] suggest that the entire syndrome is possibly due to an autonomic imbalance which is demonstrated by a high degree of emotional instability of both of their cases and the disturbance of water balance; the slow, regular, vagotonic electrocardiographic tracing, and the marked fluctuations of the basal metabolic readings in one case.

REPORT OF CASE.

Family History.

The historical information was obtained from the patient's parents and was considered to be fairly reliable. The paternal and maternal grandparents were native Americans. There were no significant factors in the history of the paternal or maternal grandparents, uncles, aunts or cousins with the exception of one paternal cousin who was committed to this institution and was classified as a case of dementia præcox, hebephrenic type.

Neither the grandparents nor the parents were related. The father was born in Illinois and is 73 years of age. He was formerly a school teacher and is at the present time a farmer. The mother was born in Missouri and is 70 years old. She is fairly obese. Considering their age, the parents are well both physically and mentally.

Including the patient, there are twelve siblings. Excluding the patient, we note that three of the children are definitely obese, three have some disturbance of vision, one has some difficulty in hearing, and another one, the third offspring, is evidently a mental defective. The essential features of the family tree are summarized in Fig. 1.

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[3] Jenkins, R. L., and Poncher, H. G.: Pathogenesis of Laurence Moon Biedl syndrome, Am. J. Dis. Child., 50: 178 (July) 1935.

[4] Rathmell, T. K., and Burns, M. A.: The Laurence-Biedl syndrome occurring in a brother and sister. Arch. Neur. and Psych. 39: 1033 (May) 1938.

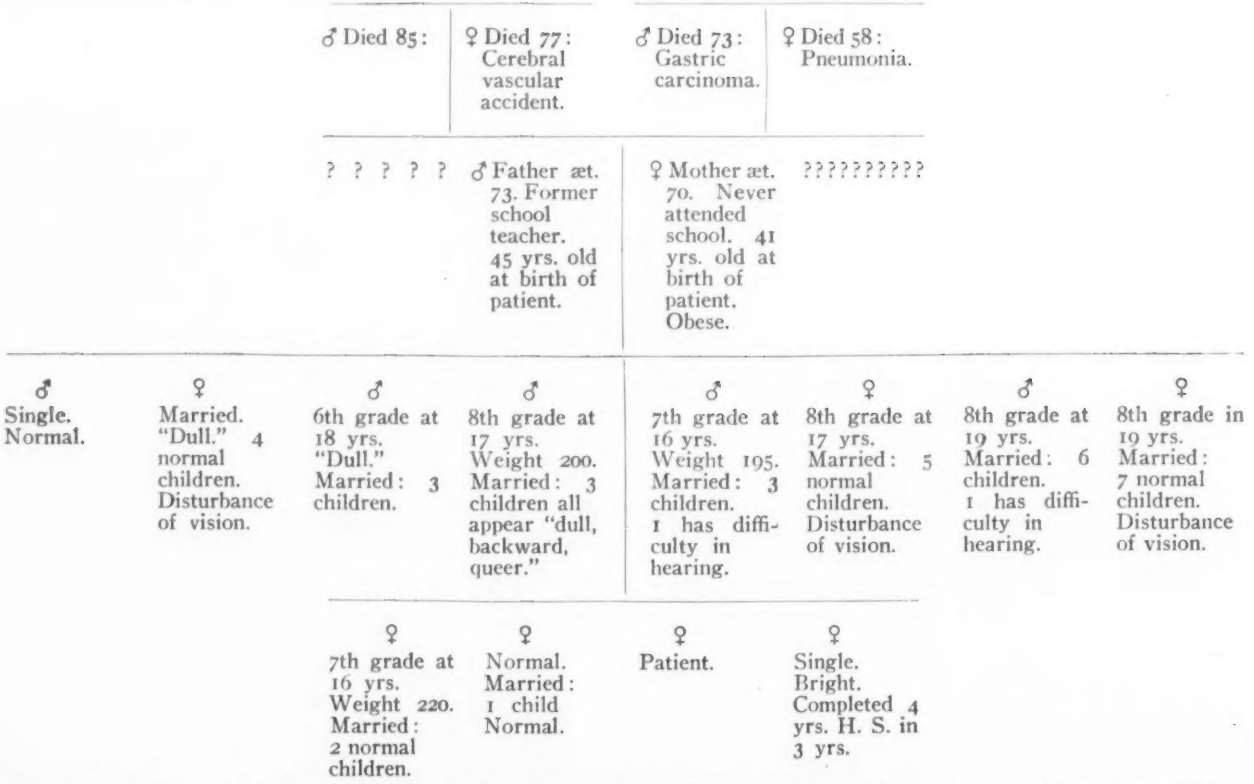


FIG. 1.—Genetic record of the family showing the Laurence-Moon-Biedl syndrome. Although none of the other children shows a definite syndrome, most of them show mental retardation.

Personal History.

Following a normal, full-term pregnancy, the patient was born January 1, 1910. The delivery was normal. There were no convulsions or cyanosis at birth. The early developmental history was apparently normal. The child walked and talked at about the same age as the other children. She started school at the age of six but always had trouble at school and when she reached the sixth grade the teacher advised the parents that further schooling would be useless. The patient was then kept at home until her admission to this institution.

Prior to admission the patient had measles, mumps and whooping cough in a mild form and no sequela were observed. An appendectomy was performed in 1937.

She was first admitted to this institution January 29, 1937. Examination at that time revealed a well nourished, well developed, obese, young, white female. Temperature 99.6: pulse 80: respiration 20: bloodpressure 150/70. She was 27 years of age, 61½ inches tall, and weighed 157 pounds. (Prior to admission her weight had been about 200 pounds but she had been on thyroid medication at home.) There was a marked obesity of the abdominal wall and an abundant amount of fat about the hips.

Examination of the extremities revealed rudimentary thumbs on both hands and polydactylism of the right index finger. Syndactylism of the third and fourth toes and marked pes planus were also noted.

The remainder of the physical examination was essentially negative except for markedly exaggerated tendon reflexes and the presence of ankle clonus and patellar clonus. A mental examination gave her an I. Q. of 42 and she was then classified as without psychosis, mental deficiency, imbecile.

The patient went home but returned to the institution September 21, 1937, with a history of increased irritability and of having been noisy, quarrelsome and combative. Marked emotional instability was evident. On this admission the patient was classified as a case of mental deficiency with psychosis.

Further examination and more detailed study lead to the finding of the additional features by which the syndrome is clinically recognizable.

Eyegrounds.—The eyegrounds were studied by Dr. A. M. Pasley of Jacksonville, Illinois. He reported the presence of marked optic atrophy of the right eye. The remainder of the fundus examination was essentially negative.

Examination of the Genital Organs.—Pelvic examination showed apparently normal structure of the external genitalia. The vagina readily admitted two fingers. The cervix was high and freely moveable. The uterus was small. No adnexal masses were palpable. The menstrual history was that menses were established at fourteen, occurring at six weeks intervals. Observation at the hospital revealed an amenorrhea during the first five months of 1938, and then there were regular periods for the next three months followed by an amenorrhea since that time.

Roentgenological Report.—Interpretations of the X-ray plates were made by Dr. N. S. Zeitlin of Chicago, Illinois.

Examination of the left hand shows the following anomalies in the bony structures: the metacarpals are unusually long and slender, particularly the first metacarpal. There is only one phalanx in the thumb and this is unusually long. There are no epipheseal centers visible in the films. Examination of the right hand shows that the thumb has only a short metacarpal and no phalangeal bones. Extra phalanges, partially developed, are noted running parallel to the index finger. The terminal phalanx of the index finger is very small. There are no epipheseal centers.

Examination of the right foot shows malposition of the tarsal bones, particularly the first, second and third cuneiform bones. Fusion of the proximal head of the fourth and fifth metatarsals and malposition of the phalanges of the fifth toe; anomalous condition of the joint between the phalanges and the third and fourth metatarsals so that the single phalanx makes one joint with the third and fourth metatarsal heads. Examination of the left foot shows a somewhat similar malposition of the tarsal bones with complete fusion of the distal heads of the third and fourth metatarsal bones. The fused head has only one phalangeal connection. There is considerable deformity in the resulting foot.

Examination of the skull reveals no evidence of any increased intracranial pressure. A single lateral view of the skull shows no evidence of any deformity. The sellæ turcica is within normal limits.

Clinical Findings.

Weight .....157 lbs.—62.8 Kg.
Height .....61½ in.—169.12 Cm.
Blood pressure .....150/70 mm.
Electrocardiogram .....Normal.
Basal metabolic rate.....— 6.
Serological reactions .....Negative.
Urine .....Sp. Gr. 1.012: Pus, albumin, squamous epithelium and occasionally casts.
Phenosulphothalein test .....37% and 45%.
Blood:
Count:
White cells ..... 10,600.
Red cells .....4,060,000.
Hemoglobin .....85%.
Color index .....1.
Differential .....37% Polymorphonuclear leucocytes.
13% Large lymphocytes.
45% Small lymphocytes.
2% Monocytes.
3% Eosinophiles.

Personal History.

Following a normal, full-term pregnancy, the patient was born January 1, 1910. The delivery was normal. There were no convulsions or cyanosis at birth. The early developmental history was apparently normal. The child walked and talked at about the same age as the other children. She started school at the age of six but always had trouble at school and when she reached the sixth grade the teacher advised the parents that further schooling would be useless. The patient was then kept at home until her admission to this institution.

Prior to admission the patient had measles, mumps and whooping cough in a mild form and no sequela were observed. An appendectomy was performed in 1937.

She was first admitted to this institution January 29, 1937. Examination at that time revealed a well nourished, well developed, obese, young, white female. Temperature 99.6: pulse 80: respiration 20: bloodpressure 150/70. She was 27 years of age, 61½ inches tall, and weighed 157 pounds. (Prior to admission her weight had been about 200 pounds but she had been on thyroid medication at home.) There was a marked obesity of the abdominal wall and an abundant amount of fat about the hips.

Examination of the extremities revealed rudimentary thumbs on both hands and polydactylism of the right index finger. Syndactylism of the third and fourth toes and marked pes planus were also noted.

The remainder of the physical examination was essentially negative except for markedly exaggerated tendon reflexes and the presence of ankle clonus and patellar clonus. A mental examination gave her an I. Q. of 42 and she was then classified as without psychosis, mental deficiency, imbecile.

The patient went home but returned to the institution September 21, 1937, with a history of increased irritability and of having been noisy, quarrelsome and combative. Marked emotional instability was evident. On this admission the patient was classified as a case of mental deficiency with psychosis.

Further examination and more detailed study lead to the finding of the additional features by which the syndrome is clinically recognizable.

Eyegrounds.—The eyegrounds were studied by Dr. A. M. Pasley of Jacksonville, Illinois. He reported the presence of marked optic atrophy of the right eye. The remainder of the fundus examination was essentially negative.

Examination of the Genital Organs.—Pelvic examination showed apparently normal structure of the external genitalia. The vagina readily admitted two fingers. The cervix was high and freely moveable. The uterus was small. No adnexal masses were palpable. The menstrual history was that menses were established at fourteen, occurring at six weeks intervals. Observation at the hospital revealed an amenorrhea during the first five months of 1938, and then there were regular periods for the next three months followed by an amenorrhea since that time.

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Color index .....1.
Differential .....37% Polymorphonuclear leucocytes.
13% Large lymphocytes.
45% Small lymphocytes.
2% Monocytes.
3% Eosinophiles.

CLINICAL FINDINGS.—CONTINUED.

Blood—Continued:
Chemistry:
Non-protein nitrogen .....60 mgm/100cc.
Fasting sugar .....80.64mg/100cc.
Glucose tolerance:
Fasting ..... 81.8 mg.
½ hr. .....107.1 mg.
1 hr. .....136.3 mg.
2. hrs. .....107.1 mg.
3. hrs. ..... 90.8 mg.
4. hrs. ..... 70.9 mg.
Cerebrospinal fluid:
Kahn .....Negative.
Colloidal gold .....0000000000.

COMMENT.

The diagnosis of the syndrome was based on the presence of obesity, genital dystrophy, mental deficiency, optic atrophy, skeletal deformities and familial history.

Evidence of marked emotional instability and autonomic imbalance is shown by frequent crying spells, temper tantrums, localized hyperidrosis, clammy hands and feet and dermographia.

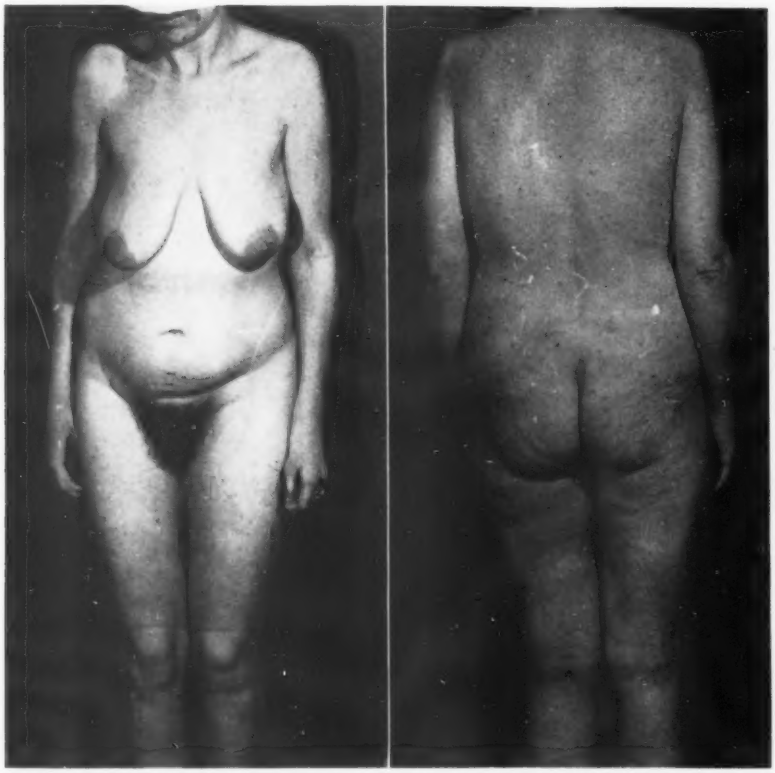
The emotional instability and autonomic imbalance demonstrated in this case and in the other reported in literature, and the autopsy reports of the Philadelphia General Hospital[5] suggest that the lesion is situated in the central autonomic nervous system.

SUMMARY.

A case of the Laurence-Moon-Biedl syndrome with all of its classical symptoms is presented.

This is the first report of this syndrome in conjunction with a psychosis.

[5] Rathmell, T. K., and Burns, M. A.: The Laurence-Biedl syndrome occurring in a brother and sister. Arch. Neur. and Psych., 39: 1033 (May) 1938.



A B

FIG. 2.—Anterior and posterior views of the patient.



FIG. 3.—Patient's hands.



FIG. 4.—Patient's feet.

Pictures taken by Dr. M. Summers of Jacksonville, Illinois.



FIG. 5.—Roentgenogram of the patient's hands.



FIG. 6.—Roentgenogram of the patient's feet.

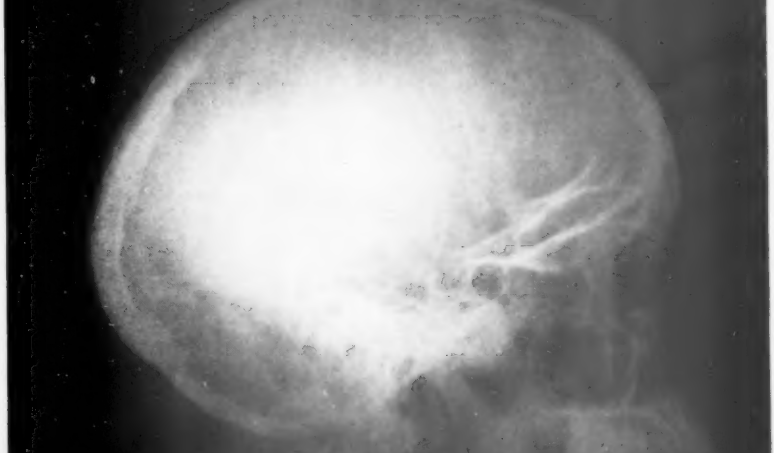


FIG. 7.—Roentgenogram of the patient's skull.

PSYCHIATRIC ASPECTS OF PORENCEPHALY.*

By E. LENNARD BERNSTEIN, M. D., Arlington Heights, Mass.

The absence of any reports of statistical data regarding the mentality of patients with porencephalic lesions has been noted in a recent publication by Patten, Grant and Yaskin.[1] A survey of the literature on porencephaly during the last 20 years confirms this observation. It therefore seems desirable to record the psychiatric findings in a series of such cases.

Seven of approximately 1000 cases admitted to the psychiatric clinic of the Yale School of Medicine in the years 1935-38 were found, upon roentgenographic examination, to have porencephalic lesions. Only one of the seven was known to be porencephalic on admission, and all presented themselves with psychiatric problems, either in the sphere of social adjustment, or arising from the occurrence of repeated convulsions. All had been seen by outside physicians or in other hospitals without the presence of the lesion being suspected, except in one case (case 3).

All patients had negative blood Kahns. In two of the cases, neurological signs and symptoms were present in such a minor degree that they were thought, before the lesion was detected, to be insignificant. In the other five cases, they conformed to the clinical pictures described by Patten *et al.*

The following abstracts summarize the neurological and psychiatric features of these cases:

Case 1.—A 13-year-old schoolboy, the son of a laborer, whose mother and five half-siblings were mentally deficient. He had no history of any head injury. After nine years of schooling he had only attained second grade standing, and had never spoken a spontaneous word to his teachers or fellow-pupils throughout his career. Grand mal convulsions began two months before admission, with more twitching on the left side than on the right. Physical examination showed an apathetic, slouching, untidy, underdeveloped, hydrocephalic boy, with large cyanotic extremities, who was very myopic in the right eye (V. A. 20/70), and right-handed, but weaker on that side. Encephalography revealed an enlarged skull with a deformity of the right side

* From the Department of Psychiatry and Mental Hygiene, Yale University School of Medicine.

of the calvarium, and multiple large defects in the brain substance, the two largest being in the right and left frontal lobes, respectively.

His I. Q. was just below 60 on repeated examinations; his energy output was extremely low. He was suspicious, sullen, irritable, uncommunicative, fearful of strangers and stubborn. He was reported to be dishonest and untruthful. Temperamentally, he was cold, with shallow affect; he had never made friends with anyone except one teacher, for whom, after three years in the same class, he formed a dependent attachment.

At times he grinned in a silly fashion and mumbled to himself. Retention of meaningful material seemed nil, but he could repeat six digits. No pathological mental content was obtained.

His mother used to beat him when he had a convulsion, and his oldest brother always said that he was ashamed of him. At school he was treated sympathetically by his teachers. He himself had no understanding of his condition. Following the encephalogram the convulsions ceased; he developed rapidly, and in the next two years he grew several inches in height, learned to read, and mixed with other boys as he had never done before.

CASE 2.—A 15-year-old schoolboy, who came from a stock of stupid, sullen morphine addicts and alcoholics. He was an unwanted child, normally delivered. He was said to have developed normally and to have been timid, polite and obedient, until he suffered severe injuries to his left cranial bones in an automobile accident when he was eight years old, at which time he was unconscious for 72 hours. Since then he had had temper-tantrums directed against the male members of his family, and habitually told "weird stories" about his adventures with aeroplanes, tigers and machine-guns. At the age of 14, he had begun to masturbate openly whenever he was angry or excited, and had written an "obscene" note to his teacher. On admission, he showed signs of the Fröhlich syndrome; he had gained 75 pounds in the previous 18 months. Neurological examination was negative. Encephalography revealed a tissue defect in the right frontal lobe.

He was of borderline intelligence, lazy, but with occasional explosive bursts of energy. He was said to be impulsive, vengeful, quarrelsome and aggressive. He carried five knives, and threatened his father with them. He hated his older brother and played malicious tricks on him, such as tangling up his radio and putting glue in his rubbers. He was irritable, fearful, sensitive, preferred solitary play, sought friendship with older men on the ward and had some effeminate mannerisms. In the clinic he appeared uniformly cheerful. In doing tests, he was lazy, suggestible, slow, uncritical, unable to make a sustained effort or to generalize. He gave up easily, could not attend to details, and had poor auditory and visual apperception.

His mother used to walk him about town on a leash, whipping him to stop his habit of biting women's legs. His father beat him and sold the patient's dog to buy drink. His teachers and fellow-pupils were afraid of him because he carried knives. The patient admitted that he got angry easily since

his accident, but said that the accident had nothing to do with it, and that it was all due to his treatment at home. He blamed others for the obscene note.

CASE 3.*—A 23-year-old man who came from a middle-class family. His birth was a difficult forceps delivery and he had postnatal jaundice. He did not thrive physically as a child and was very dependent on his mother. At the age of 6 he received a seemingly mild cranial injury, which was followed by a short psychotic episode and temporary paralysis of the right side. After he recovered from this, it was noted that his right leg frequently twitched while he was asleep; this continued up to the time of admission. Following another head injury at the age of nine he was unconscious for two hours. Since the first injury he had been quite unmanageable; he had temper-tantrums, masturbated frequently, exhibited himself, made sexual advances to young girls, stole, lied and wandered around the country. On examination, it was noted that he had a large head and a weakness of the right side of the palate, and that his left pupil was slightly larger than his right. Roentgenography at the age of 21 showed a defect in the central part of the right parietal bone, and a large cyst in the left frontal lobe. At operation, the adhesions impeding circulation to the latter area were freed, and it immediately became pink. The right frontal lobe was noted to be atrophic or agenetic. Following this operation at the age of 21, his condition remained unchanged; subsequent encephalography at the age of 23 demonstrated the lesion on the right but not that on the left.

His intelligence and energy output were average, with occasional short periods of torpidity. He was overly-polite, emotionally shallow, euphoric and impulsive, with transitory bursts of enthusiasm. In conversation, he was facetious, evasive, boastful and unsure of himself. He described a periodic compulsion to wander; during his wanderings he could do without food or rest, but eventually returned home exhausted. At other times he improved for a few weeks so that his parents would begin to think that he was cured. In character he was said to be fearful, suggestible, cold and remorseless; imprudent and irresponsible, living only for the moment, and unable to foresee the consequences of his acts. He was very sensitive to alcohol.

In test situations, he was able to handle materials in view very well, but was unable to build up ideas about things not seen; he was careless and unable to concentrate.

He was beaten by his father, detested by the neighbors, hated by his fellows and repeatedly expelled from school. His mother had always been worrisome and over-solicitous. He had been regarded as a psychopathic personality and treated by rigid disciplining.

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* This case is included in the series through the courtesy of Dr. Spafford Ackerly, of Louisville, Ky., who first thoroughly studied the patient. Dr. Ackerly has observed the case over a period of four years, and will report it in detail in a future publication. The operation was performed by Dr. Glenn Spurling, of Louisville.

CASE 4.—A 27-year-old man of middle-class family, who complained that he could not control himself. He had no history of any head injury. His parents dated his illness from an injection of diphtheria antitoxin at the age of six months. His motor and mental development were markedly retarded, and he suffered from weakness and atrophy of the right side of the body, and tics of the right hand. He adjusted fairly well within his intellectual limits until he was 25, when he began to be hypersensitive to sounds, to smile foolishly, to talk to himself and to have delusions of reference, with visual and auditory hallucinations, together with many compulsive symptoms and religious obsessions. On examination he was found to have a Fröhlich type of dysplasia, and signs of a lesion in the left cerebral hemisphere. Encephalography demonstrated a large dilatation of the left lateral ventricle, especially in its anterior portion; cerebral atrophy in both parietal regions, particularly on the right; and a probably atrophic cerebellum. There was also bilateral atrophy and sclerosis of the cranial bones.

His I. Q. was 50; he had always been fidgety and unable to concentrate, seclusive, fearful, irritable, sensitive and paranoid. He had been a passive butt for his companions, and his teachers had been unable to control his fidgetiness. His family was sympathetic, but patronising.

CASE 5.—A 30-year-old male who worked in his father's gasoline station in a rural community. He was delivered with forceps. He developed normally, except that he could not exert much strength or perform any fine movements, such as buttoning his clothes, with his left hand. At the age of 21 he was in an automobile accident, and received a laceration in the right occipital region and bilateral fractures of the mandible; he was unconscious for 24 hours. At the age of 25, following an alcoholic-sexual debauch, he had his first convulsion. Noting that his grand and petit mal attacks tended to follow the frequent and intense debaucheries reported, he stopped the latter, without any ensuing feeling of deprivation. Following this, there was a remission of the attacks for six months, and then they began again. His condition was diagnosed by several physicians as "idiopathic epilepsy," and by treatment with phenobarbital the frequency of his convulsions was considerably diminished. Physical examination on admission revealed signs indicating a lesion in the right parieto-occipital region, and encephalography demonstrated the largest single defect in the series, involving the right corpus callosum and replacing the greater part of the occipital and parietal lobes on that side. An interesting observation was made after air had been injected into the cerebro-spinal system, just before roentgenograms were taken; namely, that the site of the lesion could be outlined by percussion, both from its tympany, and from the sensations experienced by the patient when the overlying area was struck.

The I. Q. was in the low average range. The energy output was said to have been enormous, according to report long hours of work being followed by a prolonged debauch, but for the three or four years preceding admission, he had been "taking it easy, living quietly and going to bed early." His actual work output was steady, but slow and laborious. He was defensive, sensi-

tive and somewhat irritable, adjusting to new situations with a touch of defiance and considerable tension; he complained of a lack of self-confidence. Usually he felt cheerful and sympathetic, at other times he was moody. He had a defect of visual apperception, and his attention fluctuated in an "epileptic" manner. During a psychotic episode following a lumbar puncture at the age of 30, he had attempted suicide. (He was said to have been excited and confused for two days at this time; no further details could be obtained.)

CASE 6.—A 31-year-old woman who came from a stock of left-handed, very religious, rigidly proper, middle-class people. She was delivered normally but had post-natal cyanosis. As a child she was noted to be seclusive, irritable, over-conscientious and excessively prim, even in comparison with her own prim and conscientious relatives; she idolized her father. A few months after her menarche, when she was 15 years old, she began to have grand mal convulsions, preceded by a throbbing in the left side of the neck and a feeling that she was leaving the world, and followed by furors directed against her female relatives. At the beginning of an attack, she always turned her body and eyes to the right and raised her right arm. She had worked hard and done very well at school, but left because of the convulsions. Since that time she had remained at home, supervised without remission by her mother, who was very domineering towards all the children, especially after the father left the family. This happened when the patient was 24 years old, and from that time on the three children, of whom the patient was the oldest, joined the mother in expressing their hatred for him. Physical examination of the patient revealed signs pointing to a lesion in the left parietal region; encephalography demonstrated a cystic dilatation in the left lateral ventricle, together with a thickening and eburnation of the skull and a peculiar vascular marking in the left parietal area. Decapitation of the cyst has given relief of the convulsions for 15 months, to date.

The patient was of high average intelligence. Her energy output was slow and steady. She had always been left-handed, and good at detail work, such as embroidery. She was perfectionistic, pedantic and excessively tidy. Her hypercriticalness, bashfulness, sensitivity, jealousy and envy made it impossible for her to keep friends of either sex. She was emotionally cold, at times cruel, and had many temper-tantrums; she had a gushing, self-pitying, martyr-like attitude towards life.

She successfully used her illness to control her oversolicitous family in certain respects, to such an extent that her sister, whom she hated and envied, was beginning to have severe anxiety symptoms from fear of making a noise which might precipitate a convulsion; her mother's hypertension was attributed to the same cause.

CASE 7.—A 51-year-old housewife who came from an upper middle-class, psychopathic family. She had no history of any head injury. She was badgered by a domineering mother and always had an uncontrollable temper.

She did well at college in a pedantic way, but never made any friends. At the age of 18 she began to have grand mal convulsions, often followed by furors lasting several days, and tending to occur when she was tired or about to menstruate; also frequent absences with rigidity. At the age of 28 all the attacks ceased, "due to Christian Science." She married at 39; following the birth of her daughter at 41, the attacks recurred, increasing in frequency to status epilepticus. She attributed them to her really very docile husband's "cruelty," and her child's ill-temper. Physical examination was negative except for moderate signs of pituitary disorder of a Fröhlich type. Roentgen examination revealed hyperostosis frontalis interna, dilated ventricles with cortical atrophy, and three porencephalic cysts in the left parieto-occipital region.

Her I. Q. was 130; her energy output sporadic, with marked fatiguability. She said she felt irritable, sensitive, insecure and unable to cope with new situations. She was stubborn, domineering, suspicious, and had a marked anancastic trend, with perfectionism, rituals, aboulia, mysophobia and religious obsessions. She was emotionally cold, never had had a friend, and never had loved her husband, but clung to him because he made her feel secure in new situations. She continually accused him, in a paranoid way, of being unfaithful. She reacted quickly to criticism by a depression of mood. On the ward she was loud, verbose, irritable, resentful, aggressive, impulsive, over-reactive and apprehensive. She complained of progressive inability to concentrate, wandering attention and failing memory, all unconfirmed by tests.

The Site of the Lesion and Its Correlations.

Cases 2 and 3, who had frontal lobe lesions, were somewhat different from the others psychiatrically, and exhibited a syndrome which was not prominent in any of the cases not having lesions in this region. They stole and otherwise behaved anti-socially, without remorse, and then lied and attempted to shift the blame to others. They indulged in grossly anomalous sexual activities such as open masturbation and crude sadism. These two patients also tended to act more impulsively and with less foresight than the others, and were more imaginative, expansive and buoyant. Aside from these manifestations, no distinct differences were found, within the scope of the studies undertaken, between the cases with frontal and the cases with parietal lesions. Case 1, who had lesions in both frontal lobes, among others, exhibited some of the anti-social type of behaviour mentioned above, but otherwise was rather different from cases 2 and 3.

Discussion.

All the cases except perhaps 3 and 4 came from psychopathically tainted family stock. In early childhood, even before any convulsions or motor weaknesses had manifested themselves, most of the patients had been regarded as abnormal by their families. Four of them were retarded in school, two by more than four years; but more striking than the intellectual deficiency was the fact that only one (case 5) of the group made even a fair social adjustment in the school environment. Of the five who were old enough to work, only two ever paid their way; the other three made no attempt whatever to support themselves. All the patients were, if not grossly maladjusted, at least very poorly integrated in the sexual sphere.

The personality studies showed that all five male patients were of less than average intelligence, two of them failing to rise above the moron level, while the women were both of superior mental capacity. All the patients seemed to have an attenuated supply of energy, and were either sluggish and plodding, or if not, tired very easily. Several complained frankly of a feeling of inadequacy and inferiority. Traits of dependency, seclusiveness, stubbornness, touchiness, envy, resentfulness and defensiveness, were common. Three of the patients had well-developed anancasms and two exhibited a marked craving for attention and prestige. Temperamentally, the group seemed to be shallow, irritable, explosive, unsociable, cold, and far more ready to receive than to give sympathy and warmth. Four of them had a definite history of temper-tantrums. The mood was one of the most variable characteristics, varying greatly between different individuals, and in the same individual at different times.

In all cases the sensorium was intact at the time of examination. One patient (case 4) was a schizophrenic, two (cases 6 and 7) had had repeated prolonged post-convulsive furors, and one (case 5) had had a psychotic episode and had attempted suicide following a lumbar puncture. No notable loss of memory could be demonstrated in any of the cases, although both female patients complained of such a loss. The outstanding feature of the mental status was an inability to concentrate, complained of in all cases and confirmed in five. Three of the four patients who were studied in this respect, were found to be deficient in ability to generalize and to handle

abstract material (cases 1, 2, and 3). The patients tended to be uncritical and careless in answering questions. There was a defect of visual apperception in cases 2 and 5.

In every case, the family had an over-emotional attitude towards the patient and his illness, the treatments ranging from cruel beatings to domineering oversolicitousness. Several of the patients reacted to such treatment by blaming the attitude of the family for all their symptoms, and three of them took active steps to revenge themselves. Both female patients definitely used the threat of a convulsion to dominate their families in certain respects.

It is not felt that the psychiatric picture presented by these patients differs significantly from that presented by any other group of "epileptics," except possibly in one or two features of the mental status: *e. g.*, the inability to concentrate, and the deficient power of generalization. Further, the personality and mental status alone might often not permit differentiation of porencephalics from patients with other types of brain lesions, particularly some syphilitic, encephalitic and neoplastic ones.

The findings in these cases emphasized certain principles in the diagnosis of porencephaly. In all patients exhibiting epileptic phenomena, minor or major, the possibility of a porencephalic condition must be borne in mind, even in the absence of a history of cranial injury; and the slightest neurological irregularities should be thoroughly investigated before this diagnosis is ruled out. In children who exhibit the type of anti-social behaviour which has been described above, the possibility of porencephaly should also be carefully considered before it is discarded. In the absence of localizing physical signs, a study of the objective events and subjective experiences occurring before and during a convulsion may be of assistance in localizing the site of the lesion. It should be noted that porencephalics may exhibit abnormalities of behaviour in early childhood, long before any convulsive phenomena have occurred.

The therapeutic aim in a case of porencephaly should not be merely the elimination of epileptic phenomena, if such occur, but should include a reorganization and reorientation of the whole personality and social situation, according to the life history and the individual circumstances.

The great improvement in case 1, apparently following simple encephalography, is noteworthy.

SUMMARY.

Seven cases of porencephaly are presented from a psychiatric point of view. Patients with lesions in the frontal lobes seemed to present a slightly different picture from that of patients with lesions in other parts of the brain. The group, however, did not present any features which are thought to be specific for this type of epileptic patient, except perhaps a rather consistently occurring inability to concentrate. It is felt that the diagnosis of porencephaly should not be ruled out in the case of any epileptic until even slight neurological irregularities have been investigated. Porencephalics may exhibit abnormalities of behavior in many spheres for years before any convulsive phenomena occur. Patients with porencephaly should receive social and psychiatric as well as medical and surgical treatment.

BIBLIOGRAPHY.

Patten, Grant, and Yaskin: Arch. Neurol. and Psychiat., 37: 108, 1937.

Comment.

THE ADMINISTRATION OF PSYCHIATRY.

In connection with the timely symposium in this issue, consisting of papers by Overholser, Ebaugh and Hamilton on topics relating to mental hospital administration, it is well to reflect upon the varying fortunes of psychiatric practice in several of the state jurisdictions within the recent past. In 1925 the Committee on Standards and Policies, under the chairmanship of Dr. William L. Russell, formulated the famous schedule of minimum standards which was adopted by the Association as representing the indispensable conditions for efficient hospital organization and management. In surveys that have since been made, both in the United States and Canada, this schedule of standards has served as an invaluable guide.

Psychiatry is the one branch of medicine which from the beginning, insofar as its major activities are concerned, *i. e.,* the operation of its public hospitals, has been state controlled; and this circumstance must be assigned as one of the reasons for the slower development of mental medicine as compared with other disciplines which, cultivated in general hospitals and medical schools, have longer afforded fields for teaching and research, and inviting prospects for men and women of teaching and research caliber.

During several years past the Committee on Standards and Policies assembled data on the various types of state administration of mental hospitals, and on the basis of this information was able to make a rough grouping of the several jurisdictions under three headings, A (good), B (fair), C (poor), with respect to the efficiency of the administrative system in promoting the welfare of patients and the interests of the community and the advancement of psychiatry. With the creation of the Mental Hospitals Survey Committee it became possible for the first time however to make a country-wide study of mental hospital services

which should take account of every angle of the work on the basis of personal investigation by competent psychiatrists, members of the Association. Dr. Samuel W. Hamilton was the man logically chosen to take charge of this survey, and with his removal to Washington it is anticipated that he will be given opportunity to complete the study already so splendidly carried forward.

But there are disturbing notes in certain recent developments in the political field. In Michigan, an excellent plan of central control of the state hospitals, which was established on a strictly nonpolitical basis by former Governor Frank Murphy and which was being very ably carried out by Dr. Joseph E. Barrett, was nullified after Governor Murphy's defeat by the action of his successor in forcing the resignation of Dr. Barrett, entirely on political grounds. It is even reported that the present administration in Michigan plans to leave idle most of the buildings so sadly needed which were constructed under Dr. Barrett's supervision.

More recently the unfortunate effects of untrained control of hospital affairs have been demonstrated in Rhode Island. Governor Vanderbilt, on his election, appointed as director of the State Department of Social Welfare a jewelry manufacturer with no training in either welfare or medical work. It was generally hoped that with a new administration the assignment of employees by the State House and other similar types of interference would cease, but such was not to be the case. Dr. S. F. H. Howes, for many years in the Rhode Island state service, and for three years the competent and conscientious superintendent, asked for assurances that he would be permitted to conduct his own institution, but finding that he was still forbidden to employ anyone not previously approved by the central office in Providence, he resigned. It is to be hoped that his successor, Dr. Charles P. Fitzpatrick, formerly clinical director at Butler Hospital, will not be thus hampered as have been his immediate predecessors.

Such happenings, exemplified further in the political ousting of Dr. A. B. Stewart from the superintendency of the New Mexico State Hospital, should apprise the members of the Association that state care of the mentally ill while representing a remarkable advance beyond the methods which it replaced is still far from being the last word in this vital medical and social problem; and much educational work will still have to be done to bring the public to

demand that the abuses of spoilsmanship and privilege inherent in this form of state medicine shall cease, to the end that high standards of hospital administration in keeping with the best medical tradition and practice may everywhere prevail.

BIOGRAPHICAL DATA OF ASSOCIATION MEMBERSHIP.

For a long time, a need has been recognized for more detailed and better organized information about the members of the Association. The application-for-membership forms adopted within recent years contain valuable biographical material, but there is little available about the majority of the members. For example, inquiries are frequently received as to qualified psychiatrists who may be available for desirable opportunities of responsibility and we have repeatedly been impressed with our unpreparedness for such service.

For this reason and also in view of requirements should any national emergency develop, it has been decided to send to all known psychiatrists in the United States, members and non-members, biographical and military questionnaires for return to the New York office as soon as possible. In Canada only the biographical questionnaires will be distributed.

The biographical questionnaire will be used as a basis not only for the preparation of a professional directory of all members and fellows of the Association, but also in the estimation of the total psychiatric assets of the United States and Canada as to medical personnel.

These questionnaires were prepared after careful consideration by Dr. Chapman's Special Committee and the Military Mobilization Committee of which Dr. Steckel is chairman. The questionnaires are obviously of great importance and I hope each member and fellow will fill them out as completely as possible, returning them promptly to the New York office. Your full cooperation will be greatly appreciated.

W. C. S.

News and Notes.

THE RESEARCH COUNCIL ON PROBLEMS OF ALCOHOL.—The Research Council on Problems of Alcohol held its annual meeting in New York on Tuesday, October 17, 1939. Announcement of three grants of financial aid to the Council was made. The Carnegie Corporation has appropriated $25,000 for a critical survey of all work done to date on the effects of alcohol upon the individual, a project sponsored by the department of psychiatry of the College of Medicine, New York University. It is under the supervision of Dr. Norman Jollifee, associate professor of medicine, who recently directed a survey of alcoholic psychoses in Europe. The American Philosophical Society is financing a study of the toxic factors in alcoholism, conducted at the New York Psychiatric Institute under the direction of Dr. George A. Jervis, and the Dazian Foundation for Medical Research has granted funds for research on the rôle of alcohol in liver cirrhosis, a project being initiated by the College of Medicine at New York University. Some 200 patients with liver cirrhosis will be subjected to clinical, chemical and pathological study in an effort to reach a clearer understanding of the relation of alcohol to this disease.

The Council also decided to concentrate upon the problem of alcoholism until the end of 1941. The following is quoted from the announcement released at the time of the meeting: "The objective of the Council is to discover the causes of alcoholism and better methods for its prevention and treatment. Prejudice, fear, secrecy and ignorance are among the obstacles to its control. The majority are disposed to think of alcoholism as a criminal or moral offense, instead of as a disease for which the patient is often not responsible. Alcoholism and the alcoholic psychoses constitute one of the great public health problems of modern times, perhaps the greatest not being systematically attacked. The Council plans to deal with alcoholism in the same general fashion as other public health agencies combat tuberculosis, cancer, syphilis, heart disease and other major threats to human health."

Dr. Winfred Overholser, superintendent of St. Elizabeth's Hospital, Washington, D. C., was elected chairman of the Executive Committee, to succeed Dr. Karl M. Bowman, director of the Division of Psychiatry of Bellevue Hospital, New York City. Dr. Bowman was elected chairman of the Scientific Committee to succeed Dr. Hans T. Clarke, professor of biochemistry, Columbia University. Dr. Edward A. Strecker, professor of psychiatry at the University of Pennsylvania, Philadelphia, Pa., was elected vice-chairman of the Scientific Committee.

K. M. B.

THE MENNINGER CLINIC RESIDENCIES.—Four accredited residencies annually, two beginning January 1, and two July 1, are available at the Menninger Clinic. Intensive training is given in psychoanalytic psychiatry, neurology, child psychiatry, psychotherapeutic and other psychiatric techniques. Applications for July, 1940 openings are now being received. Address The Menninger Sanitarium, Topeka, Kansas.

PENNSYLVANIA PSYCHIATRIC SOCIETY.—The definitive organization meeting for the founding of the Pennsylvania Psychiatric Society was held as a dinner meeting in the Forum Room of the Hotel William Penn, Pittsburgh, Pa., at seven o'clock the evening of Thursday, October 5. There were 56 psychiatrists present, most of whom are members of The American Psychiatric Association.

Dr. William C. Sandy spoke on the preliminary organization, purposes, aims and functions of the Society, the following being an abstract of his address.

A state-wide group of psychiatrists has met several times as the "Founders of the Pennsylvania Psychiatric Society" and has formulated a constitution and by-laws, also selecting officers, for final consideration at the definitive organization meeting on October 5.

The need for and usefulness of such a professional society may be realized when one considers what similar organizations have accomplished for psychiatry. For example, higher standards for mental hospitals, for membership in The American Psychiatric Association and for the officially recognized specialty in general, have resulted from the activities of special medical committees and boards. In Pennsylvania, state-wide mental hygiene committees have brought about increased interest in and support of activities for the general welfare of mental patients.

Briefly, such a society should become affiliated with the national association; should exercise a state-wide general leadership in psychiatry, being a co-ordinating body to which controversial and other questions of psychiatric interest may be referred for an authoritative opinion; should encourage general medical interest by participation in society meetings; and should assist in developing and supporting proper psychiatric standards.

Officers were elected as follows:

William C. Sandy, M. D., president.

Dr. Sandy is the director of the State Bureau of Mental Health, Harrisburg, Pa.

Henry I. Klopp, M. D., president-elect.

Dr. Klopp is superintendent of the Allentown State Hospital, Allentown, Pa.

LeRoy M. A. Maeder, M. D., secretary-treasurer.
Philadelphia, Pa.
(Chancellor Hall, 206 South Thirteenth St.)

COUNCILLORS.

James S. Hammers, M. D., Lancaster, Pa.
Charles H. Henninger, M. D., Pittsburgh, Pa.
Baldwin L. Keyes, M. D., Philadelphia, Pa.
S. DeWitt Ludlum, M. D., Philadelphia, Pa.
Howard K. Petry, M. D., Harrisburg, Pa.
George W. Smeltz, M. D., Pittsburgh, Pa.

AUDITORS.

Leslie R. Chamberlain, M. D., Danville, Pa.
Arthur P. Noyes, M. D., Norristown, Pa.
George J. Wright, M. D., Pittsburgh, Pa.

THE AUSTEN RIGGS FOUNDATION.—On October 21, 1939, the Medical Advisory Board of the Austen Riggs Foundation held its annual meeting in Stockbridge, Massachusetts with between 40 and 50 psychiatrists and internists in attendance. A paper discussing certain aspects of cases falling in the neurotic-psychotic borderline was read by Dr. V. Gerard Ryan, who holds a fellowship in the Austen Riggs Foundation under a grant from the John and Mary R. Markle Foundation. The meeting was conducted by

Dr. Austen Fox Riggs, medical director of the Austen Riggs Foundation. Among others the paper was discussed by Dr. Adolf Meyer of Baltimore, Drs. Macfie Campbell and Stanley Cobb of Boston, Earl Bond and Kenneth Appel of Philadelphia and Arthur Ruggles of Providence, Rhode Island.

DR. GRAVES HONORED BY ST. LOUIS MEDICAL SOCIETY.—On October 24, 1939, a meeting was held at the St. Louis Medical Society in honor of Dr. William Washington Graves, professor of neuropsychiatry, St. Louis University School of Medicine. At this meeting Dr. Graves was awarded a certificate of merit and a gold medal for scientific accomplishment for his classification of scapulae and other inherited characters, and for his discovery of "The Age-Incidence Principle of Investigation."

A copy of this certificate of merit is subjoined.

THIS CERTIFICATE OF MERIT

PRESENTED BY

THE ST. LOUIS MEDICAL SOCIETY

TO

WILLIAM WASHINGTON GRAVES, M. D.

IN CONSIDERATION of the USEFULNESS of his CLASSIFICATION of SCAPULAE and other INHERITED CHARACTERS, and of his DISCOVERY of "THE AGE-INCIDENCE PRINCIPLE of INVESTIGATION," both of which resulted in NEW APPROACHES to the QUALITATIVE EVALUATION of INHERITED VARIATIONS in relation to the INHERITED QUALITIES of HUMAN CONSTITUTION, expressed in INHERITED PREDISPOSITION to HEALTH or DISEASE, and in INHERITED CAPACITY for EDUCATION, for ADAPTABILITY, and for LONGEVITY.

Given by Authority of the St. Louis Medical Society,
October 24, 1939.

JEROME I. SIMON, M. D.
Secretary

ALPHONSE MCMAHON, M. D.
President

(SEAL)

The St. Louis Medical Society has given this certificate of merit for scientific accomplishment on only two previous occasions, and the award to Dr. Graves was therefore an outstanding event in the St. Louis Medical Society.

The previous awards were made to Dr. Evarts A. Graham and his associates Drs. Glover H. Copher, Warren H. Cole and Sherwood Moore, for their work in cholecystography; and to Dr. Edward A. Doisy, for his achievements in hormone chemistry and physiology.

INSTITUTE OF GENERAL SEMANTICS.—Announcement has been made of the schedule of seminar lecture courses in general semantics conducted by Count Alfred Korzybski, 1939-1940, at the Institute of General Semantics in Chicago. There are three intensive one week courses December 27 to January 3, June 19 to June 26 and August 23 to August 30, including morning, afternoon and evening sessions.

There are also three evening seminar courses January 24 to February 14, April 3 to April 24, and July 8 to July 29. In these courses there are three evening meetings each week.

Seminar enrollments are limited. Registration begins three weeks in advance of the first lecture of each series. Fees for the evening series are $25.00; for the intensive series $40.00. A registration fee of $5.00 will be credited to tuition. A few half tuition grants are available for students living at a distance from Chicago.

Information may be obtained by writing to the Executive Secretary, Institute of General Semantics, 1234 East 56th St., Chicago.

THE NEUROPSYCHIATRIC INSTITUTE, UNIVERSITY OF MICHIGAN. *The University of Michigan Official Publication* of October 25, 1939, contains a description of the new Neuropsychiatric Institute, which opened its doors for the admission of patients on March 8 of this year.

It will be recalled that Michigan was the first state to establish a psychiatric service in connection with its University and as a part of its general hospital and medical education program.

The original institution, the State Psychopathic Hospital at Ann Arbor, opened its doors February 7, 1906, with Dr. Albert M. Barrett, as director.

Dr. Barrett's long and distinguished career, his splendid service to the University and the State and his noteworthy contributions to medical education and research constitute a unique historical background for the new Institute.

The Neuropsychiatric Institute is a five story building situated on the University Hospital grounds and providing 62 beds for adults, and 20 beds for children, as well as an out-patient department. The new Institute will be the psychiatric teaching, training and research centre of the State and the University, and will afford under very favorable conditions enlarged opportunity for investigation and therapy in the field of psychiatry such as have long been traditional at Ann Arbor.

Dr. Raymond W. Waggoner is director of the Neuropsychiatric Institute and there are ten physicians attached to the staff.

Dedication Exercises, Arthur H. Harrington Assembly House, Howard, R. I.—On Wednesday, October 25, 1939, the new auditorium building at the State Hospital was dedicated to Dr. Arthur H. Harrington and named the "Arthur H. Harrington Assembly House."

The program consisted of two parts, a memorial service held at 12:30 in the congregate dining hall, at which organ selections and selections by the choir were rendered and prayers offered by the three hospital chaplains. Following this, lunch was served, and at 2:30 the dedicatory exercises were held in the main hall of the auditorium. Mr. Vincent Sorrentino, director of Social Welfare for the State of Rhode Island, presided. A preliminary address was given by the Honorable James O. McManus, Lieutenant-Governor, following which Dr. John E. Donley gave the dedicatory address and formally dedicated the building as the Arthur H. Harrington Assembly House.

Dr. Donley paid a very eloquent tribute to Dr. Harrington and the work for which he was responsible at the State Hospital. The exercises were opened and closed with selections by the symphony

orchestra of the Works Projects Administration music project of Rhode Island. There was a large attendance representing friends of Dr. Harrington in professional and business life, and there were many present representing various social welfare organizations in the state. Psychiatric representatives from adjoining New England states also took part.

ANNUAL MEETING, AMERICAN PUBLIC HEALTH ASSOCIATION.—The 68th annual meeting of the Public Health Association was held October 17 to 20, 1939, in Pittsburgh, Pa. More than 2,500 professional public health workers from the United States, Canada, Cuba, Mexico and five foreign countries were in attendance. The conference represented a complete index of all current occupations and activities of the modern public health program, including upwards of 200 scientific sessions and listing more than 300 speakers and discussers. The syphilis control program and a session on cancer were especially notable. An unusually intensive health exhibit was participated in by 95 commercial organizations and health agencies.

The American Public Health Association has ten sections—Health Officers, Laboratory, Vital Statistics, Public Health Engineering, Industrial Hygiene, Food and Nutrition, Child Hygiene, Public Health Education, Public Health Nursing and Epidemiology.

The new officers elected for 1939-40 are:

President: Edward S. Godfrey, Jr., M. D., Albany, N. Y.

President-Elect W. S. Leathers, M. D., Nashville, Tenn.

1st Vice-President: Elisabeth C. Smellie, R. N., Ottawa, Canada.

2nd Vice-President: Domingo F. Ramos, M. D., D. Sc., Havana, Cuba.

3rd Vice-President: Wilton L. Halverson, M. D., Pasadena, Calif.

Treasurer: Louis I. Dublin, Ph. D., New York, N. Y.

Executive Secretary: Reginald M. Atwater, M. D., Dr. P. H., New York, N. Y.

Chairman of Executive Board: Abel Wolman, Dr. Eng., Baltimore, Md.

The 69th annual meeting of the Association will be held in Detroit, Mich., in October 1940.

SCIENTIFIC EXHIBIT AT THE CINCINNATI MEETING IN 1940.—Space has been provided for the scientific exhibit at the next annual meeting of The American Psychiatric Association, to be held May 20-24, 1940, at the Netherlands Plaza Hotel, Cincinnati, Ohio. The scientific exhibit offers an opportunity for investigators and visiting members to meet and discuss informally the various phases of the research work presented. The exhibit is also of special advantage to persons reading papers, since the exhibit can be used to supplement the paper read. The scientific exhibit at the annual convention of the American Medical Association has come to be recognized as a week of opportunities for graduate education not equaled by any similar opportunity available to the medical profession. It is hoped that in the future the scientific exhibit of The American Psychiatric Association will fill the same purpose in the field of psychiatry.

The following topics for the purpose of general orientation are suggested: interesting material relating to research in new methods of treatment, clinical psychiatry, psychopathology, mental hygiene, child psychiatry, administrative psychiatry, diagnostic methods, new laboratory procedures, neuropathology, neuroanatomy, etc.

Important aids toward a successful scientific exhibit are personal demonstration; plainly lettered posters; statement of purposes or justification of exhibit; and neatness and care in planning material. The Association furnishes wall space, consisting of a beaver-board wall eight and one-half feet in height, and signs with the title of the exhibit and names of the exhibitors. Application for space with a short description of the exhibit material for the program, not exceeding 150 words, should be sent to the Director of the Scientific Exhibit, Dr. Walter L. Bruetsch, Central State Hospital, Indianapolis, Indiana, not later than March 1, 1940.

AMERICAN ORTHOPSYCHIATRY ASSOCIATION MEETING.—The 17th annual meeting of the American Orthopsychiatric Association, an organization for the study and treatment of behavior

and its disorders, will be held at the Hotel Statler, Boston, Mass., on February 22, 23, and 24, 1940. For information address the secretary, Dr. Norvelle C. LaMar, 149 E. 73rd St., New York, N. Y.

THE EMMA PENDLETON BRADLEY HOME.—The Emma Pendleton Bradley Home, East Providence, R. I., announces a postgraduate course in practical nursing care of neuropsychiatric disorders of childhood. A twelve weeks course is provided, limited to the acceptance of two postgraduate nurses each period, beginning the first of January, April, July and October, 1940. There is an all graduate nursing staff. Full maintenance is given.

The Emma Pendleton Bradley Home is a fully equipped, 50 bed hospital, dealing exclusively with neurological and behavior disorders of children of normal intelligence under thirteen years of age. Four divisions of the Home include the nursery school, cerebral palsy ward and two general wards for older and for younger children. Facilities include opportunity for individual conferences with the various trained members of the staff, observation of each child on the ward and in school or play, and complete medical, social, psychological and psychiatric records are available on all cases. Further details may be had from Dr. Arthur H. Ruggles, superintendent, Dr. Charles Bradley, medical director, or Miss Kathleen Thornton, R. N., director of nursing.

CHILD STUDY HOME OF MARYLAND.—The Board of Directors of the former Nursery and Child's Hospital of Baltimore has reorganized its functions to create a much-needed psychiatric in-patient unit for children. The new Child Study Home, located on 721 Woodbourne Avenue, in a suburban area, has been recently erected to suit all modern requirements. It has a 47-bed capacity and, in addition to serving the community agencies, is equipped to receive private patients. It has a full-time resident pediatrician-psychiatrist (Dr. Georg Frankl), a staff of teachers and an occupational therapy department. All remedial activities are carried out under the guidance and supervision of Dr. Leo Kanner, director of the Children's Psychiatric Service of the Johns Hopkins Hospital. Inquiries may be addressed to Miss Elizabeth Mertz, superintendent.

JOURNAL OF CRIMINAL PSYCHOPATHOLOGY.—Number 1 of this publication made its appearance July 1939. It proposes "to provide a channel for the prompt publication of clinical investigation, medical or otherwise, into the field of mental abnormalities among criminals." The Journal is published by the Woodbourne Institution for Defective Delinquents, New York State Department of Correction, is issued quarterly and distributed free of charge, as a project of the Institution's class in journalism. Each issue will contain original articles, abstracts from current literature and book reviews. Contributors to the first two numbers include V. C. Branham and J. J. Brooks, Bernard Glueck, Aleš Hrdlička, Ben Karpman, C. P. Oberndorf, Chester D. Owen, J. Rubin, Jack Schuyler.

This noteworthy undertaking is under the editorial direction of Dr. V. C. Branham, superintendent of the Woodbourne Institution for Defective Delinquents. There are six assistants on the editorial staff.

FIFTIETH ANNIVERSARY, DELAWARE STATE HOSPITAL.—The fiftieth anniversary of the Delaware State Hospital at Farnhurst, Delaware, and dedication of the newly erected chapel took place September 28, 1939. To commemorate this event a special memorial brochure was issued containing an interesting historical résumé on the care of the mentally ill in Delaware together with photographs of the governors, members of the boards of trustees and superintendents of the hospital who have administered the institution during those fifty years.

An impressive program was arranged. Greetings were extended by his Excellency, Richard C. McMullen, Governor, and other state officials, and many eminent representatives of American psychiatry and mental hygiene participated. Dr. M. A. Tarumianz, the superintendent, presided at the meeting, and Dr. Edward A. Strecker, professor of psychiatry at the University of Pennsylvania, the principal speaker, discussed "The Social Implications of Psychiatry."

Delaware was the first state to assume the responsibility for the care and treatment of its mentally ill. The hospital opened its doors August 1, 1889, with 99 patients; today it houses 1200. (Normal accommodation, 900.) The development and scientific activities of

the hospital during the last half century are especially noteworthy. Provision of better housing conditions for patients, increased medical staff and personnel, modern treatment facilities, the development of an occupational therapy department which cares for 600 patients daily, the establishment of a social service department, and the organization of a training school for nurses with affiliation with all of the general hospitals in Wilmington, are conspicuous advancements which have been made.

A sterilization law was passed in the State of Delaware in 1923 and amended in 1929. To date there have been sterilized 525 patients in the hospital proper and 38 cases at large. Future plans include the provision of proper facilities for the care of children who can only be cared for at present on the same ward with adults; and a negro unit, the patients to be cared for by physicians, nurses and other employees of their own race.

Book Reviews.

THALASSA: A THEORY OF GENITALITY. By *Sandor Ferenczi, M. D.* Translated by *Henry Alden Bunker, M. D.* (Albany: The Psychoanalytic Quarterly, 1938.)

This work is devoted to the exposition of the thesis that the whole purpose of human evolution, therefore likewise of the sex act, is no other than an attempt on the part of the ego to return to the mother's womb, where there is no such painful dysharmony between ego and environment as characterizes existence in the external world. It is further submitted that all animal existence was originally aquatic, air and land existence being later acquisitions in our evolution; and that the basic human wish to return to the mother's womb is but a personal expression of the unconscious cosmic wish to return to our original aquatic existence. In the mother's womb surrounded by amniotic fluid we feel once again as if returned to our original thalassic existence.

Ferenczi arrived at this idea through the consideration of certain clinical data. He submits that in its early days, psychoanalysis concerned itself to a relatively much greater degree with pregnancy and birth, with acts preparatory to coitus, and with the perversions than with the meaning and explanation of the phenomenon of the act of coitus itself. In the main, the work is an elaboration along biological lines of Freud's *Three Contributions to the Theory of Sex*, more especially with reference to the evolution and development of the act of coitus which Freud conceives as the final phase in the total course of sexual development; an onto- and phylogenetic theory of genitality.

Ferenczi's reflections on the subject had their origin in certain psychoanalytic observations on impotence in the male. Abraham in discussing the so-called "pregenital organizations," has attributed ejaculatio præcox to a too intimate association of urethral erotism with genitality, and believed that these patients who suffer from ejaculatio præcox manifest the same attitude of indifference toward their semen as they would toward their urine, that is to say, as valueless excretion. On the other hand, according to Ferenczi's observations, there are patients who are excessively economical of their semen, many of them so much so that they suffer, properly speaking, only from impotentia ejaculandi: that is, they are capable of erection and intromission, but are incapable of ejaculation. In the unconscious, and to some extent, even in the conscious, mentations of these patients, the equating of coitus with the act of defecation plays an important part. Not infrequently, these patients have displaced upon the sexual act the self-will and obstinacy with which in childhood they opposed the strict regulation of their excretory

habits, imposed on them by convention. They are very capricious in their sexual behavior. They are impotent when the partner desires intercourse, they have erections only when the performance of the act is for some reason impossible or impracticable (*e. g.* menstruation). Because of this, Ferenczi feels it is fair to assume in these patients as intimate an association of anal elements with the act of coitus as Abraham has shown to be true of urethral elements in the case of ejaculatio præcox. Such specific anal technique in impotence in the male equally may be found, according to Ferenczi, in less pronounced disturbance of the act of coitus. Many men have the compulsion to defecate before performing the sexual act; moreover, severe nervous digestive disturbances may disappear when emotional inhibitions of sexuality have been analytically resolved; one is familiar with the obstinate cases of constipation which is a not unusual consequence of excessive masturbation and squandering of semen. Among the "character regressions" may be cited the case of men who, in other respects generous, are very petty and even niggardly in the matter of giving money to their wives.

The simple consideration of the nature of the sex act from the intromission of the penis to ejaculation would seem to support the above assumptions. The terminal event of coitus, the ejaculation of semen, is undoubtedly an urethral phenomenon, which has in common with the voiding of the urine not only its channel of excretion but the fact of being the ejection of a fluid under great pressure; on the other hand, during the frictional process, inhibitory influences, in all probability of sphincteric origin, seem to assert themselves and to be capable, if they gain the upper hand to an untoward degree, of bringing about complete absence of ejaculation. Everything points to the fact that the urethral (*i. e.* ejaculatory) tendency is at work from the beginning, throughout the entire frictional process, and that in consequence an unceasing struggle occurs between the evacuatory and the inhibitory purposes; between expulsion and retention, in which the urethral element is eventually victorious. This two-fold innervation might, among other things, manifest itself also in the to-and-fro motion of the frictional process, in which penetration would correspond to the ejaculatory tendency, withdrawal to an ever recurring inhibition. Naturally one would have to ascribe significance also to the increase of excitement consequent upon continued friction, and to assume that this increase is capable, on exceeding a certain level, of finally overcoming the spasm of the sphincter. This assumption presupposes a highly complicated and finely graduated coordination, a disturbance of which would produce just that ataxia and dyspraxia which one may describe as premature and inhibited emission.

What has been described above in physiological terms as a coordination of urethral and anal innervations may be expressed in the vocabulary of the sexual theory as a synthesis or an integration of anal and urethral erotism into genital erotism. Such synthesis of two or more erotisms in a higher unity is termed by Ferenczi amphimixis of erotisms or instinct-components, and represents to him the passing of the hitherto active autoerotisms (excitations of the so-called erogenous zones) from a primary position to a posi-

tion subordinate to the primacy of the genital zone. Between urethral and anal autoerotisms there seems to exist a kind of reciprocity well prior to the establishing of genital primacy. It is conceivable that besides genital amphimixis there are as well progenital fusions of different sorts. On the basis of these observations Ferenczi has come to the conclusion that the act of ejaculation is essentially a phenomenon of urethro-anal amphimixis.

Whether one agrees with Ferenczi or not, the concept provides some very stimulating suggestions for neurophysiology and neuroanatomy to consider, to wit, the interrelationship between genital and anal innervations, and between genital and other innervations. There is a great deal about the act of coitus about which neurophysiology and neuroanatomy are completely silent. What, for instance, is the relationship between orgasm and ejaculation? The subjective experience of man fuses these two processes into one; but clinical material suggests that they are probably separate. This, however, can hardly be determined in normal subjects, but only by the observation and study of psychopathological material. The reviewer has had in his experience the case of a man who experienced orgasm without ejaculation. This was traced to an incest phantasy which unconsciously occupied the man's mind during coitus; ejaculation was prohibited for it would lead to pregnancy; at the same time the man as a neurotic would not forego the pleasure of the orgasm—a beautiful instance of pleasure without guilt and of neurotics' capacity to eat the cake and have it at the same time. But in terms of neurophysiology, are we dealing here with two different innervations, two sphincters, or what?

From this point on Ferenczi is led to more intimate considerations of the problem. Infantile erotisms, on becoming forepleasures in the adult, serve to set in motion the genital mechanisms proper, the actual discharge of the excitation taking place only at the moment of ejaculation. It is as if all these erotisms have been transmuted into the channel of another, more powerful erotism. It is the summation of simple, independently acting erotisms into one, all-inclusive genital erotism, which on attaining a certain vehemence lead to erection, and the urge to intromission and friction, to culminate in the amphimictic phenomenon of ejaculation already mentioned. Thus, the genital center comes to represent in it other parts of the organism, acting in the rôle as it were of an executive manager, providing for the discharge of sexual tension on behalf of the entire organism. The phallus, suggests Ferenczi, may thus be viewed as a miniature of the total ego, as the embodiment of the pleasure-ego. In dreams and other products of phantasy, this miniature ego represents the total personality.

Ferenczi thus arrives at the conclusion that in the sex act and in whole life, the purpose is to return to the mother's womb. The sex act achieves this transitory regression in a threefold manner: the whole organism attains this goal by purely hallucinatory means, somewhat as in sleep; the penis, with which the organism as a whole has identified itself, attains it partially or symbolically; while only the sexual secretion possesses the prerogative, as representative of the ego and its narcissistic double, the genital,

of attaining in reality to the womb of the mother. It thus seems to Ferenczi that from the moment of birth onwards the human being is dominated by a continuous regressive trend toward the reestablishment of the intrauterine situation, holding fast to this by magic-hallucinatory means. However much reality sense is attained, this regression is never fully renounced; in sleep, in dreams and phantasy life and in sex life, the striving toward the fulfillment of that primitive wish is still clung to. In the first oral stage of infantile sexual organization those who take care of the child are still solicitous that the illusion of the intrauterine state is preserved to it; they provide the warmth, the darkness and the quiet which are requisite to that illusion. The excretory functions are for the time being entirely uncontrolled, and the actual activity of the newborn infant is limited principally to sucking the mother's breast. The rhythmicity of sucking remains permanently fixed as an essential ingredient of every subsequent erotic activity, and becomes incorporated amphimictically in the act of masturbation and coitus.

That such regressive trends are found in some of the severe neuroses, the reviewer does not doubt, for he has seen such phantasies uncovered. He seriously questions, however, the universality claimed for them by Ferenczi and even doubts their moderate frequency. But when Ferenczi further expresses a belief that he expects this train of thought will find striking confirmation in the observation of the sexual life of animals and further submits as a biological proof of it the fact that in many animals the sex act is of well nigh interminable duration, the reviewer feels that the author has passed the limit of logical acceptance. He does not see how the long duration of the act is proof of a maternal regression trend. Generally speaking, the duration of the sex act proper throughout the animal kingdom is rather brief than long. Where it is of long duration there may be special anatomical reasons that have nothing to do with maternal regression.

From here on, Ferenczi's attempt to prove the universality of the regressive maternal trend becomes more and more speculative and his biology more and more shaky. Taking up next the problem of the erection, he submits that this too may derive its explanation from the maternal womb theory of genitality. He assumes that the permanent invagination of the glans penis within a fold of mucous membrane (the foreskin) is itself nothing but a replica in miniature of existence in the maternal womb. If so, the reviewer wonders whether the millions of people in this world who have been circumsized for ritual or medical reasons, would still come under the operation of this trend. Discussing next the frictional movements which for some duration precede ejaculation, Ferenczi submits that these movements may be explained by the biological process of autotomy, consisting in the fact that the organs which irritate or otherwise annoy the animal body are simply detached from the rest of the body by the action of certain muscles, or, in the literal sense of the word, dropped. Ferenczi sees in autotomy an organic prototype of the process of regression, of withdrawal cathexis from the painful, an evidence of organic regression. In a wider sense he thinks of it as equivalent to the death instinct. He sees in this type of reaction a fundamental

property of all life and a biological antecedent stage of regression; and in the ejaculation he sees nothing less than the desire, in the sense of autotomy, to cast off the organ under tension, erection perhaps being only an incompletely achieved attempt to detach the genital, a kind of self-castration. This, it seems to the reviewer, is a most phantastic speculation and strains to the utmost the biologic facts on which it is presumably based. J. Arthur Thompson (*Biology for Every Man*, E. P. Dutton & Co., Vol. 1, p. 209) describes autotomy in the crab, in which it is best studied. When a walking leg is seriously injured toward the outer end, the crab throws off the bulk of the appendage; immediately thereafter, a thin membrane covers the wound, a process of rejuvenation begins to take place and a new limb is soon formed from the stump. Thompson emphasizes that in order that such autotomy take place it is necessary that there be a serious injury to the leg, or at least that the animal perceive that it is in danger. Thompson emphasizes that the amputation is essentially in the nature of a reflex action that does not require the activity of the brain and has very little in it to suggest instinctive behavior.

With reference to the emotions during the sex act, Ferenczi would maintain that the mutual attraction is nothing but the expression of the phantasy of veritably merging one's self with the body of the partner or perhaps of forcing one's way into it (as a substitute for the mother's womb). The physical concomitants of the act of coitus (dyspnoeic respiration, increased pulse rate, etc.), suggest to Ferenczi a recapitulation of the process of accomplishing the extensive adjustment which the change from the foetal to the extrauterine mode of oxygen supply demands. But it suggests itself to the reviewer that similar physical manifestations are observed under all sorts of tension conditions that have no relation to the sex act.

Ferenczi notes the lessening or abolition of consciousness during and following the sex act, this suggesting to him the unconscious hallucinatory attainment of the goal of coitus on the part of the individual as a whole, the attainment of the intrauterine state. He also finds a close relation between coitus and sleep. He points out—there obviously can be no clinical proof for this—that the first sleep of the newborn is a replica of the intrauterine state. The infant, shaken by the traumatic experience of birth, is soon lulled back into this sleeping state which creates in him a feeling as if no such shock occurred at all. The body posture of the first sleep is that of "foetal posture." There is thus a relation between coitus and sleep, for the penis and the genital secretion do in fact penetrate the uterus. Characteristic of both phenomena, sleep and coitus, is the exclusion of external stimuli, the abandonment of sense of reality, both thus imitating the intrauterine mode of existence. In further favor of a genetic relationship between sleep and genitality there is likewise the frequent occurrence of autoerotic, masturbatory and pollutional acts in sleep, and this is perhaps also one of the causes of enuresis nocturna. The neutral state in sleep compares with that of orgasm; there is complete gratification, freedom from all desire, a true reestablishment of the intrauterine state of repose. Dreams are concerned either with the œdipus phantasy,

or existence in the maternal womb, or return thereto. Ferenczi adduces many more comparisons between sleep and coitus, which are presented with great ingenuity, even if not always entirely tenable, but all toward maintaining the main thesis, namely, the return to the maternal womb.

In sleep and intercourse, the entire psychic organism, and in some degree the organic also, regresses to the ante-natal and probably likewise to a phylogenetically ancient mode of existence. According to the same assumption, sleep and coitus, but especially the development of the amnion with its amniotic liquor, and internal fertilization and intrauterine development in general, are nothing but arrangements for the reestablishment of that seemingly superseded period of development. The similarity of this process to the psychic processes of repression and return of the repressed immediately occurs. This explains to Ferenczi developmental efforts in biology; the wish for restoration of a compulsorily relinquished state of equilibrium never completely ceases to exist, but, temporarily put aside, is prevented of realization by a biological censorship which is created by contemporary ego-interests; a modification of the pleasure principle in the biological sphere.

Thus, on Ferenczi's hypothesis, coitus is naught else than the freeing of the individual from the burdensome tension, simultaneous with the gratification of the impulse toward regression to the maternal womb and to that prototype of everything maternal, the sea. The sensation of orgasm is looked upon as representing not only the repose of the intrauterine state, the tranquil existence in a more friendly environment, but also repose of the era before life originated, in other words, the death-like repose of the inorganic world. This would seem to be a restatement by Ferenczi of Freud's concept of the death instinct; an expression of the second law of thermodynamics. As further evidence of the regressive death trend, Ferenczi states that life always has to end catastrophically, even as it began in birth, with a catastrophe. . . . . Immediately before the individual breathes his last, often indeed somewhat sooner, complete resignation supervenes, nay, expressions of satisfaction which proclaim the final attainment of a state of perfect rest, somewhat as in the orgasm after the sexual struggle which has preceded it. Death exhibits utero-regressive trends similar to those of sleep and coitus, once again expressing regression to the maternal womb. . . . . The first sleep of the newborn is described by Ferenczi as a rather faithful replica of the state of repose existing prior to birth. This condition of sleep, as indeed is likewise true of every subsequent sleep, may signify the hallucinatory gratification of the wish to be in the unborn state. So far as the reviewer can see these are nothing but words and phrases given for the reader to accept it on mere say-so, but submitted without the least proof. And this is the greatest pity, the largest defect of the book, for though Ferenczi has had an exceptionally rich experience in analysis, he has not found it desirable to submit any clinical material whatever in support of his speculations. Not a single case is quoted in the entire book.

Ferenczi looks upon conjugation in unicellular organisms as the prototype of fertilization as seen in higher animals. To bring this in line with the problem of human coitus, Ferenczi prefers to see in conjugation "a refined

sort of eating each other up on the part of the sexually conjugating organisms," neither one of the participants succeeding in annihilating its opponent. Such biologizing by Ferenczi does not at all accord with what we know about the process of conjugation, which is regarded neither as a cannibalistic nor a symbiotic process, but essentially as one of rejuvenation. (See for instance: Sonneborn, Tracy, M. *Sexuality and Related Problems in Paramecium.* The Collecting Net, Vol. XIV, No. 4, July, 1939.) Even a premedical student in biology knows that much. The works of Just, Streeter, Sonneborn and others, show quite clearly that in the process of conjugation, accumulated waste matter is eliminated, the two cells having given each other the best that is in them, can now survive, whereas they would have perished otherwise. After the rupture of the graafian follicle, the human ovum, unless promptly fertilized by the spermatozoon, soon perishes; spermatozoa are being constantly destroyed in the human body unless given an opportunity to fertilize. It is the mixing up of complementary impedients that starts and keeps the fertilization going, not their eating each other up.

Throughout the study, Ferenczi would emphasize the preferential position of the male sex in the sex act. This is merely a restatement of Freud's view that all libido *per se* is "masculine," even when, as in the female, it seeks goals of gratification of a passive type. This view, however, has long been questioned by many competent analysts. Paul Bousfield has traced it to Freud's over-estimation of his own unanalyzed masculine component and, to him, unrecognized drive for power. But when Ferenczi says that this principle is applicable to the entire animal kingdom, he shows a definite lack of knowledge of established biological facts. In the lower animal kingdom it is the female that is the dominant sex. Many insects are ruled by queens, not by kings. In all too many instances the female is so much larger than the male, that the two may look as if belonging to different species. In many instances the male is so small as to be nothing more than a mere appendage to the female. Some males live only for a single erotic expedition, and this done, they die. In many instances, the male is not needed at all, as in parthenogenesis, during which the female is able to reproduce generations after generations. Commenting on the importance of odor in relation to sex activity, Ferenczi states that the rabbit becomes impotent if his olfactory nerves are cut. The reviewer has it on good authority that this statement does not at all accord with established anatomic facts of experiments. He cannot take further issue on this as throughout Ferenczi does not give specific references or sources. But the author reaches the nadir of nonsense when, in speaking of odors as sexual stimuli, he states that the special odor of the female genitalia has a sexually exciting effect "even if only through the fact that the odor awakens the longing for the mother's womb." No further comment is necessary.

Ferenczi is not inclined to accept Darwin's principle of natural selection as a factor in evolution, because it ascribes all change to accident and leaves nothing to the workings of, so to speak, immanent tendencies. By preference he is inclined to Lamarck's more animistic and more psychological idea of evolution, because it postulates a motive and concedes a rôle in phylogenesis

to motives and instincts. He would posit that the bioanalytic conception of developmental processes perceives everywhere at work only the wish or desire for restoration of earlier stages of life or death. Bioanalysis (the analytic science of life) has learned from the psychoanalytic study of hysteria, that the mental power of the wish is operative in the organic sphere also, that a wish may become "materialized" in the body and may even remold the body in accordance with a program of its own. Such wishful strivings must also operate in the biologic unconscious; the adjuvant rôle played by the wish is a factor in evolution in the sense of the Lamarckian theory of adaptation. To the reviewer, this seems an altogether free and gratuitous interpretation of Lamarckian concept, which correctly stated means only that such modifications as arise in animals during their lifetime come as a result of external environmental stimuli, adaptations being a direct result of use and disuse; but that environment cannot influence directly the organization of animals. Such concept implies in no way and cannot be interpreted as an expression of an unconscious instinctive drive, and to ascribe this to Lamarck is, to speak charitably, to grossly misinterpret his views. Further, Ferenczi seems to think that the modern concept of evolution is still represented by Darwin's original view. This is far from being correct. There are many theories of evolution, the most prominent one being represented by the geneticists and cytologists respectively. (See Morgan, T. H. *A Critique of the Theory of Evolution;* Dobzhansky, Th. *Genetics and the Origin of Species;* Just, E. E. *The Biology of the Cell Surface.*)

Lest the reader get the impression that the reviewer has been overcritical in his charges, it is only fair to state that Ferenczi himself has from the beginning been quite acutely conscious of his limitations as a biologist. The work was completed during the World War, when he had no access to other sources of reference but a few textbooks and treatises of the older biologists, practically all antiquated; and by his own admission he had no access to the more recent works. Constantly and profusely, he keeps on apologizing for indulging in speculations for which he admits he has no basis or justification; nonetheless, and immediately afterwards, he again plunges headlong into more speculation in the manner of the man, who having just purged himself of guilt by confession, immediately goes on blissfully in his own sinning way. In so far as he speculates on purely psychoanalytic lines, it is still more or less legitimate, even if there may be many a reader who would not accept these speculations. He outdoes himself even more than the medieval monks who speculated on how many angels could dance on the point of a needle, when, for instance, he posits that the first dream of the new born concerns returning to his mother's womb, from which it has just emerged with it as a wish fulfillment and as an argument pro of the universal wish of regression to the maternal womb. It is doubtful whether even all orthodox Freudians would be willing to accept such speculations. But he completely misses the point when he attempts to speculate on the psychoanalytic aspects of some biological phenomena. Facts are strained to the utmost of human credulity and given a meaning which they could not

possibly possess. One can only admire the marvelous ingenuity of the man who can extract so much speculation out of so few facts. The reviewer read and studied this book with wonderment and disappointment; wonderment at the intellectual agility, disappointment at the so few and meager results obtained. A psychoanalytic study of biological phenomena is yet to come. It is quite certain, however, that it will not come from anyone who is primarily trained in psychoanalysis, but in all likelihood from the close cooperation of two individuals oriented biologically and psychoanalytically respectively who will complement each other and perhaps inhibit each other in getting too far away from established facts.

For all the limitations as pointed out, the book is worth owning and reading for the stimulating and intriguing suggestions it contains. The translation is excellent; though the book is difficult and involved, the text reads easily and smoothly.

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CLINICAL AND EXPERIMENTAL STUDIES IN PERSONALITY. By *Morton Prince.* Edited by *A. A. Roback, Ph. D.* (Cambridge, Mass.: Sci-Art Publishers, 1939.)

By giving the best of Prince's work this book becomes a fundamental text for both clinicians and investigators in the field of abnormal psychology. Dr. Roback is to be congratulated for having ably completed his editorial task; his preface and essay on "Prince's Place in Psychology" make an excellent introduction.

The chapter on "The Rôle of Meaning in the Psychoneurosis," though not a deep analysis in the Freudian sense, shows that in 1912 Prince was far ahead of his time in comprehending the importance of subconscious motives. In fact, his description of subconscious and conscious phenomena is more exact than the unreal, dualistic division of everything into conscious or unconscious, of the more recent psychoanalysts. The chapter on "Association Neurosis," modified from the original paper of 1891, shows Prince as a pioneer in the field of "conditioned reflexes." Hypnosis is too often disregarded by psychologists; it used to be important in therapy and in fact may still be more important than one realizes. Prince gives good evidence (chapter V) that what one usually calls hypnosis is an artificial narrowing of the field of consciousness associated with de- and re-personalization. States of abstraction, revery, absent-mindedness, trances and fugues are the same sort of phenomena. The state of abstraction induced by lying on the couch during psychoanalytical free-association may be an important element in the therapeutic repersonalization. Prince does not say what hypnosis is; he calls it a riddle; but at least he logically reduces it to component phenomena which one can better understand: inhibition, dissociation, depersonalization and repersonalization.

Other chapters are on such problems as consciousness, dreams and hallucinations. They show the same keen, logical mind willing to use in a

catholic way data from academic psychologists and experimental psychologists, neurologists and psychiatrists. Prince was at times polemical, but he rode no hobby. Most of his work had little popular acclaim during his life, but in the ten years since his death the study of personality, which he did so much to initiate, has made important advances. The first edition of this book, which came out in 1929 is exhausted, and readers today will find this new and enlarged edition of permanent value.

S. C.

CRIME AND THE COMMUNITY. By *Frank Tannenbaum.* (New York: Ginn and Company, 1938.)

Crime and the Community presents the thesis that the professional criminals are a natural product of the community. To support the thesis the book cites an unusual amount of material drawn from reports of investigations such as the Seabury Investigation and from Thrasher's *Gangs* and similar classical treatises.

Perhaps for the average reader the most interesting are the early chapters, "Education for Crime," "Organized Crime," "Politics and Crime" and "Politics and Police." These chapters carefully portray the gradual transition of the individual from a thoughtless, carefree young truant into a hardened professional crook, and afford a liberal education for those who have no direct contact with the criminal courts. Here the author discusses various environmental factors including the effect of a low cultural neighborhood and association with older companions of criminal tendencies, the crystallizing and hardening effect caused by the reaction of the rest of society and climaxed by arrest, the inefficiency of our penal systems, the subtle linkage of politics with gangster activities and the "manufacture" of new crimes by new legislation. These aspects of the crime situation indicate the inevitable development of criminals and explain the demoralization of would-be honest and efficient officers of the law because of certain conditions too often existing in our cities.

Chapter VII on "The Philosophy of the Professional Criminal" gives an excellent picture of the manner in which the criminal rationalizes his aggressive life. The author says: "And so we come to a summary of the attitude. The professional criminal has grown up in an atmosphere of conflict and in a world filled with enemies. All life is a life of war, with the danger of treason and death always lurking in the shadow, and all this in a world which seems corrupt and dishonest. From childhood up he seems to himself to have had the wrong and the unfair end of life thrust upon him. He has always been persecuted and mistreated. Other men as much implicated as he have escaped, some have achieved power and wealth and influence and public standing, but he, for reasons beyond his own comprehension, has been mistreated. All talk of honesty, goodness, justice, morality, he knows to be false and hypocritical."

In chapter VIII the author points out the weakness of past and present theories of crime. He cites the Lombrosian theory and the familiar explosion of this theory by Goring's exhaustive study. He made an excellent review of

the shortcomings of present day psychological and psychiatric theories of crime and calls attention to the wide disagreement among psychiatrists with regard to the factors underlying the behavior of criminals. He aptly states: "The complete absence of standardization of methods, of standardized definition of terms, of invariable classification of traits, makes the work of a psychiatrist an individual personal judgment not comparable with the work of other people and not reducible to statistical form and percentage bases."

Professor Tannenbaum then proceeds to outline the specifications of a theory of criminology. Having already formulated in several different places the proposition that crimes are a natural outgrowth of society's existing institutions, he says here: "A theory of criminology must first of all be a theory of behavior, of which criminal conduct is only a part. . . . . Unless and until our theory is broad enough to explain the growth and development of all types of integrated behavior systems, it cannot hope to explain any of the forms of behavior, criminal or otherwise." Here to my mind the book reaches its highest point, for in this paragraph the author seeks for an explanation that goes beyond the obvious. Here he throws open the question of crime as being but one aspect of human behavior. One wonders, though, how the author could have overlooked in his long list of references so pertinent a study as Trigant Burrow's "Crime and the Social Reaction of Right and Wrong," which appeared in 1933 in the *Journal of Criminal Law and Criminology*, as well as some of his other writings. Burrow, who has for years investigated the social or community manifestations of the neuroses, finds crime to be but one of the numerous expressions of this broader behavior anomaly.

In part two of *Crime and the Community* the author proceeds to other aspects of the problem and exposes the distortions and injustices of what is called the administration of justice. This is another very informative section.

The third part of the book embodies a comprehensive account of the punitive process and stresses the most important aspect of this phase of the problem, namely, its utter futility. Included is a discussion of the two more hopeful projects, parole and probation. The final chapter summarizes the discrepancies of our procedure in dealing with crime. To quote: "One thing is clear, that what we do now is wrong, perhaps completely wrong. We have failed to reform the criminal. In plain honesty we ought to admit that in what we have been doing we have missed our ends. The criminal career, once set upon, has apparently defied our efforts to redirect its energies into more social paths." The book ends with the simple but forceful statement that we require an alternative to punishment and to our attempts at reformation of the criminal.

I am again reminded of the broader biological thesis courageously set forth by Burrow in his book *The Biology of Human Conflict*. Here is no mincing of issues, no dalliance with what Tannenbaum calls the "individual personal judgment" of this or that psychiatrist, but a square confronting of the mind of man and its disorders of function, as these disorders are incident to man's "affecto-projective" or "socio-symbolic" development as a species.

We who are psychiatrists and criminologists, we who are students of behavior generally, cannot but agree with Tannenbaum's sweeping indictment in regard to the incompetence and ineffectiveness of our procedure. As one eminent psychiatrist recently remarked, "There are many things in psychiatry which are extremely nebulous." But our attack has to be upon a broader front. To restrict the definition of crime only to the conduct of those whose quest for economic security leads them to defy the prevailing mores is one-sided and short-sighted. A clear encompassment of crime requires that we include the investigation also of those whose quest for economic security finds its guarantee no less in *conformity* to a socially prescribed norm of conduct. For it is quite overlooked that conformity to the socially current mores merely for the sake of one's own private advantage is as antisocial as the criminal individual's more frankly ruthless deviations. But this wider encompassment includes ourselves as well as "the criminal" whose social deviation we too easily judge by contrast with our own conformities. It includes ourselves despite our obstinate unwillingness to be thus included in so antisocial a category. It is some such phylobiological breadth of outlook toward which Tannenbaum's able thesis would seem to be groping, and in his forthright questioning of ourselves and our own psychiatric responsibility the author has taken high ground.

Crime and the Community stimulates thought on the problem usually referred to as crime and is a book which should be widely read.

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A MANUAL FOR STATE HOSPITAL ATTENDANTS. Second edition. From the Elgin State Hospital, *Charles F. Read, M. D., Managing Officer.* (Illinois: Department of Public Welfare, 1939.)

Most, if not all, hospitals or systems of hospitals have devised an instruction manual for their personnel; few, however, have attempted anything as pretentious as this of the Illinois State Hospitals, covering, as it does, the whole range of anatomy, physiology, psychology, social service and social relationships, as well as the usually included fields of management, therapeutic procedures and psychiatry. With such an ambitious scheme it is not surprising that parts of the discussion should be sketchy and superficial. The only question which would arise would be that of its practical value and since the present copy is a second edition that question would seem to be satisfactorily answered. The repeated emphasis on the attendant's part in the program of treatment rather than his merely custodial functions, the stress on attitude, personal appearance, kindness and understanding throughout the whole didactic portion of the book, reinforced by the unobtrusive explanation of rating scales of efficiency in one of the concluding chapters, are features worthy of special comment.

Some of the rules and procedures are designed of course with a view to the Illinois hospital system and might not prove suitable in other settings, but with this reservation in mind the book is one that might be adapted

readily to the special requirements of many other hospitals. Certainly those who have as one of their responsibilities the training and supervision of hospital staffs would be well repaid for the time spent in reading this manual.

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STUDY OF THE DISTRIBUTION OF MEDICAL CARE AND PUBLIC HEALTH SERVICES IN CANADA. (Toronto: The National Committee for Mental Hygiene (Canada), 1939.)

This report constitutes a work of reference of very considerable value. It represents the first attempt to present in a single volume "a factual picture of public health and medical care services in Canada."

All existing agencies dealing with various aspects of the health problem are covered in concise form, with discussions of administrative setup and statistics showing volume of work, operating costs, etc. The survey, based upon the latest reports from all parts of the Dominion, deals also with the economic aspect of illness with respect to existing facilities and present and future needs.

This study was undertaken by The National Committee for Mental Hygiene (Canada) through its Division on Public Health and Medical Services (Grant Fleming, M. D., Hon. George Hoadley) because of the conviction that a service organized to foster mental health must base its policies and plans upon an appraisal of the total health situation in the area it seeks to serve. The report under consideration represents such an appraisal.

In its concluding chapter the report reviews the distribution of medical facilities and public health services in the Dominion and submits some carefully thought out recommendations to equalize, extend and improve medical care throughout Canada.

Medical administrators, public health officials, in fact all who are interested in the professional and economic aspects of medical care as a public service will find this study a full and convenient source of information.

C. B. F.

In Memoriam.

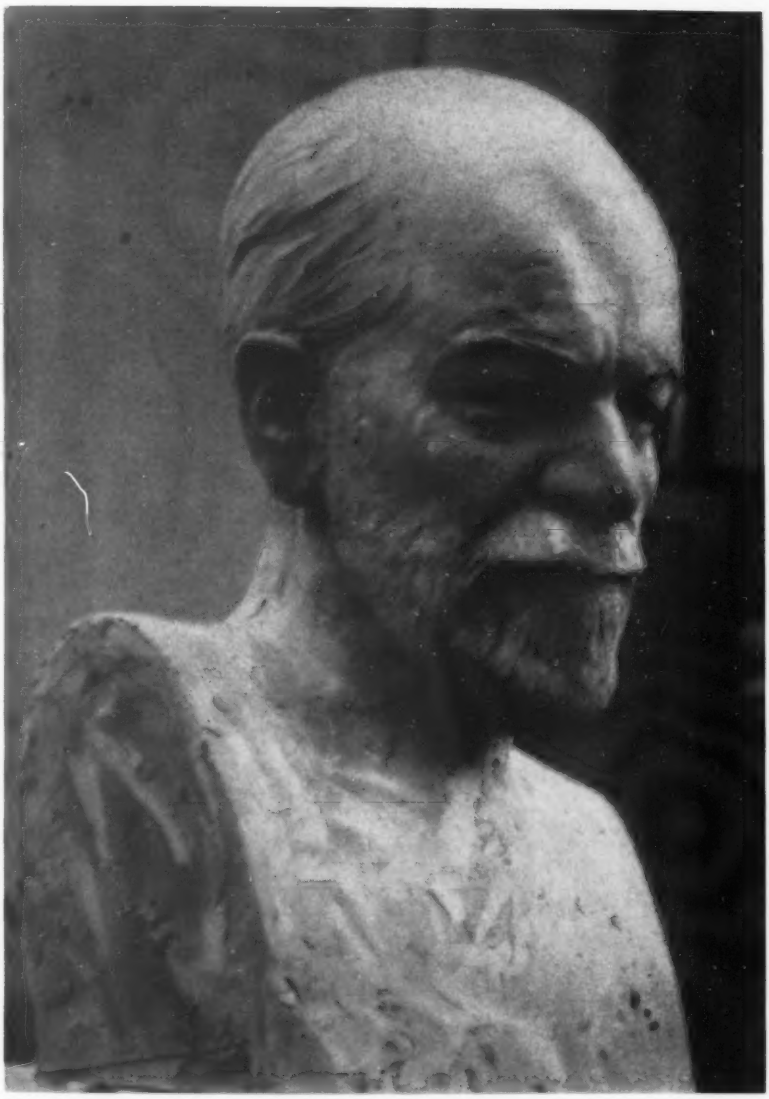
SIGMUND FREUD.

1856-1939.

It is difficult to give verbal expression to the loss which we have sustained in the death of Sigmund Freud. Although he died at the mature age of 83 years and left us 12 volumes of his *Gesammelte Schriften* (Collected Papers) and additional works, enough to fill two more volumes, his absence will be sorely felt. For unlike those who gradually slow up with advancing age he was an active and productive worker until the end. His last work, *Moses and Monotheism,* appeared in English only a few months ago, and like all his other works is pregnant with new and stimulating ideas, and no less provocative than *Sexuality in the Neuroses* and *The Interpretation of Dreams,* which appeared in 1898 and 1900 respectively.

Outside of his more intimate circle, few knew that he had been the victim of a carcinoma of the superior maxillary for over 15 years. The malady was fairly well controlled until recent years by surgical and other procedures, but the frequent operations and the subsequent adjustment to new dental plates irritated and often incapacitated him more or less physically. For the last four or five years he was also suffering from endocarditis and was often confined to bed for short and long periods. Yet, such was his indomitable spirit that during all these years of suffering he continued his practice; he saw patients up to about five weeks before his death and published some of his best works. The last two volumes of his *Gesammelte Schriften* (Vols. XI and XII) are made up of works which he produced during these years of invalidism.

Those of us who were in close contact with him, and have seen him from time to time, were amazed at his composure and courage in the face of this dreadful situation. I saw him last in July, 1936,



DR. SIGMUND FREUD.

while he was confined to his bed following an operation. He was visibly uncomfortable, but his mind was as clear and alert as ever. As I listened to his conversation, which despite his discomfort was now and then punctuated by witty remarks, I was perforce reminded of our first meeting in 1907. One of the deep impressions that I carried away when I left him at that time was his incomparable sense of humor. In our conversation I hald told him that Bleuler, whom I had visited on my way to Vienna, wished to be remembered to him. He then mentioned the fact that he had seen Bleuler a few months before, and remarked that although Bleuler was of his own age he seemed as hale and energetic as ever. This was the only indirect complaint that he made about himself throughout the week that I visited him daily.

No justice can be done to Sigmund Freud in a cursory review of his brilliant and outstanding career. In his autobiography he tells us that he stood first throughout his college years. Of all the medical specialties psychiatry alone appealed to him. He decided to study medicine because he wished to study the human mind. Judging by the status of psychiatry at that time his preparation for it seemed most adequate. He spent six years in Bruecke's physiological laboratory (1876-1882) as a research student. He began his studies on the spinal cord of a lowly fish, the *Ammocoetes Petromyzon*, and then turned his attention to the central nervous system of man, devoting most of his studies to the *medulla oblongata*. From Bruecke's laboratory he went to the Institute for Cerebral Anatomy, where he worked with Meynert. The results of these anatomical and histological investigations can be seen in his works on cerebral diplegia and aphasia, and reading them today, one can already discern the paths which led him from the organic to the psychic.*

That he was already at that time looking beyond the horizon of orthodox psychiatry is quite evident from his interest in cocaine. This alkaloid was little known in 1884 when Freud investigated its local anaesthetic effects. He told about his interest in this drug to two of his friends, Drs. Koenigstein and Koller, and suggested that they who specialized in ophthalmology might test the anaes-

* Smith Ely Jelliffe: *Sigmund Freud as a Neurologist*, Journal of Nervous and Mental Disease, Vol. 85, No. 6, June, 1937.

thetic effect of cocaine on the human eye. Dr. Koller after testing its effect on animals then demonstrated the local effect of cocaine on the human eye before the Congress of Ophthalmology in Heidelberg.

This little known episode in his early scientific career shows that whereas consciously the enigma of the human mind, as such, attracted him to medicine, unconsciously he was primarily interested in the alleviation of human pain and suffering. For he was well aware that the therapeutic procedures then in vogue were ineffectual in the treatment of the neuroses and were based on mere guesswork. But to find the remedy one must know first the nature and mechanism of the disease.

For his meritorious work in neuroanatomy Professor Bruecke warmly recommended him for a stipend to enable him to spend a year (1885-1886) in Paris. There he became one of Charcot's favorite pupils, as well as his German translator.

When he returned to Vienna, he wished to demonstrate what he had learned from Charcot—namely, that there is a "male" hysteria and that hysterical symptoms can be produced and removed by suggestion. These views were so alien to the Viennese school of neurologists and psychiatrists that he soon became the object of strong opposition. It is usually believed that the resistance to Freud and psychoanalysis began in 1898 when he emphasized the sexual factor in the neuroses. As a matter of fact, his Viennese colleagues looked askance at him from the time he demonstrated Charcot's views. His statement that in a normal *vita sexualis* no neurosis is possible, which he said 12 years later, simply enhanced this already existing opposition. By this time both he and the old school were already conditioned to mutual opposition and distrust.

His career since then is too well known to dilate upon here. The best that one can do is to mention a few formulæ, a few catchwords, as it were, to keep the record straight, and leave the rest to the reader. With Breuer in 1893 he published *The Psychic Mechanisms of Hysterical Phenomena,* which later formed the first chapter of the *Studies in Hysteria.** In this epoch-making

* Translated by A. A. Brill, Nervous and Mental Disease Monograph Series, No. 61, New York, 1936.

work the authors described the origin, but not the nature of hysterical symptoms. They emphasized the importance of the *affective* life of the individual, the significance and difference between *conscious* and *unconscious* mentation, and stressed the *dynamic* and *economic* factors in the symptoms by showing that the symptom originates through a damming up of an affect and through the transformation of a sum of energy, which should have been utilized elsewhere (*hysterical conversion*). The patient was cured by means of *abreaction*, by re-living the repressed episode through verbal expression. It was a sort of mental purging or *catharsis*. The technical procedure was to hypnotize and then question the patient concerning the origin of the symptoms.

The mounting opposition to these new concepts caused Breuer to withdraw his collaboration. Freud then continued alone. The first and most important change in the technical procedure instituted by Freud soon thereafter was to eliminate hypnotism and substitute it by what he called the "free association" method. This procedure was more difficult and more time consuming, but it led directly to the deep recesses of the mind and showed how human beings actually react to the demands of life. The free association method marks the beginning of psychoanalysis, which Freud gradually developed and amplified into a system of study and treatment of normal and abnormal activities. But as he traced the origin of the symptoms, the associations perforce led him to the childhood of the individual and thence, to that of the race. One can clearly see this course of evolution by following chronologically his basic productions.

The Collected Papers on the Neuroses, The Psychopathology of Everyday Life, The Interpretation of Dreams, his books on *Sex, Wit,* and *Totem and Taboo* * show: (1) that normal and neurotic behavior differ only in degree, (2) that sexuality plays the same important rôle in both, (3) that in our unconscious, *i. e.*, in our dreams we recapitulate not only our recent and remote past, *i. e.*, our childhood but also the childhood of the race. Sigmund Freud was the first mental scientist who dared take the decisive step from the present to the primeval past of mankind. In 1912

* *The Basic Writings of Sigmund Freud,* The Modern Library, New York, 1938.

he published his *Totem and Taboo,* in which he demonstrated the close resemblance between the psychic life of the neurotic and the savage, and by comparing the results of the unresolved conflicts of the modern child to the taboos of savages he opened new vistas to the origin of most of our cultural institutions. In his last work, *Moses and Monotheism,* he continues these trends of thought and demonstrates through monotheism that the Œdipus complex, which is common to all children, is a direct transmission from the past.

The amount of material that he collected in his effort to coördinate these various steps is prodigious. Sigmund Freud not only made great contributions to normal and abnormal psychology, but also to biology, mythology, folklore, sociology, anthropology and literature. His direct contributions to psychiatry cannot be too highly evaluated. He gave us original concepts on the neuroses and psychoses. No psychiatrist can now afford to omit the psychogenetic and unconscious factors in the study of patients. His theories of paranoia and melancholia have opened new vistas to these puzzling diseases. These are only a few of the high spots of his contributions.

Psychiatry would be quite barren if his contributions were taken away from it. The interpretative psychiatry, of which Bleuler is the spiritual father, was made possible through the tools invented by Freud.

Sigmund Freud represents a distinct era, not only in the development and progress of psychiatry, but also in modern culture. His death has deprived the psychiatric world of its most ingenious and most original worker, the civilized world of one of its greatest thinkers, and The American Psychiatric Association of its great honorary member. Whether all his views will stand the test of time does not matter; there is no doubt, however, that he has influenced the world in a fateful manner. One can truly apply to him the words of Cicero: *"Vita enim mortuorum in memoria vivorum est posita."* His life's work is stored in the memory of the living. He survives through his great works and through his numerous followers the world over, and psychiatry is richer and better because he lived.

A. A. Brill, M. D.



DR. ALBERT COULSON BUCKLEY

ALBERT COULSON BUCKLEY.

1872-1939.

Dr. Buckley was born in Philadelphia, the son of a doctor who had a large general practice of medicine and because of this hard grueling family practice it was the father's wish that the son would choose some other vocation but, as it has been often said, "A true scientist is born, not made"—so it was in the case of Dr. Buckley. Before graduating from Central High School his interest and enthusiasm in his course in physiology and the use of the microscope were so keen and so fixed at that time that he could never consider any vocation other than the science of medicine.

He graduated from the Medico-Chirurgical College in 1897 and for several years continued as demonstrator of anatomy and histology until 1900 when he was made associate professor of histology. About this time he made his first trip abroad and spent six months working and studying in the laboratories and wards of the Berlin City Hospital.

In 1906 he went to the Friends Hospital as assistant physician and in 1918, upon the retirement of Dr. Robert H. Chase, Dr. Buckley was appointed superintendent, which position he held until the time of his death.

Thirty-three years of service in one institution is a long time but I don't think it seemed long to Dr. Buckley. He was always busy, thinking and planning as to what might be done for the betterment of science, his hospital, those entrusted to his care, and social problems in the community at large. Not only was he anxious to study and learn for himself, but much of his time was spent in teaching, and he was on the staff at the University of Pennsylvania, the Post Graduate School of the University, the Philadelphia General Hospital, and was chief of the neurological service of the Frankford Hospital. He belonged to both local and international psychiatric and neurological societies and was not an infrequent contributor to the scientific medical journals on subjects pertaining to both psychiatry and neurology. In 1920 his textbook *The Basis of Psychiatry* (psychobiological medicine) was published, and in 1927 appeared the first edition of *Nursing Mental and Nervous Diseases* (from the viewpoints of biology, psychology and neurology). This latter book has passed through five editions

and has been used extensively in Canada and England as well as in this country.

He made several trips abroad visiting the different clinics and laboratories in Europe and upon each return came enriched with knowledge, enthusiasm, and the same spirit of true scientific research. His latest study was on *Myelination in the Central Nervous System of the Albino Rat treated with Thymus Extract.*

In 1904 Dr. Buckley married Harriet Ellis Bailey of Kentucky. Mrs. Buckley passed away November 6, 1937, and on January 2, 1939, he married Mary Sandifer Palmer of Fort Thomas, Kentucky. The cause of his death, which occurred on August 17, 1939, was cardiovascular collapse complicated with bronchopneumonia.

JAMES P. SANDS, M. D.

ARTHUR H. HARRINGTON.

1856–1939.

In the death of Dr. Arthur H. Harrington, American psychiatry lost not merely one of its oldest members but one who through his courteous manners, kindly spirit and love of the fine arts stood high among those who have lent it culture and dignity.

Dr. Harrington was born in Worcester, Mass., July 25, 1856, and died in Providence, R. I., March 12, 1939. Springing from a line of physicians, merchants and teachers, he retained the best of a New England heritage and added a catholicity of mind that this has sometimes lacked. He graduated from Brown University in 1879 and received the degree of Doctor of Medicine from Jefferson Medical College in 1882. After two years of private practice in Providence Dr. Harrington entered upon his psychiatric career, his introduction being as assistant physician at the Danvers State Hospital. In 1894 he was appointed medical director of the Bridgewater State Farm, an institution caring for a heterogeneous group of indigent infirm, chronic alcoholics, minor offenders and criminal insane. Largely through the efforts of Dr. Harrington a department known as the Massachusetts State Asylum for Insane Criminals was established for the purpose of providing psychiatric care and treatment for the neglected group last named. Here he appointed as his assistant a young physician just graduated in medicine, Dr. Harry W. Mitchell, whose distinguished career as a

psychiatrist and as president of The American Psychiatric Association is already recorded in the JOURNAL. In 1898 Dr. Harrington returned to Danvers State Hospital as superintendent, remaining until 1903 when he resigned to become superintendent of the New York Eye and Ear Infirmary. Through his business ability and sound judgment he soon rescued this institution from the financial disaster which threatened it. Dr. Harrington's real interest was in psychiatry, however, so when, in 1907, he was offered the superintendency of the Rhode Island State Hospital for the Insane, he promptly accepted the appointment.

In going to Rhode Island Dr. Harrington regarded the situation as a challenge. The physical plant of the institution was in wretched condition and there had been great lag both in the care and treatment of patients and in psychiatric interest. By enlisting the aid of the medical profession and by wisely informing both the public and legislators concerning the needs and work of the institution he gradually transformed the physical plant, at the same time making improvements in the standards of psychiatric and other medical activities. Perhaps the most important of the physical improvements accomplished by Dr. Harrington in the hospital at Howard was the construction in 1912 of a reception building. This was probably one of the most complete and best adapted buildings which had then been constructed for the purpose indicated. Although always handicapped by limited appropriations Dr. Harrington established a laboratory, introduced occupational therapy, social service and other scientific and therapeutic facilities as they became recognized as useful psychiatric instruments.

A subject in which Dr. Harrington felt the keenest interest and developed to the pleasure and benefit of his patients was music. A great lover of this art and a skillful pianist he believed that, properly directed, it could be made a valuable therapeutic agent, not so much through the instruction of patients in its technique as by group participation in its production, especially in the form of chorus singing. Although he had already highly developed this form of music Dr. Harrington recognized the need of a pipe-organ as accompaniment in the large congregate dining-room where the patients were accustomed to assemble for singing. By means of a personally directed drive, he raised $10,000 through contributions from the public and secured the desired instrument.

Dr. Harrington was doubtless the foremost of American psychiatrists in advocating music as a form of therapy for mental patients and often contributed papers or discussion on this subject. In 1930 he was invited to participate in a conference and discussion held in Paris on painting, drawing and sculpture produced by patients in a mental hospital. His contribution, "Music as a Therapeutic Aid in a Hospital for the Insane" was published in *Archives Internationales de Neurologie.*

In 1926, having passed his seventieth birthday, Dr. Harrington resigned as superintendent. After a period of European travel with his old friend and Brown classmate, Dr. Walter L. Monroe, he returned to Providence where he continued to be active in the Rhode Island Society for Mental Hygiene of which he was a founder and past president and in various other community projects. During the last three years of his life a cardiac disability made a limitation of his physical activities necessary, but made possible the direction of his energies to a life-long dream—musical composition, particularly in the form of opera. Only a few close friends knew that this period was devoted to the creation of imagery based on Norse mythology and to its recording in verse and music. At the time of his death Dr. Harrington had completed a sequence of four poems in heroic verse in which he had received helpful suggestions from an old psychiatric friend, Dr. G. Alder Blumer. The accompaniment of these verses by some twenty interpretative musical scores had been nearly completed. On Sunday evening, March 12, while seated at his piano playing one of these scores to friends, he was suddenly seized with a fatal coronary attack.

The circumstances of his death were fittingly symbolic of Dr. Harrington's personality. He was a lover of literature and music, devoted to the welfare of his patients who held him in affectionate regard, loyal in his friendships, and generous and uncritical both of those who erred or of those who opposed him. During his career he witnessed many changes in psychiatric concepts with the evolution of which he was always sympathetic; he readily accepted and incorporated into his psychiatric thought the views of the present dynamic psychiatry and its added insight into human behavior. Although retired and nearly eighty years of age when the American Board of Psychiatry and Neurology was organized, he applied for

its certificate as an expression of his sympathetic support of the purposes for which the Board was created.

Dr. Harrington was a life member of The American Psychiatric Association, a member of the Boston Society of Neurology and Psychiatry, a past president of the Rhode Island State Medical Society, past president of the Rhode Island Society of Neurology and Psychiatry, and past president of the New England Society of Psychiatry. In spite of these many honors those who were privileged to know him best felt that his self-effacing modesty prevented him from receiving the even wider recognition which they were convinced his rare professional and personal qualities merited.

ARTHUR P. NOYES, M. D.

WILHELM WEYGANDT.

1870-1939.

Those who attended The First International Congress on Mental Hygiene in Washington in the summer of 1930 will recall that Dr. Wilhelm Weygandt was the representative of Germany at that memorable meeting and took a distinguished part in its proceedings. He was a member of the Committee on Institutions and participated in the founding meeting of the International Committee for Mental Hygiene. At the first general session at which was presented a world view of mental hygiene Dr. Weygandt spoke for Germany, outlining the status of psychiatry and mental hygiene in that country. "Some of the features most worthy of note in our system," he said, "are scientific research, intensive treatment by occupational therapy, and a very extended provision for the care of mental cases outside the hospital."

Professor Weygandt, who died at Wiesbaden, January 22, 1939, had for many years been one of the most conspicuous figures in German psychiatry. With a wide and informed interest in the arts and sciences he exemplified the qualities which a leader in psychological medicine needs to cultivate. He entered upon his professional work under especially favorable auspices. He had been a pupil of the great philosopher-psychologist Wundt * at Leipzig,

* The great Heidelberg philosopher Kuno Fischer is said to have summed up German philosophy in his day, thus: "Es gibt nur zwei grosse deutsche Philosophen—der andere wohnt in Leipzig."

the same under whom Kraepelin had begun his career, and with the latter he acquired his early psychiatric experience. With foundation thus well and truly laid, Weygandt's advancement was rapid. He was called to Hamburg where he remained for the rest of his professional life as director of the University Psychiatric Clinic and professor of psychiatry at the University of Hamburg.

A particularly significant contribution of Weygandt, fittingly culminating a long list of publications of wide range, was the *Lehrbuch der Nerven- und Geisteskrankheiten,* which in collaboration with other German psychiatrists and neurologists he brought out in 1935. Of the 640 pages of text, 200 were written by Weygandt, including the sections on mental deficiency and schizophrenia. With this publication he rendered the service of presenting the first German textbook covering the whole field of nervous and mental diseases in their natural relationship and between two covers. It is a valuable work and will remain as an authoritative exposition of contemporary German psychiatry and neurology.

With the death of Weygandt the psychiatric world lost one of its most distinguished leaders.

C. B. F.

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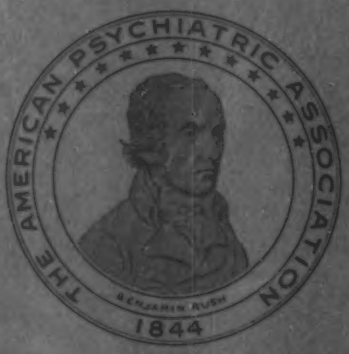
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THE BIOCHEMISTRY OF EPILEPSY.

A REVIEW.[1]

BY H. GOLDSTEIN, PH. D., AND R. A. McFARLAND,* PH. D.,
Columbia University.

"Affliction with seizures is an ancient and widespread condition. In the United States, if we may accept for the whole population the incidence among drafted men, something less than 500,000 persons are subject to 'epilepsy.' The lot of these persons is a difficult one. The care of those who need it constitutes a heavy charge on families or communities; their treatment is oftentimes no more rational than was given in the dark ages and beyond" (100).

If epilepsy is to be considered a distinct disease entity it surpasses any other single nervous or mental disorder in the number of its victims. Yet both understanding and treatment of this malady are still on a hit-and-miss basis.

It is true that the overt physical manifestations that we have come to recognize as the convulsions or fits of epilepsy are fairly easily discernible and tend to make the diagnosis of epilepsy too much of a snap judgment and too often erroneous. The mere recognition of a symptom does not imply that it is a symptom of only a single disease entity. It may very well be a symptom of many such entities. It is realized that many diseases have some symptoms in common and that some diseases have many symptoms in common. Just as a high temperature is characteristic of typhoid fever it is also characteristic of pneumonia. Yet no one but the most naive would say that these two conditions resemble each other in either etiology, diagnosis or prognosis.

"Increasing knowledge has demonstrated that pathological conditions of various types may cause similar manifestations" (178).

* Now at the Fatigue Laboratory, Harvard University, Soldiers Field, Boston, Mass.

[1] This article is the fourth and last of a series dealing with the blood biochemical studies of the psychoneuroses (116), dementia præcox (117), manic-depressive insanity (115), and epilepsy.

Many investigators have stated their belief that the so-called "epilepsy" is in reality a group of epilepsies, which may or may not bear etiological or physiological relationship although they are similar from a symptomatic viewpoint. Others look upon epilepsy as merely a symptom of some more fundamental, disturbed, general physiology of the body rather than as a disease entity. These individuals, therefore, should not expect any single abnormality to be present in all epileptics.

The label idiopathic epilepsy indicates a functional condition for which there is no known or obvious organic cause.[2] In view of the fact that this disorder may usually occur in conjunction with a well-functioning nervous system without lesions of any kind,[3] it must be apparent that any relation that may occur between the incidence of this disorder and defective brain and neural processes must be incidental or casual and not causal.

There are many investigators who believe that epilepsy is a disease "dependent upon a defective functioning of the whole organism" (*27*). This view, of course, would allow new approaches to be made to an understanding of the underlying etiology through such diverse fields as social and clinical psychiatry, physiology, behavioristic and social psychology, sociology, biochemistry and social anthropology.

While the textbooks are rather filled with exact and minute descriptions of the various clinical manifestations of petit mal, grand mal, Jacksonian epilepsy, status epilepticus, to mention a few, the underlying basis of this disorder has not kept pace with its descriptive phase. As a result we have had a vast multitude of causes assigned for its incidence without adequate empirical basis. Theoretical considerations have at various times given as essential reasons for seizures the presence of congenital brain disease, heredo-degenerative processes, traumatic lesions, tumors, parasites, syphilis, tuberculosis, cerebral abscess, brain swelling, meningitis,

[2] Kanner (*80*) has stated that "the diagnosis of 'idiopathic' epilepsy should be made only after a very careful physical examination has excluded the presence of neurological or gross metabolic disorders. When no such disturbances are found, the condition must be differentiated especially from hysteria, fainting spells, narcolepsy, breathholding spells and tetany."

[3] The lesions of the nervous system sometimes found in epileptics may very well be a result of the convulsive seizures rather than a cause.

chronic arachnoiditis, encephalitis, multiple sclerosis, presenile gliosis, senile cortical degeneration, toxins, disturbance of circulation, decreased consumption of brain cells, disturbed equilibrium of electrolytes and of acid-base elements in the tissues, edema of tissue and permeability of nerve cell membranes, decreased blood supply to the nerve cells, pathological chemistry of the brain substance, psychoanalytic repressions, demoniacal possession, failure of adjustment to environment, epileptic personality type, vascular spasms, cutaneous vaso-constriction, organic and functional abnormalities in basal metabolism, endocrine dysfunction,[4] toxicity of the blood, alcoholism, etc. And the theories are still being elaborated at great length. As can be seen from a study of the literature, specific lesions of one kind or another of the central nervous system,[5] functional instability of either the central or sympathetic nervous system, bacterial, toxic or metabolic damage, or psychogenic causes that have their origin in little demons that gloat maliciously over the wretched epileptic or a malfunctioning ego, have all at various times been accused of being *the* cause. But so far it has been found that neither starving brain cells nor acute indigestion nor chronic constipation nor phantastic toxins in the circulation nor "pin-worms embedded in the anal mucosa nor complexes buried in the personality are in themselves sufficient to produce a convulsion, though it is self-evident that such inciting causes when present should be removed" (100). There are no data to demonstrate that any of the above abnormalities are more common in individuals subject to epileptic attacks than in normal individuals. The theories of epilepsy have been plentiful but unfortunately the facts have been conspicuous by their scarcity.

Because of the plenitude of theory the therapies advocated have been numerous and varied. It must, however, be indicated that some measures of treatment have proved to be of value while others have been worthless. Lennox and Cobb (100) in an excel-

[4] "About all that investigation has demonstrated in regard to the connection between epilepsy and the various endocrine glands is that there is no relation between the symptom complex and endocrine disorder, and that whenever there is a relation it is probably secondary and not the cause of the epileptic manifestations" (178).

[5] Cobb (30) has stated that, "probably the greatest cause of epilepsy is cerebral (at birth or later), closely followed in importance by infections of the brain and meninges in infancy and youth."

lent monograph have summarized the theories and facts concerning the etiology of epilepsy, the physiological mechanisms involved in convulsions, and some of the therapies utilized.

Until recent years most of the investigations of epilepsy have been undertaken by neurologists or neuroanatomists searching with eternal hope for pathological lesions or malformations in the structure of the nervous system. More recently, however, physiologists and biochemists have joined their neurological colleagues in their search for facts in the hope that a more thorough understanding of the physiology and biochemistry [6] of the convulsions might result in more effective therapy. The use of the electro-encephalogram has in recent years been in vogue in helping to diagnose epileptic conditions. Electrical theories of the nature of epilepsy have varied, some describing the disease as a "cerebral dysrhythmia" (Lennox), others as a "hypersynchronism" (Jasper). All indicate that epilepsy is marked by abnormalities in the "electric" rhythm of the brain cells. Working on the assumption that abnormalities in the rhythm of the brain cells arise from modifications of the body chemistry, other investigators, such as Merritt, Putnam, and Cobb, have experimented with the administration of certain drugs in an effort to regulate or remove these abnormalities.

Unfortunately, the biochemist has not been able to contribute very much to an elucidation of this baffling disorder. Too many uncontrolled factors and errors have crept into his investigations and, as a consequence, the results have often been either distorted or misleading. Conflicting results reported by equally competent investigators of the same problem have not been infrequent. Among the distorting factors might be mentioned faulty psychiatric diagnosis, poor research technique in sampling of cases,[7] unsatisfactory

[6] Timme (182) expresses the hope of the psychiatrist in a chemical solution. "As for the vegetative level, now that biochemistry is beginning to assume such magnificent proportions in our medical uncomfortableness, that is the line upon which—having failed in all others—we are now concentrating our attacks. If the reason for a genuine epileptic seizure—no matter upon what point it is centred—is one of lowered threshold of the synaptic junction or other salient point of defense in the nervous system, then it may be questioned whether such lowered threshold is not determined by a critical change in the biochemistry of the circulating fluids or else of the neural elements."

[7] Talbot (178) in discussing the results of previous investigations states that examination of these studies "reveals that the large mass of data have

experimental procedures, and inadequate statistical treatment [8] of the obtained results. Previous articles have discussed these factors as they influence studies of the psychoneuroses (116), dementia præcox (117), and manic-depressive insanity (115).

Criticism of biochemical studies of epileptics have generally been directed to the following points: (1) As a general rule, chronic institutional cases have been used in the majority of the studies. The effect of institutional routine, in terms of diet and activity, is difficult to evaluate and to control. (2) The difficulty of ascertaining exactly when a seizure is to occur makes it often impossible to obtain blood samples immediately before a seizure or group of seizures. (3) Many seizures occur at night during sleep. The difficulty of obtaining samples of blood in relation to fits occurring at that time is obvious. (4) Investigators often do not record the time of the seizure or its relation to other seizures, that is, elapsed time since the last seizure. As a result these seizures may be interpreted as inter-paroxysmal seizures when they may really be pre-paroxysmal or post-paroxysmal.

Since there is no specific test to establish or corroborate diagnosis, as in the case of many organic diseases, it is probable that cases of tetanic, uremic or hysterical convulsions, to mention a few, may have been included in some studies of epilepsy. The convulsive state, which has been the basis of classification of epileptics, cannot be regarded as the clear-cut result of any one particular physiological disturbance. There are some who "believe that instead of, or indeed in addition to, the motor release in genuine epilepsy there may be a pathological release in other domains and on other levels than the purely motor cortical" (182).

The biochemist in such instances is not at fault because he relies fully on the adequacy of the psychiatric diagnosis. It is to be hoped that with a fuller knowledge of etiology will come a greater certainty of diagnosis. Study of a few isolated cases without any

not been collected according to any uniform plan, and that selection for study of a disproportionate number of epileptics from institutions renders generalized conclusions invalid. Such patients form a special class which in no way represents the entire epileptic population of a community."

[8] Lennox and Cobb (100) stress this point particularly as it applies to epilepsy. "In any disease of unknown etiology, much can be learned from carefully gathered statistics. But with epilepsy, which is only a symptom, confusion is to be expected unless data are thoroughly digested."

matched normal controls has too often been the point from which to jump off into uncharted seas of speculation.[9]

The literature on the subject is quite confused. This paper will concern itself with some biochemical studies of blood that have been made of epilepsy.

The biochemical constituents that have been investigated most frequently are blood sugar, acidity and alkaline reserve, blood gases, calcium and phosphorus, nitrogenous substances (proteins, total nitrogen, non-protein nitrogen, ammonia, uric acid, etc.), cholesterol, and chlorides. The list of studies cited below makes no claim to completeness. It is rather a selection of the research in this field based upon the thoroughness of the experimental procedures and observed data in each investigation.

Blood Sugar.—Kilduffe (83, p. 513) states, "The sugar metabolism is controlled by the endocrine secretions acting as inhibitors and accelerators. Normally these are accurately balanced and by their compensatory interaction maintain the blood sugar within normal limits." Normal blood contains from 80 to 120 mg. per 100 cc.

Some investigators have reported normal blood sugar for the epileptic. Table 1 presents a summary of the essentially negative findings. Further analysis of the original data reported by investigators has been necessary in some cases in this and succeeding tables.

Not all investigators, however, have reported normal values in the epileptic. Heidema (66) in 1919 using the Bang micro-method tested 5 epileptics and reported increased blood sugar values (ave. 127 mg. per cent). In 1921 Kernsten (82) using the same method studied 18 epileptics and presented 18 curves showing the concentration of blood sugar at frequent intervals throughout the day. Fourteen of these curves were made on days in which patients had seizures. The investigator stated that low values were found in the half hour or hour preceding seizures, a rise in blood sugar during the seizure, and higher values during the several hours following. Greater variability was found during convulsions. It was his belief that the fluctuating levels of blood sugar present evidence of fluctuations in the activity of the adrenal glands. However,

[9] "Too often circumstantial evidence is given equal weight with direct evidence or sweeping generalizations are made from a few observations" (100).

TABLE 1.

BLOOD SUGAR: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. per 100 cc. blood.)

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Weston (192)	1916	Myers-Bailey	6 epileptics (grand mal)	115.1	...	...	100.8-130.0	...	...	...	.....	Normal.
Kooy (86)	1919	Bang	8 epileptics	91.3	...	...	78 -102	...	...	...	.....	Normal.
Weston (191)	1920	Myers-Fine	10 epileptics	104.1	...	...	90 -121	...	...	...	.....	Normal.
Kooy (85)	1920	Bang	6 epileptics	90.0	...	...	.....	...	...	...	.....	Immediate emotional status of patient influences results.
Wuth (193)	1921	Neubauer	10 epileptics	105.0	...	...	96 -127	...	...	...	.....	Hyperglycemia present in three cases.
Wuth (196)	1922	Neubauer	40 epileptics	91.9	13.9	15.1	60 -119	...	...	...	.....	Normal.
Frisch and Walter (51).	1922	Bang	3 epileptics (39 measurements)	102.9	...	...	92.0-115.1	...	...	...	.....	Blood sugar varied with individual patient in the critical periods.
Lennox (84)	1923	.....	100 epileptics	...	...	...	.....	...	...	...	.....	Normal. No relationship between blood sugar values and the time or frequency of convulsions.
Weeks, Allen, Renner, and Wishart (188).	1923	.....	43 epileptics	86.7	18.5	21.3	46 -117	...	...	...	.....	Normal.
Pezzali (151)	1923	.....	3 epileptics (4 measurements)									Normal.
			Before fit	94.2	...	...	72.5-119.0	...	...	...	.....	
			During fit	...	...	...	.....	8.3	...	...	74-90	
			After fit	80.7	...	...	76 -89	...	...	...	.....	
Schryver (168)	1923	Lewis-Benedict.	5 epileptics	87.8	...	...	68 -105	...	...	...	.....	Normal.
Mumford and Parkin (134).	1923	Maclean	2 epileptics	110.0	...	...	.....	...	...	...	.....	No evidence of abnormal sugar values.
			1 epileptic	...	...	...	.....	180.0	...	...	.....	

TABLE 1—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Genzel (53)....	1923	Benedict.....	7 epileptics.....	...	...	...	.....	...	...	...	.....	Normal.
Hoeffel and Moriarty (70).	1924	Folin-Wu....	2 epileptics.....	86.0	...	...	75 -97	...	...	...	.....	Normal.
Sherrill (174)...	1924	Benedict.....	5 epileptics.....	107.0	...	...	101 -116	...	...	...	.....	Normal.
Holmstron (71).	1924	Bang-micro..	20 epileptics.....	94.9	9.6	10.1	71 -112	...	...	...	.....	Normal blood sugar values between seizures but increased blood sugar at time of seizure.
Shaw and Moriarty (172).	1924	Folin-Wu....	5 epileptics.....	90.3	...	...	77.3-103.7	...	...	...	.....	Normal.
Daly, Pryde and Walker (34).	1924	Shaffer-Hartman.	4 epileptics.....	92.3	...	...	81.7-120.0	...	...	...	.....	Normal but tend toward lower limits.
Nielsen (138)...	1925	.....	54 epileptics.....	97.0	...	...	.....	...	...	...	.....	Normal.
Sherrill (174)...	1924	Benedict.....	5 epileptics.....	107.0	...	...	101 -116	...	...	...	.....	Normal.
Reid (161).....	1927	Folin-Wu....	36 epileptics.....	85	...	...	69 -113	...	...	...	.....	Normal.
Labin (88).....	1927	.....	20 epileptics.....	...	...	...	.....	...	...	...	.....	Hyperglycemia in six cases, slight hypoglycemia in two cases, normal glycemia in twelve cases.
Lennox, O'Connor and Bellinger (104).	1927	Folin-Wu....	200 epileptics (omitting diabetics) (429 measurements)	94.2	13.1	13.9	63 -145	...	...	...	.....	Normal fasting blood sugar. No relation between blood sugar level and seizures. Increase of blood sugar during convulsions would seem to depend on available body glucose.
			471 controls.....	92.2	16.8	18.2	.....	...	...	...	.....	
			1 epileptic.....									
			During fit.....	...	...	...	.....	78.0	...	...	.....	
			After fit.....	78.5	...	...	.....	...	...	...	.....	
			1 control									
			During simulated convulsion.....	...	...	...	.....	103.0	...	...	.....	
			After convulsion.....	99.5	...	...	.....	...	...	...	.....	

TABLE 1—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Talbot, Metcalf, Moriarty (179).	1927	Folin-Wu	2 epileptics	88.0	...	...	81.5- 94.5	...	...	...	.....	Normal.
Osnato *et al.* (143).	1927	Shaffer-Hartman.	39 epileptics 19 females 20 males	94.1 85.7	11.4 7.7	12.1 10.0	78 -124 69 -102	... ...	... ...	... ...	....	Normal during interparoxysmal period but an increase after seizure. No hypoglycemia.
				89.7	11.3	12.6	69 -124					
Felsen (45)	1930	Folin-Wu	11 epileptics	...	...	...	.....	89.9	22.6	25.1	52.9-118.0	During paroxysm all eleven patients showed lowering of blood sugar. Changes ascribed to physical exertion.
Munch-Petersen and Schou (135).	1931	Hagedorn-Jensen.	74 epileptics (166 measurements)	84.1	7.9	9.4	.....	...	...	...	.....	Normal. Out of 166 determinations 69 per cent were between 80-120 mg. per cent, 31 per cent below 80 mg. per cent.
Jacchia and Fattovich (74).	1932	Hagedorn-Jensen.	5 epileptics During interval During fit	... ...	... ...	... ...	84 -135	... ...	... ...	... ...	 77-132	No significant hypoglycemia nor any characteristic sugar changes in the seizures. Improvement accompanied by higher blood sugar values.
Ström-Olsen (177).	1932	Folin-Wu	2 epileptics	97	...	...	90 -104	...	...	...	.....	Normal.
Minchin(131)	1933	Calvert	80 epileptics	84.9	11.7	13.8	57 -109	...	...	...	.....	Low average results.
Baudouin, Azerad, and Lewin (6).	1933	Baudouin-Lewin.	17 epileptics	83.6	10.4	12.4	62 -100	...	...	...	.....	Normal.

TABLE 1—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Haury and Hirschfelder (65).	1935	Gibson.....	21 epileptics..... During fit.....	92.5 ...	... ...	... ...	90 -110	... 168.0	... ...	... ...	....	In all cases blood glucose was normal before seizures and rose during seizures. Rise was roughly proportional to severity of seizures. No evidence of any hypoglycemia.
Guirdham and Pettit (62).	1936	Folin-Wu....	13 epileptics.....	83.5	8.8	10.5	70 -110	...	...	...	.....	Unusually low normal blood sugar.
Ziskind and Bolton (197).	1936	Benedict.....	36 epileptics.....	89.2	18.3	20.5	59 -167	...	...	...	.....	Spontaneous hypoglycemia probably not a prevalent factor in precipitation of epileptic seizures. Low normal blood sugar in epilepsy.
Pollack and Boshes (155).	1937	Randles and Grigg micro.	87 epileptics.....	79.4	10.7	13.5	52.0-113.0	...	...	...	.....	Normal blood dextrose. No relation existing between fasting sugar level and seizures or between levels and the type of epilepsy, organic or idiopathic.
Metcalfe (126).	1939	.....	6 epileptics.....	93.2	...	...	52.0-154.0	...	...	...	.....	Normal. Clinical manifestations of blood sugar levels did not parallel one another.

Lennox, *et al.* (104) remark that Kersten's use of the Bang micromethod and of such a small amount of capillary blood from the lobe of the ear increases the error of measurement.

Barlocco (5) in 1922 also using this method studied a group of epileptics and found blood glucose values above normal before a seizure but normal during the attack. Vollmer (187) with the same method examined a group of patients and reported an increasing hypoglycemia up to the time of the convulsion, this reversing with the seizure and becoming a hyperglycemia. In 1924 Dide, Fages, and Baudouin (39) made a study of several epileptics and found hypoglycemia to be the rule, that is the blood sugar value was never above but frequently below normal.

Wladyczko (195) in 1925 tested 18 epileptics and found evidences of hypoglycemia which the investigator believed could directly or indirectly provoke a seizure. In 1926 Lennox, O'Connor, and Bellinger (103) made frequently repeated measurements in an epileptic patient having serial convulsions and on a healthy person who simulated convulsions. They found increased blood sugar during convulsions but concluded that any increase in blood sugar which takes place during convulsions is the result of the muscular exertion and asphyxia of the seizure. They felt that whether or not blood sugar increases depends on the amount of readily available glycogen in the body. It was their belief that in the usual case of epilepsy there is no evidence that blood sugar plays any more than a passive rôle in the events associated with seizures while in the unusual patient extreme hypoglycemia may assist in the precipitation of seizures.

Nielsen (137) in 1934 using the Folin-Wu method studied 56 idiopathic epileptics and found an average value of 82.8 mg. per cent which was about 10 per cent below that of the average of 102 controls. There was reported a tendency to hypoglycemia in about 90 per cent of the cases. Tyson, Otis, and Joyce (186) in 1935 using the Folin-Wu method tested 92 epileptics and 16 non-epileptic controls. Of these 92 patients 85 were on luminal therapy which may cause a rise in the fasting blood sugar. Despite this fact, 56.4 per cent of the patients showed subnormal fasting blood sugar levels as compared with only 12.5 per cent of the non-epileptic controls. Neri (136) in 1935 using the Crecelius-Seiffert method studied 9 patients and reported a tendency to high blood sugar

values. Schou (167) in 1937 using the Hagedorn-Normann-Jensen method tested 100 epileptics and found that 30 had fasting blood-sugar of less than 60 mg. per cent.

Some investigators have attempted to determine what relation, if any, exists between epilepsy and sugar tolerance. Table 2 gives the negative findings in this regard.

Not all workers in the field have reported normal sugar tolerance in the epileptic. Kooy (86) in 1919 using the Bang method tested eight epileptics and reported an abnormal sugar tolerance although the initial and final values were below normal. The investigator felt that the emotional factors present in epilepsy account for the hyperglycemia. In 1924 Tsuchiya (185) using the same method made a study of four patients and found that while they showed the same type of blood sugar curve as normals, at the end of one and five hours the curve was higher than in normal persons. Craig (32) in 1927 employing the Calvert method studied 15 epileptics and reported that while there was no characteristic blood sugar curve for epilepsy there was present an apparent defect in carbohydrate metabolism since only five patients could be considered to give the normal response to the glucose. The investigator believed that irregularities might be due to lack of balance in the endocrine system since improvement was secured by thyroid and adrenaline treatment.

MacKay and Barbash (110) in 1931 tested the glucose tolerance of 66 epileptics and in 68 per cent of these patients found values below normal, apparently due to a low renal threshold. The occurrence of a fit caused a temporary fall in blood sugar level. No relation was noted between type of epilepsy (such as irritable, confused, or demented) or frequency of fits and blood sugar level. While they could find no typical glucose tolerance curve in epilepsy, they reported a high percentage of epileptic patients with a curve of a definitely subnormal type.

Nielsen (137) in 1934 using the Folin-Wu method made glucose tolerance tests of 50 epileptics and a group of 102 controls and reported that in the epileptics the drop in blood sugar at the end of the second hour was to a lower level than that in the controls. Tyson *et al.* (186) employing the same method tested seven epileptics and reported that there was a correlation between abnormal glucose tolerance and the frequency of the seizures. In a study of nine epileptics in 1935 Telatin (181) obtained glycemic curves denoting

TABLE 2.

SUGAR TOLERANCE: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. per 100 cc. blood.)

Investigator.	Date.	Method.	Subjects.	Change.			Comments or conclusions.
				Mean.	S. D.	C. V.	
Schwab (170)	1922	Myers-Bailey	10 epileptics	...	...	...	Normal in almost all forms of epilepsy except where there are marked mental symptoms. The curves are of no diagnostic value.
Holmstrom (71)	1924	Bang micro-	26 epileptics	+57.1	21.2	37.1	Normal between seizures.
Drury and Farran-Ridge (42).	1925	Calvert	15 epileptics	...	...	...	Sugar metabolism in quiet stages approximates most closely to the normal. Only one case showed abnormality.
Lennox and Bellinger (99).	1927	Folin-Wu	140 epileptics	+ 8.1	8.8	108.6	No evidence of abnormality of carbohydrate metabolism that might in itself induce seizures. A seizure had no apparent effect on the shape of the curve. Seventy per cent of initial curves were normal, 24 per cent were high, and 6 per cent were low. A large percentage showed marked variation in the form and level of successive sugar curves, indicating signs of marked variability in tolerance responses.
			8 flat curves.	+50.7	20.9	41.2	
			98 normal curves.	+99.5	41.2	41.6	
			34 high curves	+60.1	36.3	60.4	
			23 controls	+44.7	29.1	65.1	
Munch-Petersen and Schou (135).	1931	Hagedorn-Jensen.	74 epileptics	...	...	...	Normal blood sugar curves.
Strôm-Olsen (177)	1932	Folin-Wu	2 epileptics	+65.0	...	...	Glucose tolerance not related to epileptic condition but to state of nutrition.
Jacobi and Koritter (77).	1932	Crecelius-Seiffert.	3 epileptics	...	...	...	No peculiarities in blood sugar metabolism.
Minchin (131)	1933	Calvert	66 epileptics	+39.3	25.8	65.6	Tendency to flat curves.
Neri (136)	1935	Crecelius-Seiffert.	9 epileptics	+67.8	13.0	19.2	Normal.
Pollock and Boshes (155).	1937	Randles-Grigg.	87 epileptics	+66.0	35.2	53.3	No evidence of an increased or decreased tolerance. Curves were essentially normal. Twenty-six per cent were high, 21 per cent were low, and 53 per cent were normal. Curves could not be related to incidence of attacks or to type of epilepsy, organic or idiopathic.
			54 males	+63.9	41.0	64.2	
			33 females	+65.2	37.5	56.9	

accelerated metabolism of the carbohydrates and therefore curves characteristic of subjects with a primary para-sympathetic neuro-vegetative system. Schou (167) in the study reported above found evidence of an alimentary hypoglycemia in 34 of 100 epileptics, that is, their blood sugar rise during a tolerance test rose to below 120 mg. per cent. Another 43 had a fall to below 50 mg. per cent after the initial rise. By examining seven patients from 7 in the morning to 10 at night, each hour, it was found that patients with low curves also had low sugar curves throughout the day. The investigator believed that the hypoglycemic patients had a tendency to get their fits when their blood-sugar curves were low, although he admits that this connection is not absolute. In order to test whether or not alimentary hypoglycemia was a constant phenomenon, he re-examined 23 patients by means of two or three tests at intervals up to one year and found that 15 of these patients who had a low curve on the first examination again had a low curve on one or more after-examinations. This fact suggested to the investigator that in most cases the hypoglycemia was a permanent phenomenon.

Acidity and Alkaline Reserve.—Kilduffe (83, p. 59) in speaking of the pH of the blood states, "The reaction of the blood is maintained, in spite of the ingestion of acids and basic salts and the metabolic production, neutralization, and excretion of acids and bases, by a very sensitive mechanism of adjustment. The three processes mainly concerned are the excretion of acids and non-volatile bases by the kidney, the base forming properties of hemoglobin, and the excretion of carbon dioxide through the lungs, the two latter being more rapid and important. The buffer system of the blood also functions, but is not of as great importance nor capable of as rapid adjustments as those just mentioned." Normal blood plasma has a pH from 7.3 to 7.5. The CO_2 combining capacity of normal blood plasma is between 50 and 75 volumes per cent and from 23.0 to 32.0 millimoles per liter. The normal range for total base is from 149.0 to 163.0 milliequivalents per liter serum.

Many investigators have attempted to determine whether there is any relation between the acid-base equilibrium of the blood and epilepsy. Some investigators have reported normal results. Table 3 gives the essentially negative findings.

Many other investigators, however, have reported abnormal acid-base values for the epileptic. Charon and Briche (26) in 1897

TABLE 3.

ACIDITY AND ALKALINE RESERVE: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

[Results in plasma volumes per cent (alkaline reserve), except where otherwise indicated.]

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Lin (108).....	1898	.....	13 epileptics..	...	...	...	0.292-0.348	...	...	...	.....	Normal but nearly always within lower limits.
			During fit..	...	...	...	.....	...	...	...	0.279-0.320	
Schultz (169)...	1907	Friedenthal modifications.	11 epileptics..	1.0×10^{-7}	...	...	(1.0×10^{-7})-(1.1×10^{-7})	...	...	...	.....	No deviation of blood acidity from normal as determined by hydroxylon concentration.
								...	...	...	.....	
Mørdre(133)...	1922	Van Slyke...	5 epileptics...	65.1	...	...	60.1-69.0	...	...	...	.....	Alkaline reserve a little higher than average, normal value, but there is no distinctive alkalosis. Some existence of past paroxysmal acidosis and acidosis which may be result of increased output of lactic acid during the paroxysm.
			2 epileptics During attacks..	...	...	...	.....	46.8	...	...	.....	
			12 hours after....	63.7	...	...	.....	...	...	...	.....	
Lennox (90)....	1923	Van Slyke...	100 epileptics.	...	...	...	.....	...	...	...	.....	Normal. No relationship of alkali reserve to time or frequency of convulsions.
Bigwood (11)...	1923	Cullen pH colorimetric.	40 epileptics..	...	...	...	7.4-7.5pH	...	...	...	.....	In an "essential non-convulsive form" of epilepsy find normal pH. In convulsive type, find alkalosis preceding seizures. Following seizures may find alkalosis, normal pH, or even acidosis.
Weeks, Allen, Renner and Wishart(188).	1923	Van Slyke....	49 epileptics..	58.8	4.2	7.1	54.3-64.2	...	...	...	.....	Normal.

TABLE 3—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Geyelin (54)...	1923	.....	.....	...	...	...	.....	...	...	...	.....	No pH curve definitely characteristic of epilepsy. However, find a distinctly wider range of pH from day to day and hour to hour than is normal.
Hoeffel and Moriarty (70).	1924	Van Slyke alkali reserve, Cullen pH.	1 epileptic....	54.7 7.37pH	...	...	.....	...	...	...	.....	Normal.
Geyelin, Bigwood and Wheatley (55).	1924	.....	38 epileptics.. 28 controls.	... ...	... ...	... ...	7.25-7.50pH 7.36-7.43pH	... ...	... ...	... ...	....	Normal pH in epileptics. Range greater in patients than in normals.
Shaw and Moriarty (172).	1924	Van Slyke and Cullen (Henderson-Hassalbach formula).	5 epileptics... (alkali reserve) 3 epileptics...	57.6 7.43pH	... ...	... ...	48.0-66.4 7.40-7.48pH	... ...	... ...	... ...	.....	Normal.
Hamilton (63)..	1925	Fiske total fixed base, Van Slyke bicarbonate.	17 epileptics Total base.. Bicarbonate	157.7 27.6	3.2 1.9	2.0 6.9	153-161 24-31	... ...	... ...	... ...	....	Normal. (Concentrations of bicarbonate and total fixed base in the serum expressed in cc. of 0.1N in 100 cc. serum.)
Marrack and Thacker (112).	1926	Hastings-Sendroy pH.	3 epileptics... (20 measurements) Bicarbonate (20 measurements) 10 controls (12 measurements) Bicarbonate	7.38pH 25.2 7.42pH 27.3	.15 ... .18 ...	2.03 ... 2.43 ...	7.31-7.47pH 22.0-30.5 7.35-7.50pH 2.40-29.5	... ...	... ...	... ...	....	Normal. (The pH and bicarbonate of venous plasma expressed in thousandths of a mole.)

TABLE 3—*Continued.*

TABLE 3—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Frisch and Fried (50).	1926	Hollo-Weiss pH Cullen bicarbonate.	23 epileptics.. 9 epileptics (bicarbonate).	7.55pH 62.7	.19 3.6	2.52 5.7	7.46-7.77pH 57.9-71.2	... ...	... ...	... ...	....	pH values in pre-, past-, and paroxysm-free intervals were normal in majority of cases. Indicated that the actual blood reaction bore no relation to paroxysm. Bicarbonate was normal.
Lennox, O'Connor Bellinger (103).	1926	Van Slyke...	6 epileptics (10 measurements)	67.2	...	...	59.7-75.3	...	...	...	.....	Normal plasma bicarbonate in epileptics.
Talbot, Metcalf, and Moriarty (179).	1927	Van Slyke...	2 epileptics...	58.6	...	...	.....	...	...	...	.....	Normal.
Ballif and Reznic(4).	1927	Van Slyke...	75 epileptics During intervals After attacks	64.0 48.0	... ...	... ...	52-72	... ...	... ...	... ...	....	During intervals alkali reserve normal. Immediately before seizure alkali reserve is still normal. Immediately after clonic-tonic seizure alkali reserve falls, degree of fall depending on intensity of muscular contraction and resulting lactic acid.
Osnato *et al.* (143).	1927	Cullen pH... McQuarrie-Shohl pH. Van Slyke alkali reserve.	19 epileptics.. 9 epileptics (alkali reserve)	7.39pH 62.4	.24 5.7	3.25 9.1	7.33-7.50pH 52.3-73.0	... ...	... ...	... ...	....	Normal.
Shera (173)....	1928	Austin-Cullen pH. Van Slyke alkali reserve.	47 epileptics.. (alkali reserve) 7 controls.... (alkali reserve)	7.35pH 66.0 7.36pH 68.8	... ...	... ...	....	... ...	... ...	... ...	....	No marked deviations in pH or alkali reserve in epilepsy from normal.

TABLE 3—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Lennox and Allen (95).	1928	Van Slyke...	100 epileptics 2 epileptics During intervals. During fits.	64.0 47.9 ...	... ...	... ...	55-73 44.8-50.9	... ... 54.2	... ...	... ...	.... 51.3-57.8	Normal acid-base equilibrium in general. Abnormality when present is in the direction of increased alkalinity. Acidosis during convulsions is the result of convulsions. Daily measurements of several patients did not show striking daily variation.
McQuarrie and Keith (123).	1929	Van Slyke alkali reserve, modified Cullen pH.	.....	...	...	...	.....	...	...	...	.....	During frequent seizures there is a tendency for blood reaction to become alkaline. At other times, it is within the normal range.
Gozzano(59)...	1930	Van Slyke...	14 epileptics (38 measurements) 1 epileptic Before fit... After fit...	... 76.3 71.5	... ...	... ...	30.3-76.3	... ...	... ...	... ...	....	In one-third of the cases, the alkali reserve was higher than normal but in others it was normal. During attack, the blood became more acid.
Torres Lopez (184).	1930	Van Slyke...	17 epileptics..	55.0	10.5	19.1	32.8-71.0	...	...	...	.....	Normal alkali reserve in 59 per cent of cases, slight acidosis in 35 per cent, marked acidosis in 6 per cent. No cases of alkalosis. There was a slight tendency (+.10) for the alkali reserve to be greater as the interval since the last seizure increased.
Massazza (113).	1930	Van Slyke alkali reserve and Michaelis Walpole pH.	"Symptomatic epileptics"	...	...	...	.....	...	...	...	.....	Normal pH and alkaline reserve.

TABLE 3—*Continued.*

TABLE 3—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Felsen (45)....	1930	Levy, Marriott and Rowntree pH.	19 epileptics..	...	...	...	7.6-7.8pH	...	...	...	.....	No significant changes in pH during intervals or during paroxysm. Some variations encountered in non-epileptics. After seizures no changes not accounted for by physical exertion.
			3 epileptics...	...	...	...	.....	7.6pH	...	...	7.5-7.8pH	
Ballif and Gherscovici (3).	1931	.....	30 epileptics..	...	...	...	.....	...	...	...	.....	Normal pH and alkali reserve of plasma in epileptics.
Katzenelbogen (81).	1931	Van Slyke...	31 epileptics..	63.7	...	...	54-74	...	...	...	.....	Alkaline reserve normal.
McLaughlin and Hurst (119).	1933	Van Slyke Alkaline reserve Hurst pH. Friedemann, Cotonio, and Shaffer lactic acid.	*Alkali Reserve* 18 epileptics During interval.	69.1	2.6	3.8	61.5-74.0	...	...	...	.....	During interval, alkali reserve and lactic acid variations were normal but pH variations were slightly above normal. Average lactic acid is normal but average alkali reserve and pH values tend to be slightly above normal. Alkali reserve and pH during 2 hour pre-fit period were not different from interval period. (Lactic acid expressed in mg. per cent.)
			9 epileptics During fit	...	...	...	.....	60.0	5.6	9.3	49.5-68.0	
			12 controls.	64.4	...	...	57-73	...	...	...	.....	
			pH During interval (20 measurements) (on epileptics)	7.35pH	...	...	7.30-7.39pH	...	...	...	.....	
			During fit. (7 measurements)	...	...	...	.....	7.29pH	...	...	7.15-7.37pH	
			After fit (9 measurements)	7.36pH	...	...	7.31-7.40pH	...	...	...	.....	
			6 controls..	7.32pH	...	...	7.30-7.36pH	...	...	...	.....	
			Lactic Acid During interval... (8 measurements)	13.0	...	...	10-18	...	...	...	.....	
			8 controls..	13.5	...	...	10-18	...	...	...	.....	

TABLE 3—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Hopkins (72)...	1935	Hastings-Sendroy pH, Van Slyke alkali reserve.	*Plasma pH* 7 epileptics 5 controls.. *Alkali Reserve* 7 epileptics 5 controls..	 7.49pH 7.40pH 59.9 59.1	 .19 .24 1.58 2.08	 2.54 3.24 2.64 3.55						Plasma pH showed little variation from hour to hour. The epileptic group showed a uniformly higher level of pH than normal controls. Alkali reserve of epileptics did not vary greatly from hour to hour.
Simpson and Barker (175).	1938	Van Slyke Cullen alkali reserve.	11 epileptics..	55.2	4.9	8.9	48.6-61.7	...	...	...	.....	Acid-base balance of the epileptic in the interparoxysmal periods appears to be normal.

using the Landois method tested eight epileptics and found that the degree of alkalinity of the blood underwent constant variations during the course of each day. The convulsive attacks presented variations equally constant, occurring alternately with the variations in alkalinity of the blood. In 1902 Pugh (158) tested 40 epileptics and 20 controls and reported that the interparoxysmal period in the patients is lower in alkalinity in the average than the average for the controls. There was a marked fall in alkalinity immediately prior to the onset of the fit. There was a further fall from three to ten minutes after the fit was over with a gradual return to normal alkalinity in five to six hours. The investigator was of the belief that the diminution of the alkalinity after the fit is due to the chemical end products of muscular metabolism, that is, sarcolactic and carbonic acids, and not to substances in direct relation to the epilepsy. In 1903 Pugh (159) using Wright's method tested 40 epileptics and reported great variability in the alkalinity in the patients.

Jarloev (78) in 1921 found that the pH value of the blood of a group of epileptics rose, on the average, to 7.42 at the approach of an attack which the investigator thought to be due to intoxication with basic substances which might be neutralized during the attack by acid products formed by the contracting muscles. Frisch and Walter (51) in 1922 tested three idiopathic epileptics and noted a fall of total alkalescence with increasing diffusible alkali in the pre-paroxysmal stage. In 1923 Bigwood (11) using the Cullen method studied 40 epileptics and in the "convulsive type" of epilepsy found a "rupture of the regulating mechanism of blood pH" and a state of transitory alkalosis preceding the seizure. Following the attack there might be alkalosis normal blood pH, or even acidosis. The same investigator (9) in 1924 using the same method examined 35 epileptics making two to 11 repetitions on each and found that when the hydrogen ions were normal or higher than normal in concentration the patients had no attacks. During a long period without seizures the pH might remain normal. Usually whenever there was an alkalosis, there was a seizure. There was no relationship between the degree of alkalosis and the severity of seizures. Cases with psychic manifestations, petit mal, etc., without convulsions, did not show such marked fluctuations in pH. Plasma pH was normal in five out of six patients with

Jacksonian epilepsy. The investigator believed that the tendency toward alkalosis was not due to over-ventilation, as shown by normal measurements of blood and alveolar CO_2, or to accumulation of bases but rather to a breakdown of the mechanism which regulates the reaction of the blood.

De Crinis (37) in 1925 tested seven epileptics and reported that the alkaline reserve of the blood is generally decreased before the seizure. From this the investigator concluded that the blood had taken up so much acid and therefore had bound so much alkali that there did not remain as much alkali for taking up CO_2 as was the case with healthy subjects.

In 1926 Dautrebande (35) tested seven epileptics and reported pH values between 7.37 and 7.52 (average 7.46) and plasma bicarbonate values that varied between 55 and 65.7 volumes per cent. The investigator considered the daily fluctuations in pH and alkaline reserve abnormally large and was of the belief that the blood of epileptic patients tended to be abnormally alkaline but could not observe any relationship between alkalinity and the occurrence of seizures. He suggested that the high pH values obtained were due to the presence in the blood of epileptics of an alkaline substance which combines with difficulty with CO_2 *in vivo*.

Ballif and Reznic (4) in 1927 using the Van Slyke method tested 75 epileptics and reported that during intervals the alkali reserve varied within normal limits (52-72 vol. per cent) with an average of 64 vol. per cent. However, immediately after a clonic-tonic seizure the value falls and the degree of the fall depends on the intensity of the muscular contractions and on the lactic acid which results from them. Immediately after seizures the average alkali reserve is 48 vol. per cent, an average drop of 16 vol. per cent. The return of the alkali reserve to normal is generally effected after two hours, being directly influenced by the intensity and duration of the convulsive period.

Osnato *et al.* (143) using the Clausen method in 1927 studied 40 idiopathic epileptics and found an average blood lactic acid value of 31.7 mg. per cent which was uniformly increased, on the average, 74 per cent above normal. The investigators were of the opinion that the increase was not due to the muscular exertions during convulsions but might perhaps indicate abnormal metabolic processes. Page (143) believes that it is possible that the high

lactic acid values obtained by these investigators resulted from anoxemia. McLaughlin and Hurst (119) feel that the figures obtained by Osnato *et al.* (143) are unusually high in a number of cases and are of the opinion that it is very unlikely that they would be obtained with present more refined experimental techniques for determining lactic acid. They also criticize the lack of experimental results for normal blood lactic acid since Osnato *et al.* (143) did not have any control subjects. Bigwood (8) in 1929 after studying a group of epileptics concluded that an alteration in reaction of the blood toward increased alkalinity was an essential preliminary to the production of convulsions by a hypothetical toxin.

In 1930 Di Renzo (40) studied 21 epileptics and reported that the epileptic convulsion is preceded by a rise in the alkaline reserve of the blood manifested 24-48 hours before a seizure. Immediately before a fit the alkaline reserve decreased rapidly to normal and remained at that level throughout the fit and until the next seizure. According to the investigator the convulsive seizure appears to coincide with the sudden throwing into the blood stream of a quantity of acid which brings down the increase in the alkaline reserve. Massazza (113) in the study mentioned above reported that in the "essential" epileptics he found evidence of alkalosis as shown by increased blood pH, normal or increased alkaline reserve, and decreased CO_2 tension. In a study of nine epileptics and five controls Poli (154) reported that the oscillations in the acid-base cannot be said to be absolutely pathogenic (for the seizure itself) and must be interpreted as natural biochemical symptoms. These symptoms precede and accompany the seizure since the experimental reproduction of modifications in the alkali reserve was not enough to determine alone the onset of the epileptic seizure.

McLaughlin and Hurst (119) in 1933 found that during the intervals the variations in the pH of epileptic patients extend to a range slightly above normal. They also found that the average alkali reserve (ave. 68.7 vol. per cent) and pH values tend to be slightly above normal. They reported a fall in alkali reserve during, and for varying periods after, the fit. The degree of the fall in alkali reserve was related to the severity of the convulsion. They accounted for this fall by the accumulation of lactic acid in the blood. Decrease in lactic acid during recovery was accompanied by increase in alkali reserve. The investigators also reported that the

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blood pH falls during the fit. They attributed this fact firstly to apnœa during the tonic stage, and secondly to production of lactic acid during the clonic stage. It was found that recovery to the normal pH was more rapid in epileptics than it was with normal individuals after exercise. During intervals the epileptics had a lactic acid concentration in the blood (ave. 13.0 mg. per cent) which was not different from that of the normal controls (ave. 13.5 mg. per cent).

In 1935 Mennato (124) using the Van Slyke alkali reserve method and the pH electrometric method studied a group of epileptics and reported an instability of the acid-base equilibrium with a tendency toward alkalosis in the periods before and after the attacks as well as during the intervals. During the convulsive attack the investigator noted a constant decrease in the pH toward acid values which he attributed to the respiratory imbalance or to the imbalance of the muscular mechanics. An anomalous functioning of the respiratory centers was believed to be at the bottom of the alkalosis. Caussade *et al.* (25) in 1936 tested nine epileptics for pH and reported that alkalosis was general in the patients in periods of attacks and particularly so in those who had many attacks. The alkalosis also appeared before the attacks. It usually disappeared after the attacks.

Blood Gases, Oxygen and Carbon Dioxide.—"In disease, marked variations may occur as a result of disturbance in the buffer system of the blood or disturbance in the mechanism regulating or concerned with the respiratory exchange of oxygen and carbon dioxide. Interference with the transportation of oxygen by the hemoglobin always leads to asphyxia which . . . . may be . . . . microscopic leading to interference with cellular and hence organic functional disturbances . . . . " (83, p. 45). It has been found that under certain circumstances "acute decrease in blood or oxygen supply to the brain may precipitate, and an acute increase in the oxygen supply may inhibit a patient's seizures" (92). The oxygen content of arterial blood is 17 per cent to 22 per cent by volume, while the oxygen content of venous blood is 11 per cent to 15 per cent by volume. The carbon dioxide content of arterial blood is 40 to 55 volumes per cent, while that of venous blood is 48 to 60 volumes per cent (150, p. 936). The oxygen capacity is 17.8 to 21.8 volumes per cent (83, p. 44), while the oxygen saturation of hemoglobin

for arterial blood is 93 to 98 per cent and for venous blood is 62 to 85 per cent (78, p. 47). The limits of normal of oxygen saturation for blood from an internal jugular vein is 50-70 per cent and for blood from an extremity is 65-85 per cent.

Some investigators have found normal blood gas values in the epileptic. Table 4 shows the essentially negative findings.

Not all of the investigators, however, have reported normal findings. Notkin *et al.* (140) in 1933, using the Van Slyke method, studied seven epileptics and reported that the oxygen capacity was normal in four epileptics but low in three cases; that the oxygen content was normal in two cases but low in five; that the oxygen unsaturation was normal in three cases but high in four cases. Lennox and Gibbs (101) using the Van Slyke method in 1936 made 100 measurements of blood taken from the arms of epileptics and found an average oxygen saturation of 64.5 per cent which they found to be significantly lower than the 72.3 per cent average for healthy persons as reported in the literature. Forty-one of the 100 measurements were below the normal range 65 per cent to 85 per cent. The range of basilic vein oxygen saturation was from 38 per cent to 91 per cent, and of the femoral vein from 43 per cent to 87 per cent. The observed mild degree of anoxemia of the venous blood of the arms of epileptic patients was believed to be the result of a laggard peripheral circulation due not to insufficient cardiac output but to overactive vaso-constriction preventing the quick passage of blood through the capillaries. The investigators felt that the condition was a common accompaniment of poor physique, poor posture, constipation, and an inadequately balanced vegetative nervous system, but not symptomatic of epilepsy alone. Lennox and Gibbs (102) in 1936 using the Van Slyke method studied 88 epileptics and measured the oxygen saturation of the blood of a femoral, radial, or brachial artery 125 times. They found that the average was 93.4 per cent, which was slightly below that of normal individuals (94 per cent to 99 per cent) as taken from the literature. Fifty-four per cent of the cases had oxygen saturation within normal range. However, a study of 58 non-epileptic neurological patients revealed that only 46 per cent of these patients had normal oxygen saturation values. The degree of hypoxemia which the majority of the epileptics exhibited was not in itself sufficient to explain the seizures to which

TABLE 4

BLOOD GASES: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in volumes per cent.)

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Weiss and Lennox (190).	1931	Van Slyke...	8 epileptics									Normal O_2 content, O_2 capacity, and CO_2 content from the cubital or radial artery, the internal jugular vein, the external jugular vein, and the antecubital vein of epileptics.
			O_2 content									
			Artery	21.0	...	...	17.3-23.8	...	...	...	...	
			Int. jug. vein	14.8	...	...	11.9-18.4	...	...	...	...	
			Ext. jug. vein	19.8	...	...	15.2-22.4	...	...	...	...	
			Antecubital vein	13.9	...	...	8.2-18.9	...	...	...	...	
			O_2 capacity									
			Artery	22.1	...	...	18.0-25.2	...	...	...	...	
			Int. jug. vein	20.7	...	...	16.1-23.0	...	...	...	...	
			Ext. jug. vein	21.6	...	...	16.2-25.0	...	...	...	...	
			Antecubital vein	20.4	...	...	16.7-23.4	...	...	...	...	
			CO_2 content									
			Artery	48.7	...	...	46.4-51.3	...	...	...	...	
			Int. jug. vein	56.0	...	...	51.7-58.1	...	...	...	...	
			Ext. jug. vein	50.6	...	...	47.5-53.4	...	...	...	...	
			Antecubital vein	54.7	...	...	50.7-57.2	...	...	...	...	
Lennox (91)....	1931	Van Slyke-Neill.	15 epileptics									Normal O_2 content, O_2 capacity, and CO_2 content from internal jugular vein of epileptics.
			Int. jug. vein									
			O_2 content	11.8	2.6	21.8	4.9-15.4	...	...	...	...	
			O_2 capacity	19.0	3.5	18.4	12.0-24.7	...	...	...	...	
			CO_2 content	54.9	3.8	7.0	48.6-60.2	...	...	...	...	
			4 controls									
			Int. jug. vein									
			O_2 content	11.2	...	...	8.2-12.7	...	...	...	...	
			O_2 capacity	18.1	...	...	15.1-20.0	...	...	...	...	
			CO_2 content	57.5	...	...	54.2-59.8	...	...	...	...	
Notkin *et al.* (140).	1933	Van Slyke...	7 epileptics	...	...	...	.....	...	...	...	...	CO_2 content was normal in five cases and high in two cases.

TABLE 4—*Continued.*

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Lennox and Gibbs (101).	1936	Van Slyke...	110 epileptics									Normal O_2 content, O_2 capacity, and CO_2 content of the blood from an internal jugular vein, from an arm vein, and from a femoral vein of epileptics. The O_2 saturation of blood from an internal jugular vein, while lower than the O_2 saturation from an extremity, was believed to be normal. The average values for the CO_2 content of venous were within normal limits in all groups. During the comatose state of status epilepticus in three instances, the O_2 saturation of blood from an internal jugular vein was found to be rather low, but not below the limits found in consciousness.
			O_2 content									
			Int. jug. vein.....	12.1	...	...	.....	...	...	...	...	
			Antecubital vein...	13.1	...	...	.....	...	...	...	...	
			Femoral vein.....	12.9	...	...	.....	...	...	...	...	
			O_2 capacity									
			Int. jug. vein.....	19.2	...	...	.....	...	...	...	...	
			Antecubital vein...	20.4	...	...	.....	...	...	...	...	
			Femoral vein.....	18.8	...	...	.....	...	...	...	...	
			O_2 saturation									
			Int. jug. vein.....	62.8	...	...	46-83	...	...	...	...	
			Femoral vein.....	68.5	...	...	43-87	...	...	...	...	
			CO_2 content									
			Int. jug. vein.....	54.9	...	...	.....	...	...	...	...	
			Antecubital vein...	53.3	...	...	.....	...	...	...	...	
			Femoral vein.....	52.4	...	...	.....	...	...	...	...	
			79 controls									
			Antecubital vein									
			O_2 content.....	14.9	...	...	.....	...	...	...	...	
			O_2 capacity.....	20.6	...	...	.....	...	...	...	...	
			O_2 saturation.....	72.3	...	...	65-85	...	...	...	...	
			CO_2 content.....	51.7	...	...	.....	...	...	...	...	
Lennox and Gibbs (102).	1936	Van Slyke...	88 epileptics									Normal CO_2 content of arterial blood of epileptics.
			CO_2 content.....	48.6	...	...	.....	...	...	...	...	
			58 controls									
			CO_2 content.....	48.5	...	...	.....	...	...	...	...	
Simpson and Barker (175).	1938	Van Slyke and Van Slyke-Cullen.	5 epileptics									Normal O_2 content and CO_2 content of arterial blood of epileptics.
			O_2 capacity.....	17.8	...	...	13.5-22.2	...	...	...	...	
			(11 epileptics)									
			CO_2 content.....	55.2	4.9	8.9	48.6-61.7	...	...	...	...	

the patients were subject, although it might be a contributing factor. The degree of anoxemia present was believed to be an expression of defective pulmonary ventilation in patients (as well as neurological patients) accompanied by a poor posture, a poor chest expansion, and a poor general physique. This fact was demonstrated when patients with depressed oxygen saturation increased these values to normal by deep breathing or by inhaling oxygen.

Calcium and Phosphorus.—Kilduffe (83, p. 551) remarks that "Calcium is concerned with maintenance of physiological equilibrium and the normal irritability of the nervous system." It plays an important part in the excitability of the vegetative nervous system and the motor system. In tetany there is a deficiency in the calcium concentration of the blood. The calcium content of normal blood serum is 9-11 mg. per 100 cc. Kilduffe (83, p. 570) in discussing the function of phosphorus in the blood states that, "Phosphates act as buffers but their relation to normal and pathological states is not yet determined." The normal inorganic phosphorus content of the blood plasma is from three to five mg. per 100 cc.

Some investigators have reported normal calcium and phosphorus values in the epileptic. Table 5 shows these findings.

Other investigators, however, have failed to confirm normal calcium and inorganic phosphorus results in the epileptic. Prior (157) in 1916 studied 35 epileptics and 12 normal controls and found that the blood calcium of the patients averaged 0.6 that of the normal, indicating thus markedly decreased values. In 1922 Barlocco (5) in the study mentioned above reported distinctly low values in periods preceding the attack. Frisch and Weinberger (52) in 1922 using the Clark method tested three epileptics and found that the blood calcium was increased pre-paroxysmally and also during the height of the attacks and that the tissue calcium decreased by diffusion into the blood stream. The investigators felt that the relative calcium decreased in the tissues seemed to be an important factor in the increase of irritability of the central nervous system.

In 1923 Pezzali (151) studied four epileptics before, during, and after seizures. The calcium content was much higher during attacks. Bigwood (7) in 1924 made a study of 35 epileptics and found the existence of a hyperalkalosis together with a lowered

TABLE 5.

CALCIUM AND INORGANIC PHOSPHORUS: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

[Results in mg. per 100 cc. serum (calcium) and in mg. per 100 cc. plasma (inorganic phosphorus) unless otherwise indicated.]

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Weston (191)...	1920	Lyman.....	10 epileptics.....	7.70	...	...	6.9-8.8	...	...	...	...	Normal serum calcium.
Salvesen (166).	1923	Kramer-Howland	6 epileptics.....	9.88	...	...	9.32-10.56	...	...	...	...	Normal serum calcium.
Koechig (84)...	1924	Lyman.....	1 epileptic.....	10.3	...	...	.....	...	...	...	.....	Normal serum calcium.
Hamilton (63)..	1925	Kramer calcium, Briggs inorganic phosphorus	11 epileptics calcium... 15 epileptics inorg. phos.	5.3 2.3	0.9 0.4	17.0 17.4	4.8 - 6.4 1.5 - 3.5	... ...	... ...	... ...	....	Normal serum calcium and plasma inorganic phosphorus values (expressed as cc. of 0.1 N in 100 cc.).
Reiter (162)....	1925	Kramer-Tisdall.	12 epileptics.....	...	...	...	.....	...	...	...	.....	Serum calcium values have no relation to the convulsions or to other clinical phenomena. Values within normal but tend to level slightly lower than normal. Considerable variability in each individual.
Lennox, O'Connor Bellinger (103).	1926	Kramer-Tisdall calcium.... Bell-Dorsy inorganic phosphorus.	1 epileptic (2 measurements) calcium..... 2 epileptics (3 measurements) inorg. phos.....	11.0 3.31	... ...	... ...	....	... ...	... ...	... ...	....	Normal plasma calcium and plasma inorganic phosphorus.
Armstrong and Hood (1).	1927	Clark-Collip..	7 epileptics.....	9.6	...	...	8.7 -10.8	...	...	...	.....	Normal serum calcium.
Osnato *et al.* (143).	1927	Clark-Collip calcium, Benedict-Theis inorg. phos.	19 epileptics, calcium... 11 epileptics inorg. phos.	10.9 3.6	1.2 0.6	11.0 16.7	9.5 -12.8 2.5 - 4.5	... ...	... ...	... ...	....	Normal serum calcium and plasma inorganic phosphorus.

TABLE 5—*Continued.*

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Longo (106)....	1928	Kramer-Tisdall.	20 epileptics.....	9.68	...	...	8.10-12.00	...	...	...	.....	Normal serum calcium.
			20 controls.....	11.13	...	...	9.50-12.70	...	...	...	.....	
Patterson (149).	1929	de Waard....	100 epileptics.....	...	...	...	9.65-16.97	...	...	...	.....	Normal serum calcium. About 20 percent were outside normal limits. Wide limits of individual variation. No relation of calcium level to type of attack, nature of the epilepsy, time of examination, or diet.
Canterow (23).	1929	Clark-Collip..	4 epileptics, (total serum calcium).	...	...	...	9-11.2	...	...	...	.....	Normal in total serum calcium [three within normal limits (9-11 mg.), one had value of 11.2 mg.]. Normal non-diffusible serum calcium.
			4 epileptics, non-diffusible calcium).	...	...	...	4.15-6.00	...	...	...	.....	
Mondio (132)..	1929	Briggs.....	73 epileptics									Inorganic phosphorus of epileptic blood is normal. Epileptics with few fits had less inorganic phosphorus than those with frequent fits.
			49 males.....	3.1	.65	21.0	1.8-4.4	...	...	...	.....	
			24 females.....	3.4	.40	11.8	2.7-4.5	...	...	...	.....	
				3.2	.50	15.6	1.8-4.5	...	...	...	.....	
			31 controls									
			22 males.....	2.9	.47	16.2	1.6-3.6	...	...	...	.....	
			9 females.....	3.6	.30	8.3	2.7-4.3	...	...	...	.....	
				3.1	.37	11.9	1.6-4.3	...	...	...	.....	
			58 epileptics									
			38 frequent attacks...	3.4	.57	16.8	2.3-4.5	...	...	...	.....	
			20 few attacks.....	3.2	.62	19.4	2.5-3.9	...	...	...	.....	
Felsen (45)....	1930	Clark-Collip..	16 epileptics.....	...	...	...	7.0-10.0	...	...	...	.....	Normal serum calcium.
			1 epileptic.....	...	...	...	.....	8.4	...	...	.....	

TABLE 5—*Continued.*

TABLE 5—*Continued.*

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Lennox and Allen (96).	1930	Clark-Collip..	77 epileptics.....	9.79	...	...	7.5-12.25	...	...	...	.....	Epileptic serum calcium values on the average are within normal limits but show a much wider scattering than non-epileptics. 34 per cent of epileptics lie outside normal limits while only 12 per cent of non-epileptics do.
			51 controls.....	10.06	...	...	9.3-11.1	...	...	...	.....	
Massazza (113).	1930	Kramer-Tisdall.	6 epileptics.....	...	...	...	.....	...	...	...	.....	Normal total calcium in all forms of symptomatic epilepsy and two cases of essential epilepsy.
Katzenelbogen (81).	1931	Clark-Collip..	86 epileptics.....	...	...	...	8.1-11.0	...	...	...	.....	Normal cerum calcium. Only eight cases were below normal. No relation between serum calcium values and the mental states of the patients.
			47 epileptics, with mental deficiency	...	...	...	8.1-11.0	...	...	...	.....	
			13 epileptics with imbecility	...	...	...	8.8-10.6	...	...	...	.....	
			18 epileptics with psychosis.	...	...	...	8.5-10.8	...	...	...	.....	
Ballif and Gherscovici (3).	1931	.....	30 epileptics.....	...	...	...	.....	...	...	...	.....	Normal serum calcium in great majority of cases.
Parhon and Werner (148).	1932	.....	13 epileptics.....	10.7	...	...	9.5-11.5	...	...	...	.....	Normal serum calcium.
Madsen(109)...	1932	Kramer-Tisdall.	44 epileptics.....	5.4	.47	8.8	4.4- 6.3	...	...	...	.....	(Expressed in mg. equivalents per 1000 cc.) (Expressed in mg. per cent). Serum calcium normal in most cases, but with a tendency to low values during intervals and to high values in connection with crises. Epileptic attacks preceded by rises in calcium. Much wider range of variability in epileptics, than in controls. Marked individual variability, too.
			33 controls.....	5.5	.63	11.5	5.0- 6.2	...	...	...	.....	
			18 epileptics (121 measurements)	10.4	.73	7.0	9.6-11.8	...	...	...	.....	
			2 epileptics									
			During interval.....	10.6	...	...	.....	...	...	...	.....	
			During fit.....	...	...	...	.....	12.0	...	...	.....	

TABLE 5—*Continued.*

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Griffiths (61)...	1934	Kramer-Tisdall, calcium.	48 epileptics, calcium (433 measurements)									Average serum calcium is normal, although range of values for group and individual patients alike is wide and variable. Average value is greater before a seizure than after; the highest values are obtained during, and low values exist after, a series of attacks. No correlation between range of values and the frequency or type of fit. Serum phosphorus average level also normal, though it showed wide variations.
			During intervals.....	10.3	...	...	.....					
			48 hours before fit...	10.2	...	...	.....	...	...	...	.....	
			48 hours after fit.....	10.1	...	...	.....	...	...	...	.....	
			Between two fits.....	10.7	...	...	.....	...	...	...	.....	
				10.1	1.10	10.8	.....	...	...	...	.....	
			48 epileptics, inorganic phosphorus. (70 measurements)									
			During intervals.....	4.31	...	...	.....					
			48 hours before fits...	4.12	...	...	.....	...	...	...	.....	
			48 hours after fits....	4.36	...	...	.....	...	...	...	.....	
			Between two fits.....	3.73	...	...	.....	...	...	...	.....	
				4.49	1.28	29.5	1.8-11.4	...	...	...	.....	
Hopkins (72)...	1935	Clark-Collip, serum calcium, Greenburg-Gunther diffusible serum calcium, Fiske-Subbarow inorganic phosphorus.	*7 epileptics* (56 measurements)									Normal total serum calcium, diffusible serum calcium, and serum inorganic—phosphorus in epileptics. Separate inorganic phosphorus determinations for the epileptic subjects covered a much wider range of variations than for the normal group, indicating possibly a disturbed phosphorus metabolism. The most pronounced preconvulsive chemical change was in the elevation of inorganic phosphorus level.
			Serum calcium.....	10.57	.30	2.84	.....					
			Diffus. serum calcium	5.46	.19	3.48	.....	...	...	...	.....	
			Inorganic phosphorus.	3.96	.41	10.35	.....	...	...	...	.....	
			5 controls (40 measurements)									
			Serum calcium.....	10.74	.23	2.14	.....	...	...	...	.....	
			Diffus. serum calcium	5.69	.27	4.75	.....	...	...	...	.....	
			Inorganic phosphorus.	3.96	.27	6.82	.....	...	...	...	.....	

TABLE 5—*Continued.*

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Tingey and Hardwick (183).	1935	Clark-Collip.	9 epileptics (33 measurements)									Serum calcium within normal range but with a tendency to lower values. In individual cases, a greater fluctuation of serum calcium within normal limits observed than in normals. Serum calcium normal during fit and after fit. However, tendency for serum calcium to increase during fit and to decrease after fit.
			During intervals	9.7	...	...	8.8-10.9	...	...	...	.....	
			Before fit	9.4	...	...	8.2-10.3	...	...	...	.....	
			During fit	10.6	...	...	9.9-11.2	...	...	...	.....	
			After fit	10.2	...	...	9.2-11.0	...	...	...	.....	
			Between two fits	9.9	...	...	9.0-10.7	...	...	...	.....	
				9.87	...	...	8.2-11.2	...	...	...	.....	
			20 controls									
			2 epileptics									
			Before fit	9.3	...	...	.....	...	...	...	.....	
			After fit	9.9	...	...	.....	...	...	...	.....	
			3 epileptics									
			During fit	...	...	...	.....	10.8	...	...	.....	
			After fit	10.2	...	...	.....	...	...	...	.....	
Michaels and Searle (129).	1935	Clark-Collip.	1 epileptic									Normal calcium content of blood serum and of serum ultrafiltrate.
			Serum calcium	9.9	...	...	.....	...	...	...	.....	
			Ultrafiltrate of serum calcium	4.7	...	...	.....	...	...	...	.....	
Fletcher and Peden (46).	1935	Jenner-Kay plasma-phosphotase.	*Serium Calcium*									Normal serum calcium, serum inorganic phosphorus and plasma phosphotase. No significant differences between blood at quiescent period and during fit except posibly a slight rise in calcium during fit but this rise is not reliable.
			During interval (32 epileptics)	9.94	1.24	12.5	8.4-11.4	...	...	...	.....	
			During fit (32 epileptics)	...	...	...	.....	10.28	3.06	29.8	8.6-11.7	
			Serium Phosphorus									
			During interval (30 epileptics)	2.88	1.97	68.4	1.9- 4.0	...	...	...	.....	
			During fit (30 epileptics)	...	...	...	.....	3.00	2.25	75.0	2.2- 4.5	
			Plasma Phosphotase									
			During interval (30 epileptics)	5.16	5.15	99.8	2.5- 8.5	...	...	...	.....	
			During fit (30 epileptics)	...	...	...	.....	4.56	6.41	140.6	2.3-9.0	

TABLE 5—*Continued.*

Investigator.	Date.	Method.	Subject.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Hirschfelder and Haury (69).	1935	Kramer-Tisdall.	52 epileptics.....	...	...	...	.....	...	...	...	.....	Plasma calcium always within normal limits. In 17 epileptics with no convulsions, plasma calcium was normal.
Scott and Pigott (171).	1936	Clark-Collip..	50 epileptics During interval..... During fit.....	11.4 ...	... ...	... ...	8.0-14.6	... ...	... ...	... ...	..... 8.0-16.6	Normal serum calcium. Type of epilepsy and associated factors, such as the race, sex, age, age of disease, time of day and the presence or absence of convulsions when specimen was taken had no appreciable effect on calcium level; 93 per cent of all determinations were from 9-14 mg. per cent. Hypo-calcemia is uncommon in chronic epileptics; a normal calcium content or hypercalcemia (11-14 mg. per cent) was the usual finding.
Minchin (121).	1937	Pincussen-Schimmelpfing modification of Kramer-Tisdall.	54 epileptics 24 females..... 30 males.....	10.6 10.5 10.5	... ...	... ...	9.6-12.2 8.5-13.3 8.5-13.3	... ...	... ...	... ...	....	Normal serum calcium, although the average figures were slightly higher than those received for healthy subjects.
Simpson and Barker (175).	1938	Fiske-Subbarow inorganic phosphorus.	11 epileptics.....	3.35	.45	13.4	2.40- 4.10	...	...	...	.....	Normal plasma inorganic phosphorus.

concentration of calcium ions in the blood. When the calcium ions were normal or higher than normal in concentration the subjects had no attacks in spite of the alkalosis and their epileptic condition. In the non-convulsive seizures the calcium ion concentration remained normal. Sachs (165) in 1926 examined eight epileptics whom he tested bi-weekly and obtained normal or high blood calcium values in the inter-paroxysmal period (ave. 9.5 mg. per cent), never decreased values. During the attacks the values always increased (ave. 11.1 mg. per cent). In 1927 Kora (87) using the Kleinman modification of the Bloor nephelometric method tested five patients and found that for a few hours after attacks the amount of total phosphorus of serum increases remarkably (ave. increase 3.1 mg. per cent). Blumgarten and Rohdenburg (16) using the Kramer-Tisdall method tested four epileptics and reported low blood calcium values (ave. 6.00 mg. per cent).

In a study of 21 epileptics in 1930 Di Renzo (40) reported that the epileptic seizure appeared to coincide with an increase of serum calcium. Parhon and Ornstein (146) in 1930 using the Kramer method studied 35 epileptics and reported that in the majority of cases the serum calcium was below or at the lower limit of normal values. Massazza (113) studied a group of epileptics and reported low blood calcium. Di Renzo and Tomasino (41) in 1930 in a study of 20 epileptics and 10 normal controls found that the concentration of calcium in the blood is lower in epileptics (ave. 11.9 mg. per cent) than in the controls (ave. 12.7 mg. per cent) (that is, a hypocalcemia prevails between seizures). During the seizure and the first hour after the seizure there is a slight temporary increase in calcium values. The investigators concluded from the results that "in epileptics a disturbance in the fixation of calcium in the tissues takes place which in the blood is manifested by hypocalcemia." They believed that during the seizures there arise conditions which cause that part of the calcium fixed in some tissues to be freed and to enter the circulation. Hepburn and Neibaum (67) in 1931 using the Clark titration method studied three epileptics and reported low calcium values (ave. 9.9 mg. per cent).

Messina (125) in 1935 made a study of 12 epileptics and found that with attacks the calcium values rose and that with increasing periods of latency they tended toward normal. Hirschfelder and Haury (68) examined 67 epileptics and reported that plasma phos-

phate was sometimes increased during the convulsions. Using the Fiske-Subarrow method, Weil and Liebert (189) tested 11 epileptics and found that during the epileptic seizure the inorganic phosphorus content of the serum is increased about 34 per cent. The investigators concluded that in man the tonic-clonic contractions of the musculature during an epileptic attack are the source of this increased inorganic phosphorus. Atkin (2) in 1937 studied 15 epileptics and reported that the serum calcium tends to higher values (ave. 10.98 mg. per cent).

Nitrogenous Substances.—The picture of nitrogen metabolism is an important reflection of anabolic and katabolic activities of the body. The urea nitrogen of normal whole blood is 10-20 mg. per cent, non-protein nitrogen 28-39 mg. per cent, and creatinine 1-2 mg. per cent. In serum the total proteins content is 6.7-8.7 per cent, total albumin 4.95-7.7 per cent, and total globulin 1.0-2.5 per cent.

Some investigators have reported normal values for the epileptic. Table 6 presents the essentially negative results.

Normal findings have not been reported by all investigators. Obregia and Urechia (141) in 1919 tested 37 epileptic patients and found evidence of increased blood urea before the epileptic seizure with subsequent decrease after the seizure. Bouttier and Mestrezat (18) in a study of one epileptic in 1920 found that during the acute phase (time of the seizure) the concentration of urea in the blood did not reach 50 mg. per cent; after the crisis it increased. The investigators believed that the retention of nitrogen products was the effect rather than the cause of epileptic fits. In 1920 Dufour and Semelaigne (43) reported that the blood urea of one epileptic was about twice the normal amount 12 hours before an epileptic seizure took place.

De Crinis (36) in 1920 tested seven epileptics and reported increased serum albumin before attacks and decreased values after attacks. In 1921 Bisgaard and Nørvig (14) using the Henrique-Christiansen method studied 14 epileptics and found the blood ammonia in these patients ranging from .08 to .83 mg. per cent (normal .3-.4 mg. per cent). The highest values were found just prior to the attacks while the lowest values were found after the seizures. The investigators were of the belief that in true epilepsy the neutralization regulation is greatly altered. Felsen (45) was of the opinion that the high ammonia values reported by these investi-

TABLE 6.

NITROGEN SUBSTANCES: SUBSTANCES OF ESSENTIALLY NEGATIVE FINDINGS.

TABLE 6.

NITROGEN SUBSTANCES: SUBSTANCES OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. per 100 cc. blood.)

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Weston (191)...	1920	Folin-Dennis and Myers-Fine.	10 epileptics Total N	3.57	...	...	3.16-3.80	...	...	...	.....	Normal total N, NPN, uric acid, urea N, creatinine, and creatinine in epileptics.
			NPN	34.4	...	...	32-38	...	...	...	.....	
			Uric acid	1.99	...	...	1.76-2.30	...	...	...	.....	
			Urea N	21.2	...	...	18-24	...	...	...	.....	
			Creatinine	1.96	...	...	1.25-2.31	...	...	...	.....	
			Creatinine plus creatin	7.99	...	...	6.20-9.40	...	...	...	.....	
Frisch and Walter (51).	1922	Bang.....	3 epileptics (56 measurements) residual nitrogen	29.3	...	...	24.1-35.5	...	...	...	.....	Normal residual nitrogen during intervals. Increase before a seizure.
Wuth (196)....	1922	Autenrieth creatinine, Thannhauser non-protein nitrogen Folin-Dennis uric acid.	33 epileptics Creatinine	1.49	0.18	12.1	0.93-2.04	...	...	...	.....	Creatinine, residual nitrogen of serum and uric acid within normal limits. Six cases out of 40 showed values of residual N below 20 mg. per cent while 10 cases showed uric acid values above 3.5 mg. per cent. Average globulin-albumin ratio within normal limits. Great variability of serum protein.
			40 epileptics Residual N	23.67	4.0	16.9	14.31-30.88	...	...	...	.....	
			23 epileptics Globulin / Albumin ratio	3.03 35.06	0.79 10.7	26.1 30.5	1.50-5.40 6.13-53.07	...	...	...	.....	
Pezzali (151)...	1923	.....	4 epileptics (5 measurements) Before fit	18.40	...	...	7.25-30.0	...	...	...	.....	Urea N showed no abnormality before, during or after seizure.
			During fit	...	...	...	.....	21.19	...	...	7.35-27.5	
			After fit	20.99	...	...	7.76-45.5	...	...	...	.....	
Lennox (90)....	1923	.....	100 epileptics.....	...	...	...	.....	...	...	...	.....	Normal NPN, urea N, amino-acid N, uric acid, and creatinine in epileptics. Values have no relationship to the time or frequency of attacks.
Hoeffel and Moriarty (90).	1924	Folin-Wu....	2 epileptics (3 measurements) Uric acid	5.14	...	...	.....	...	...	...	.....	Normal uric acid, amino-acid N, creatinine, and NPN.
			1 epileptic Amino-acid N	6.79	...	...	.....	...	...	...	.....	
			Creatinine	1.20	...	...	.....	...	...	...	.....	
			NPN	32.10	...	...	.....	...	...	...	.....	
Hartenberg (64).	1924	.....	.....	...	...	...	.....	...	...	...	.....	Normal blood urea.

TABLE 6—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Lennox, O'Connor and Wright (105).	1924	Folin-Wu NPN, urea N, and creatinine, Folin amino-acid N and uric acid.	123 epileptics (163 measurements) NPN	32.5	...	...	20.0-48.7	...	...	...	.....	The blood of epileptics shows total NPN, urea N, amino-acid N, uric acid and creatinine to be within normal limits. No relation of values to type of epilepsy, that is, whether "essential" epilepsy or Jacksonian epilepsy. No significant variation found in average figures obtained from blood drawn within 24 hours of a convulsion as contrasted with blood drawn more than 24 hours before or after a convulsion. No difference in values between blood drawn within an hour or two of a convulsion and blood drawn from same patient at interval of days from a convulsion. No abnormal values or significant difference found immediately before and immediately after and during convulsion.
			103 epileptics (137 measurements) urea N	14.3	...	...	9.0-30.9	...	...	...	.....	
			30 epileptics (56 measurements) Amino-acid N	6.2	...	...	4.7-8.25	...	...	...	.....	
			116 epileptics (158 measurements) Uric acid	3.9	...	...	2.3-7.1	...	...	...	.....	
			75 epileptics (77 measurements) Creatinine	1.4	...	...	1.2-1.7	...	...	...	.....	
			15 epileptics Within 24 hours before seizure NPN	36.3	...	...	.....	...	...	...	.....	
			Urea N	16.6	...	...	.....	...	...	...	.....	
			Uric acid	3.9	...	...	.....	...	...	...	.....	
			Creatinine	1.4	...	...	.....	...	...	...	.....	
			20 epileptics Within 24 hours after seizure NPN	34.0	...	...	.....	...	...	...	.....	
			Urea N	15.5	...	...	.....	...	...	...	.....	
			Uric acid	4.3	...	...	.....	...	...	...	.....	
			Creatinine	1.4	...	...	.....	...	...	...	.....	
			1 epileptic Many hours before seizure NPN	23.9	...	...	.....	...	...	...	.....	
			Amino-acid N	5.39	...	...	.....	...	...	...	.....	
			Uric acid	5.67	...	...	.....	...	...	...	.....	
			Few hours before NPN	24.0	...	...	.....	...	...	...	.....	
			Amino-acid N	5.25	...	...	.....	...	...	...	.....	
			Uric acid	5.49	...	...	.....	...	...	...	.....	
			Few hours after NPN	24.3	...	...	.....	...	...	...	.....	
			Amino-acid N	5.32	...	...	.....	...	...	...	.....	
			Uric acid	5.55	...	...	.....	...	...	...	.....	
			Many hours after NPN	22.2	...	...	.....	...	...	...	.....	
			Amino-acid N	5.69	...	...	.....	...	...	...	.....	
			Uric acid	5.66	...	...	.....	...	...	...	.....	

TABLE 6—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Lennox, O'Connor and Bellinger (103).	1926	Folin-Wu NPN, and urea N, Folin amino-acid N.	8 epileptics (17 measurements) NPN	35.6	...	...	30.3-41.0	...	...	...	.....	Normal NPN, urea N, and amino-acid N under standard conditions.
			1 epileptic (4 measurements) Urea N	15.2	...	...	.....	...	...	...	.....	
			Amino-acid N	6.6	...	...	.....	...	...	...	.....	
Talbot *et al.* (179).	1927	Folin-Wu uric acid.	2 epileptics	2.50	...	...	2.08-2.92	...	...	...	.....	Normal uric acid.
Felsen (45)	1930	Folin-Wu urea N, NPN, uric acid, and creatinine.	55 epileptics Urea N	...	...	...	14-20	...	...	...	.....	Normal urea N, NPN, and creatinine in epileptics. Blood values immediately after seizures showed no change other than those which might reasonably be ascribed to physical exertion.
			NPN	...	...	...	20-40	...	...	...	.....	
			51 epileptics Creatinine	...	...	...	1-2	...	...	...	.....	
			11 epileptics Urea N	...	...	...	.....	14.5	1.9	13.1	11.5-19.0	
			NPN	...	...	...	.....	27.9	6.9	24.7	12.8-38.0	
			8 epileptics Creatinine	...	...	...	.....	1.21	...	...	0.70-1.87	
			3 epileptics Uric acid	...	...	...	.....	1.88	...	...	1.15-2.50	

TABLE 6—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
McKenzie and McChesney (118).	1935	Greenberg serum protein.	29 epileptics									Values for albumin and globulin are within normal range and show essentially normal averages. No apparent correlation between values for serum protein and the frequency or recency of convulsions. Protein content of blood drawn immediately after a convulsion is normal and the content remains normal over a period of from 24 to 36 hours although almost without exception there is a decrease in the amount of serum globulin after the convulsion.
			Albumin per cent	4.48	...	...	3.88-4.83	...	...	...	.....	
			Globulin per cent	1.90	...	...	1.49-2.56	...	...	...	.....	
			Albumin / Globulin ratio	2.37	...	...	.....	...	...	...	.....	
			10 controls									
			Albumin per cent	4.54	...	...	4.35-4.92	...	...	...	.....	
			Globulin per cent	1.80	...	...	1.54-1.97	...	...	...	.....	
			Albumin / Globulin ratio	2.52	...	...	.....					
			10 epileptics									
			At seizure									
			Albumin	...	...	...	.....	4.49	.26	5.8	4.11-4.90	
			Globulin	...	...	...	.....	2.14	.31	14.5	1.50-2.51	
			Albumin / Globulin ratio	...	...	...	.....	2.10	...	...	.....	
			10-12 hours after seizures									
			Albumin	4.47	.22	4.94	4.15-4.76	...	...	...	.....	
			Globulin	1.89	.22	11.6	1.60-2.24	...	...	...	.....	
			Albumin / Globulin ratio	2.36	...	...	.....	...	...	...	.....	
			5 epileptics									
			24-36 hours after seizures									
			Albumin	4.54	...	...	4.25-5.16	...	...	...	.....	
			Globulin	1.82	...	...	1.52-2.17	...	...	...	.....	
			Albumin / Globulin ratio	2.50	...	...	.....	...	...	...	.....	
Tingey and Hardwick (183).	1935	Micro-Kjeldahl for total protein N.	2 epileptics									Normal total protein nitrogen during and after epileptic fit.
			Total protein N at seizure	...	...	...	.....	7.16	...	...	6.56-7.75	
			Immediately after seizure	6.24	...	...	5.50-6.98	...	...	...	.....	
			1 hour after seizure	6.83	...	...	6.00-7.65	...	...	...	.....	
			1 epileptic									
			Interval	6.36	...	...	.....	...	...	...	.....	

TABLE 6—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S. D.	C. V.	Range.	Mean.	S. D.	C. V.	Range.	
Fletcher and Peden (46).	1935	.....	24 epileptics Total protein nitrogen. During interval.... During fit.....	8.10 ...	2.50 ...	30.9 ...	6.90-9.46	... 8.28	... 2.35	... 28.4	..... 6.74-9.40	Serum protein normal in epilepsy. No significant difference between serum protein values during quiescent period and during fit.
Hopkins (72)...	1935	Greenberg serum protein.	7 epileptics (56 measurements) Albumin..... Globulin..... 5 controls (40 measurements) Albumin..... Globulin.....	4.34 2.40 4.61 2.40	.17 .17 .07 .16	3.92 7.08 1.52 6.67	....	... ...	... ...	... ...	....	Normal serum albumin and serum globulin in epileptics.
Stone and Chor (176).	1937	.....	18 epileptics Total protein N During interval.... 5 epileptics 24 or more hours before fit..... 24 hours or more after fit.....	7.2 7.1 7.4	.51	7.1	6.0-8.0 6.3-7.5 6.7-7.8	... ...	... ...	... ...	....	Normal serum protein in epileptics. No significant difference between serum protein 24 hours or more before fit and serum protein 24 hours or more after fit.
Simpson and Barker (175).	1938	Howe-Wu-Koch-McMeekin combined methods.	11 epileptics Plasma protein.....	7.77	.89	11.5	6.09-9.00	...	...	...	.....	Plasma protein normal in epileptics.
Piotrowski (153).	1938	Adnot serum protein.	11 epileptics Globulin..... 7 controls Globulin.....	3.14 3.40	... ...	... ...	2.5-3.9 2.4-4.0	... ...	... ...	... ...	....	Normal globulin values in epileptics.

gators was due to the method used. Frigerio (48) in 1921 using the Widal method studied a group of epileptics and found an equal distribution of low and medium values of non-protein nitrogen (ave. 19.8 mg. per cent). In 1922 Wuth (196) studied 40 cases of genuine epilepsy and reported a great number of abnormally high serum protein values as well as a great variability of this constituent in his patients. The serum albumin values were high (ave. 8.3 mg. per cent, range 7.24-19.23 mg. per cent). The investigator was of the opinion that the high values obtained were possibly due to increased blood pressure and the muscular work involved during seizures. There was little daily fluctuations of measurements made of two patients for three weeks. Frisch and Walter (51) in 1922 tested three idiopathic epileptics and reported a pathological value of residual nitrogen five days before a seizure.

In 1922 Meyer and Bruhl (128) using the Kjehldahl method made a study of the serum albumin of 16 epileptics and reported that individual cases showed great variations both in the seizure-free period and also in relation to the attacks. Age, sex, degree of epilepsy, and time of first attacks had no relation to the individual changes in albumin content. There was no relation between the kind of motor effects and the height of the serum albumin content. However, in those cases where there was an albumin level above normal at the time of attack, no motor effects were seen, while those cases where the value was below normal showed marked motor restlessness. Bruhl (21) in 1923 using the same method tested 15 epileptics and found that in six cases where there were motor convulsions, the serum albumin was below normal; three cases with motor symptoms showed no change in albumin content while the cases without motor symptoms showed an increase in albumin content. In 1923 the same investigator (20) employing the Neubauer method studied nine epileptics and reported that five of these patients had increased creatinine at time of seizure. These cases also showed motor effects. There were inconstant increases in creatinine, up to 2.16 mg., after seizures. Three cases that did not show creatinine increase also lacked motor excitement in the attacks. The investigator believed that the increased creatinine values found in convulsive attacks of various geneses were due to the convulsive mechanism and not to the nature of the attack process.

Bisgaard and Nørvig (13) in 1923 tested 16 epileptics and found in the blood about three hours before the epileptic seizure a rising ammonia value which in some cases reached as high as three times that of the normal. The investigators felt that in genuine epilepsy there are constant significant irregularities in ammonia interchange as determined by the Hasselbach method. Shaw and Moriarty (172) studied a group of five epileptic children and found an increased blood uric acid after a fast of several days. Nørvig (139) in 1924 making use of the Folin method examined 22 epileptics and 10 controls and reported that in patients with genuine epilepsy pathological amounts of ammonia are found in the blood 1-6 hours before the convulsion. In the attack-free period low ammonia values are found. Sherrill (174) in 1924 using the Van Slyke modification of the Marshall urease method tested five epileptics and found high blood urea values (ave. 36.4 mg. per cent).

Luck, Thacker, and Marrack (107) in 1925 using the Luck-Seth method tested 10 epileptics and 15 controls and found blood ammonia values in the patients (ave. .18 mg. per cent) that were higher than those found in the controls (ave. .09 mg. per cent). They concluded, however, that high blood ammonia does not occur invariably before epileptic fits and is not the cause of the fits. In 1925 Meyer (127) studied 15 epileptics and reported an increase in the serum albumin as compared with the serum globulin. In 1926 Wuth (194) using the Zeiss dipping refractometer, Pregl's micro-method and the Folin method examined 16 cases of genuine epilepsy and found that non-protein nitrogen, uric acid, creatinine and serum protein showed higher values during or shortly after convulsions than during interparoxysmal periods. The changes were not always uniform in the same or in different individuals nor were they specific for any disease or for the convulsive reaction, as they might be found as the result of all physical exertion. The investigator attributed these increases to the muscular work involved in the seizures. Reid (161) in a study of 36 epileptics reported tendencies toward low non-protein nitrogen values (ave. .29 mg. per cent) and low urea nitrogen values (ave. .12 mg. per cent) in the blood of his patients.

Frisch and Fried (49) using the von Starling method examined a group of epileptics and reported pre-paroxysmally an averaged increased serum albumin. Post-paroxysmally there was an in-

creased serum globulin. In 1928 Lennox and Allen (98) using the Foster and Whipple method studied 100 epileptics and found that in 34 per cent of the patients the average fibrin value, 398 mg. per cent, was considerably above normal (ave. .306 mg. per cent). High values did not correlate with frequency or severity of seizures, duration of illness or time relative to seizures. The investigators could not state whether the increase was an evidence of an irritative process in the liver or whether it was due to an increased katabolism of body tissue.

McKenzie and McChesney (118) using the Greenberg method in 1935 studied 29 epileptics and reported decreases in the amount of serum globulin and serum albumin after the convulsion. Contini (31) using the Howe method examined 20 epileptics and found that with almost absolute constancy the epileptic attack was followed by increases in the plasma of total proteins, albumin, and albumin-globulin ratio. There was found a slight decrease in the total globulins as a result of the attack. The investigator believed that the changes in the protein picture might be caused by the action of particular toxic substances present in the circulation of epileptics following the attack. Piotrowski (153) using the Adnot method studied 11 epileptics and seven controls and reported a slightly higher total albumin content in the patients (ave. 7.96 mg. per cent) than in the controls (ave. 7.57 mg. per cent).

Lipoidal Substances.—In view of the fact that approximately 50 per cent of the brain is made up of lipoid constituents and in view of the belief that cholesterol is one of the controlling factors of cellular metabolism many studies have been made to determine whether patients showing epileptic manifestations also show changes in cholesterol metabolism. Normal blood contains from 140 to 200 mg. of cholesterol, from 250 to 450 mg. of lecithin, and from 290 to 410 mg. fatty acids per 100 cc.

Several investigators have reported normal values in the blood of the epileptic. Table 7 presents the negative findings.

Many other investigators have reported abnormal values for the epileptic. Bornstein (17) in 1911 made a study of 11 epileptics and found an increased lecithin content (ave. 349.6 mg. per cent) in the blood serum of many of the patients. The investigator believed that this was most probably due to the degradation of brain lipoids. In 1920 De Crinis (36) using the Autenrieth-Funk method tested

TABLE 7.

LIPOIDAL SUBSTANCES: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. per 100 cc. blood.)

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S.D.	C.V.	Range.	Mean.	S.D.	C.V.	Range.	
Pighini (152).....	1911	Ludwig and von Liffschutz methodsfor cholesterol and oxycholesterol.	19 epileptics.....	197	125	63.5	86-699	...	...	...	...	Serum cholesterol and oxycholesterol in most cases of epilepsy was normal.
Parhon, Urechia and Popea (147)	1913	Neumann-Hermann.	.....	...	...	...	.....	...	...	...	...	Normal lipoid content.
Cruickshank and Tisdall (33).	1916	.....	4 epileptics.....	1.35	...	...	1.2-1.5	...	...	...	...	(Expressed as total cholesterol in grams per liter of serum.)
Osnato *et al.* (143)	1927	Myers-Wardell cholesterol.	22 epileptics 8 females..... 14 males.....	134.4 144.1	21.2 19.1	15.8 13.3	93-171 111-173	... ...	... ...	... ...	... ...	Normal but tendency toward lower limits of normal.
				140.6	10.9	7.8	93-173	...	...	...	...	
Goodall (57).....	1929	Sackett's modification of Bloor cholesterol.	6 epileptics (18 measurements) 3 females..... 3 males.....	169.0 136.3	... ...	... ...	144-208 131-142	... ...	... ...	... ...	... ...	Mean value of blood cholesterol in pre-fit phases is not markedly different from that in interparoxysmal phases. The pre-paroxysmal cholesterol usually tends to be slightly lower than the inter-paroxysmal value.
				152.7	...	...	131-208	...	...	...	...	
			6 epileptics (18 measurements) Before fit 3 females..... 3 males.....	161.3 132.0	... ...	... ...	143-185 124-139	... ...	... ...	... ...	... ...	
				146.7	...	...	124-185	...	...	...	...	

TABLE 7—*Continued.*

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S.D.	C.V.	Range.	Mean.	S.D.	C.V.	Range.	
Rosen, Krasnow and Notkin (164).	1933	Myers-Wardell cholesterol, Krasnow-Rosen lecithin.	47 epileptics cholesterol lecithin 27 controls cholesterol lecithin	177.2 242.2 171 242	28.4 28.2 16.0 22.0	16.0 11.6 9.4 9.1	130-250 172-302	... ...	... ...	... ...	... ...	Averages for cholesterol and lecithin in blood of epileptics do not differ from the normal. However, there was greater variability in epileptics than in normals in cholesterol and lecithin. In epileptics, 55 per cent of the cholesterol values were outside normal limits, in normals, 32 per cent. In epileptics, 42 per cent of the lecithin values were outside of normal limits, in normals, 21 per cent.
McQuarrie *et al.* (121).	1933	Bloor cholesterol.	100 epileptics 32 controls	169 167	43.0 34.5	25.4 20.4	82-313 103-257	... ...	... ...	... ...	... ...	No significant difference between plasma cholesterol values of epileptics and those of "normal" controls. Greater variability in epileptics in cholesterol. No constant relationship was found to exist between the absolute level of cholesterol and the occurrence of seizures.
Simpson and Barker (175).	1938	Bloor-Pelkan-Allen cholesterol.	4 epileptics	172.3	...	...	162-183	...	...	...	...	Normal blood cholesterol in epileptics.

seven epileptics found that blood cholesterol was increased before a seizure and decreased after the seizure. Malerba (111) in 1921 using the Pisani method and the Liebermann-Burchard reaction studied two epileptics and reported that the blood cholesterol content fell below normal during their fits. Barlocco (5) in the study mentioned above found the blood cholesterol content slightly but constantly low during the epileptic attack. The investigator found normal blood fat values in the period preceding the attack but doubtful oscillations at other times.

Targowla, Badonnel, and Berman (180) in 1923 using the Grigant method made a study of three epileptics and reported that the cholesterol values varied in these patients. Pezzali (151) in 1923 examined five epileptics before, during, and after seizures. The cholesterol content regularly decreased during the seizure and rose afterward, as did the fat to a lesser extent. The regular fluctuations in cholesterol suggested to the investigator that this might have a protective action. In 1924 Goebel (56) tested several epileptics and found that the blood cholesterol was always higher than normal. Popea and Vicol (156) using the Grigant method in 1925 studied 16 epileptics during interparoxysmal periods in all cases but one. During the seizures blood cholesterol values were lower than during the attack-free periods. After the attack they tended to return to interparoxysmal values. The investigators believed that the lowering blood cholesterol during attacks was due to the exaggerated use of circulating cholesterol and to its destruction during convulsions. Ornstein (142) in 1925 found a predominance of hypocholesteremia in cases of epilepsy. Claude, Targowla, and Badonnel (29) in 1926 reported that the blood cholesterol in epileptics during the convulsion is generally high. Jacobi in 1927 using the Myers method tested 23 epileptics had found a strong variation of the serum cholesterol content. A definite connection of these observations with the paroxysms could not be determined. The curve of cholesterol content showed significant deviations from normal.

In 1927 Brain (19) using the Sackett modification of the Bloor method studied a group of epileptics and a group of normals and reported that the epileptics had a slightly lower (7 per cent) blood cholesterol than normals. Within 24 hours of a fit, epileptics showed 15 per cent lower blood cholesterol and 20 per cent lower

serum cholesterol than normal. There was also a fall in blood cholesterol just before the actual fit which indicated that the low blood cholesterol is not the result of the attacks. Robinson, Brain, and Kay (163) in 1927 using the same method made a study of 124 epileptics and 71 normal controls and found low blood cholesterols in the patients. The average blood cholesterol of 45 patients was 147 mg. per cent, within 24 hours of a fit of 79 patients more than 24 hours after a fit was 166 mg. per cent while the average blood cholesterol of the 71 normal controls was 173 mg. per cent. In a series of 11 patients blood determinations were made immediately before and immediately after a fit. In 10 of these there was a progressive fall in blood cholesterol preceding the seizure with the attack coming at the lowest point of the curve indicating that the cholesterol fall is not the result of the seizure. Gosden, Fox, and Brain (58) in 1929 using a modification of the Bloor method studied 21 epileptics and five controls and reported that the average plasma cholesterol level in the patients was subnormal, being exceptionally low in status epilepticus and in serial epilepsy. It was more variable at about the time of the fit than during the attack-free period. In 75 per cent of the cases a fall in cholesterol level preceded an attack.

Duncan (44) in 1930 examined 52 epileptics and found increased serum cholesterol values (ave. 212 mg. per cent). Gray and McGee (60) in 1932 employing the Bloor method studied 623 epileptics and 29 normal controls and reported blood cholesterol values for the patients (ave. 165 mg. per cent) which was below that of the normals (ave. 194 mg. per cent). Convulsions were followed in about an hour by a drop in cholesterol and thereafter by a gradual rise extending over perhaps a month if no further attack occurred, but still not reaching the average value for the normal men. McQuarrie *et al.* (122) in 1933 determined the plasma phosphatide, cholesterol, and total fatty acids repeatedly throughout the day in each of eight epileptic children. The most severely epileptic patient showed hypocholesterolemia consistently over a period of many months, except for one short period. The plasma phosphatide, at times low, was more variable than the cholesterol. The patient showed marked increase in phosphatide without a significant change in the level of cholesterol. The investigators concluded that although the absolute values for the individual lipid constituents

differed greatly in the patients, the ratio of phosphatide to cholesterol tended to be higher at or near the time of convulsions than at other times. A comparison by McQuarrie *et al.* (121) of the plasma lipid values of 100 epileptics and 32 controls revealed that, in general, the average phosphatide value of the patients (ave. 125 mg. per cent) is significantly lower than that of the controls (ave. 172 mg. per cent), that the ratio of phosphatide to cholesterol in patients (ave. 0.74) is significantly lower than that of the controls (ave. 1.04), and that the average value for total fatty acids in patients (ave. 471 mg. per cent) is significantly greater than that of the controls (ave. 312 mg. per cent). In lecithin, total fatty acids, and lecithin cholesterol ratio the epileptics had a statistically significant greater coefficient of variability than the controls had.

In 1933 Hopkins (73) in a study of seven epileptics and six controls reported that the average whole blood cholesterol in epileptics was slightly lower than in normals. The range in variation of the cholesterol at any one hour through 24-hour periods was greater in epileptics than in normals. Jacchia and Fattovich (75) using the Autenrieth and Liebermann-Burckhard method in 1934 tested three epileptics during the interval period, during and following the attacks. The cholesterol values during the interval periods proved to be constantly lower than those usually obtained in normal individuals and, in addition, there appeared to be evidence of a relationship between the clinical severity of the epilepsy and the degree of hypocholesterolemia. During the attack there was frequently found a slight increase in the blood cholesterol which, in the period following the attack, descended constantly and in large measure below the values found either during the attack or in closest interval periods. The investigators felt that the variations in cholesteremia stand in some relationship, at least an indirect one, to the alterations responsible for the convulsive attacks. Rampini (160) using the Grigant method tested 20 epileptics and reported a tendency toward hypercholesteremia during the interval periods. Immediately after the convulsive attacks there was a further increase in the blood cholesterol which was maintained for a certain number of hours but with a tendency to regain the value prior to the attack. In a study of seven epileptics McLean (120) employing the Sackett method found that the occurrence of the epileptic fit is

preceded by a considerable fall in the level of the blood cholesterol. However, the investigator pointed out that a low value does not necessarily mean an attack. Claude *et al.* (28) in 1935 using the Grimbert Laudat, and Weill method studied two epileptics and found a slightly increased or normal cholesterol but a greatly diminished fatty acid content. The increases seemed to be noted when there was an increase in emotion, regardless of malady.

Chlorides.—Chlorides are found in all the tissues of the organism. The chloride is the major anion of the blood. The chloride value of normal blood plasma estimated as sodium chloride is from 570 to 620 mg. per 100 cc. and from 99 to 108 milli-equivalents per liter.

Some investigators have reported normal blood chloride values for the epileptic, as shown in Table 8.

Other investigators, however, have reported abnormal values. Frisch and Weinberger (52) in 1922 tested three periodic epileptics, measuring the blood chloride 38 times. They found that the blood chloride level was decreased preparoxysmally and also during the height of the attacks, that is, the chlorides were retained in the tissues and in turn brought about a water retention by the tissues and edema of the brain, thus causing convulsions. Barlocco (5), in the study mentioned above, found, in general, higher than normal blood chloride values. In 1926 Freudenberg (47) made an intensive study of one epileptic and found a definite dependency of the seizures upon the height of the blood chloride. No attacks were noted when chloride values were under 500 mg. per cent. Delaville and Tcherniakofsky (38) studied four epileptics and reported an increased blood chloride above normal. Felsen (45) in 1930 using the Whitehorn method studied 55 epileptics during the interparoxysmal period and reported that 36 of these patients had increased blood chloride values (above 600 mg. per cent). Nine patients examined immediately after a seizure showed a high average blood chloride value (ave. 629.1 mg. per cent) which the investigator ascribed to the physical exertion involved in the seizure. Mazza (114) tested 16 epileptics and found an average blood chloride value (ave. 610 mg. per cent) which was higher than normal.

Variability.—When the variability of the blood constituents is studied over an extended period of time, and when the variability is compared with that of normal controls, some valuable informa-

TABLE 8.

CHLORIDES: SUMMARY OF ESSENTIALLY NEGATIVE FINDINGS.

(Results in mg. per 100 cc. blood, unless otherwise indicated.)

Investigator.	Date.	Method.	Subjects.	Interparoxysm.				Paroxysm.				Comments or conclusions.
				Mean.	S.D.	C.V.	Range.	Mean.	S.D.	C.V.	Range.	
Weston (191)	1920	Potassium thiocyanate titration.	10 epileptics....	495.8	...	...	480-512	.....	...	...	.....	Normal.
Weeks *et al.* (188).	1923	.....	43 epileptics....	567.3	19.9	3.5	536-626	.....	...	...	.....	Plasma chloride is normal in epileptics.
Pezzali (151).	1923	.....	4 epileptics Before attack During attack After attack..	552.3 ..... 480.3	... ...	... ...	349-845 345-559	..... 537.5	... ...	... ...	..... 352-664	Blood chloride values show nothing characteristic.
Sherrill (174)	1924	Whitehorn....	11 epileptics....	602.1	33.0	5.5	552-650	.....	...	...	.....	Plasma chloride was normal.
Hamilton (58).	1925	Fiske-Lin.....	13 epileptics (15 measurements)	100.2	3.2	3.2	95-103	.....	...	...	.....	Normal serum chloride (expressed as cc. 0.1 N in 100 cc.).
Lennox and Allen (97).	1930	Whitehorn....	120 epileptics Whole blood chloride. Plasma..... chloride.	....	... ...	... ...	466-666 310-687	....	... ...	... ...	....	Average whole blood chloride and average plasma chloride was normal in epileptics. 79 per cent of patients were within normal limits, 18 per cent above normal, 3 per cent below normal.
Ballif and Ghersco-vici (3).	1931	.....	30 epileptics....	.....	...	...	.....	.....	...	...	.....	Plasma chloride was normal.
Fletcher and Peden (46).	1935	.....	17 epileptics During interval. During fit....	76.66	18.09 ...	23.6 ...	64.20-92.80	..... 74.75	... 11.08	... 14.8	..... 64.30-80.10	Normal blood chloride (expressed as c.c. 0.1 N%) was normal before and during fits. No significant difference between values before fits and during fits.
Stone and Chor (176)	1937	.....	7 epileptics (14 measurements)	498.0	...	...	470.0-517.0	.....	...	...	.....	Blood chloride normal.

tion about the epileptic comes to light. "Many factors enter into the total variation and, if the data have been collected adequately, it has been possible by the technique of the analysis of variance to break up the total variation into those parts which are contributed by fluctuations *within* the individuals, by differences *between* individuals, by seasonal trends, by technical error, and by other trends" (117). Studies of the epileptic have tended to reveal both an intra-individual and an inter-individual variability that is greater than that shown by normal controls.

Relatively few studies that relate to the intra-individual variability of the epileptic have been made because of the difficulty of obtaining sufficient repetitions of a test on each patient in the same phase in order to determine his personal statistical constants.[10] Charon and Briche (26) in 1897 made a study of eight epileptics and concluded that during the course of each day the degree of alkalinity of the blood undergoes constant variations in an inverse relationship to the epileptic attacks. Kersten (82) in a study of blood sugar of 18 epileptics reported greater variability in individual cases during the convulsions. In 1922 Meyer and Bruhl (128) tested 16 epileptics and found that individual cases showed great variations in serum albumin both in the seizure-free period and also in relation to the attacks.

Geyelin (54) in 1923 examined a group of epileptics and reported a distinctly wider range of pH from day to day and from hour to hour than is normal. Holmstrom (71) tested 20 epileptics during intervals and during seizures and reported that successive measurements varied as much as 21 per cent during seizures, against 5 per cent variation in an inter-paroxysmal period. A study by

[10] In a personal communication Dr. E. M. Jellinek of the Worcester State Hospital states that "the technique of the analysis of variance enables one to obtain estimates of the average intra-individual variation when a few repetitions of the test have been carried out on each of a fair number of individuals. Furthermore, this technique enables us to determine whether the intra-individual variation is significantly different from the inter-individual variation, as well as to determine the significance of the difference between two estimates of intra-individual variation." Dr. Jellinek further states that "if we should find that individuals differ from each other significantly relative to their personal variation, we may make inferences as to the effort which the individual has to put out in order to maintain organic equilibrium in order to adapt his organism to changing conditions of the environment."

Reiter (162) of 18 epileptics revealed that in each individual there were considerable variations which were outside the limit of error of the method followed, such as, in one case, values varied from 8.3 to 11.5 mg. per cent in measurements made on three different occasions, without any relation to seizures.

Dautrebande (35) in 1926 studied seven epileptics and found that the daily variations in alkali reserve and pH were extremely large. These variations showed no connection with the appearance of the attack. Frisch and Fried (50) examined the pH value of nine patients on seven or more occasions and reported that all but one had a value outside the normal range on one or more occasions.

In 1926 Lenno *et al.* (103) studied 24 epileptics during a fast and reported that the non-protein nitrogen and urea nitrogen varied considerably from day to day. Lennox and Bellinger (99) tested 14 epileptics and found that a large percentage showed marked variations in the form and level of successive sugar curves. The investigators suggested that the great degree of variability shown by certain patients might be an expression of variability in the function of the sympathetic system coincident with consequent changes in blood flow or in the activity of endocrine glands.

In a study of 23 epileptics retested on several occasions, Jacobi (76) reported marked variation of the cholesterol content of the serum. Goodall (57) in 1929 examined six epileptics and made three estimates of each individual inter-paroxysmally and reported that the intra-individual variations pre-paroxysmally tend to be greater than those made inter-paroxysmally. A study by Gosden *et al.* (58) of 21 epileptics tested repeatedly revealed that the blood cholesterol is more variable in epileptics about the time of a fit than at other times. Madsen (109) made 121 measurements of the serum calcium of 18 epileptics and reported a marked individual variability. Hopkins (73) studied seven epileptics and six controls and found that the range in variation of the cholesterol at any one hour through 24-hour periods is greater in epileptics than in normals.

In 1934 Griffiths (61) studied the serum calcium values secured by repeated tests of 48 epileptics and found that the range of values for individual patients was wide and variable. Tingey and Hardwick (183) in a study of nine epileptics reported in individual cases a greater fluctuation of serum calcium within the normal

limits than in normals. The investigators felt that this greater variability in calcium for the same epileptic where several estimations had been made, might justly be attributed to disturbances incidental to the fit and which were not normally constant for any particular attack. Thus, some attacks, owing to spasm of the respiratory muscles, are accompanied by a tendency to acidemia. In those fits where very vigorous motor manifestations occur, the increased muscular metabolism will at first cause an increased lactic acid concentration in the blood, and subsequently this will be followed by an alkalosis, owing to the hyperpnea involved in compensation of the disturbed O_2/CO_2 ratio.

In 1935 Tyson *et al.* (186) made repeated determinations of blood sugar on nine epileptics and found a wide variation. None showed a fasting blood sugar that was consistently normal; 11 per cent showed values that were always subnormal, and 67 per cent showed values that were normal only half or less of the times tested. The investigators were of the belief that a single blood sugar determination was of questionable value.

Pollock and Boshes (155) in 1937 correlated the fasting blood sugar levels of 71 epileptics made on two occasions and reported a coefficient of correlation of +0.27. They felt that so low a correlation indicated that it was impossible to predict a second reading from a first, even within a day or so, with the patient under constant conditions. This, according to the investigators, might explain why different workers have reported widely divergent readings for the fasting levels of patients suffering from epilepsy, although all the levels have fallen within normal physiological limits. The fallacy of single readings to determine a hypoglycemic level was pointed out, since low levels were seen just before attacks in some patients as frequently as they were seen in others who had not had a seizure in years. No relation was found between the fasting blood sugar level and the seizures.

The majority of studies of the variability of the epileptic relate to inter-individual variability. Table 9 lists the results of a number of studies and indicates the extent epileptics and normal controls differ from each other in relation to standard deviation, coefficient of variation, and range in the various blood constituents. An analysis of the data in this table reveals the fact that in the majority of the studies presented, the epileptic is more variable in his internal

TABLE 9.

TABLE 9.

INTER-INDIVIDUAL VARIABILITY: SUMMARY OF FINDINGS WITH EPILEPTICS AND CONTROLS.

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V.	Range.	S. D.	C. V.	Range.	
Blood sugar.	Wuth (196)	1922	Neubauer	40 epileptics	13.9	15.1	60 -119	...	...	.....	
Blood sugar.	Weeks *et al.* (188).	1923	.....	43 epileptics	18.5	21.3	46 -117	...	...	.....	
Blood sugar.	Holmstrom (71).	1924	Bang Micro-	20 epileptics	9.6	10.1	71 -112	...	...	.....	
Blood sugar.	Lennox *et al.* (104).	1927	Folin-Wu	200 epileptics (omitting diabetics) (429 measurements)	13.1	13.9	63 -145	...	...	.....	
				471 controls	6.8	18.2	....	...	...	.....	
Blood sugar.	Osnato *et al.* (143).	1927	Shaffer-Hartman.	39 epileptics	11.4	12.1	78 -124	...	...	.....	
				19 females	7.7	10.0	69 -102	...	...	.....	
				20 males	11.3	12.6	69 -124	...	...	.....	
Blood sugar.	Munch, Petersen *et al.* (135).	1931	Hagedorn-Jensen.	74 epileptics (166 measurements)	7.9	9.4	.....	...	...	.....	31 per cent of the measurements were outside of normal limits.
Blood sugar.	Minchin (131).	1933	Calvert	80 epileptics	11.7	13.8	57 -109	...	...	.....	
Blood sugar.	Baudouin *et al.* (6).	1933	Baudouin-Lewin.	17 epileptics	10.4	12.4	62 -100	...	...	.....	
Blood sugar.	Nielsen (137)	1934	Folin-Wu	56 epileptics	13.9	16.8	58-153	...	...	.....	
Blood sugar.	Guirdham and Pettit (62).	1936	Folin-Wu	13 epileptics	8.8	10.5	70 -110	...	...	.....	
Blood sugar.	Ziskind and Bolton (197).	1936	Benedict	36 epileptics	18.3	20.5	59 -167	...	...	.....	
Blood sugar.	Pollack and Boshes. (155).	1937	Randles-Grigg Micro.	87 epileptics	10.7	13.5	52.0-113.0	...	...	.....	

TABLE 9—*Continued.*

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V.	Range.	S. D.	C. V.	Range.	
Alkaline reserve and acidity.	Hamilton (63).	1925	Fiske total fixed base and Van Slyke bicarbonate.	17 epileptics Total base Bicarbonate	 3.2 1.9	 2.0 6.9	 153-161 24- 31				Expressed as cc. of 0.1 N in 100 cc. serum.
Alkaline reserve and acidity.	Marrack and Thacker (112).	1926	Hastings-Sendroy pH.	8 epileptics (20 measurements) pH Bicarbonate 10 controls (12 measurements) pH 8 controls (9 measurements) Bicarbonate	 .15 ... .18 ...	 2.03 ... 2.43 ...	 7.31-7.47 22.0-30.5 7.35-7.50 24.0-29.5				
Alkaline reserve and acidity.	Frisch and Fried (50).	1926	Hollo-Weiss pH bicarbonate.	33 epileptics pH 9 epileptics Bicarbonate	 .19 3.6	 2.52 5.7	 7.46-7.77 57.0-71.2				
Alkaline reserve and acidity.	Osnato *et al.* (143).	1927	Cullen pH McQuarrie Shohl pH and Van Slyke alkali reserve and Clausen lactic acid.	19 epileptics pH 9 epileptics Alkali reserve 40 epileptics Lactic acid	 .24 5.7 13.0	 3.25 9.1 41.0	 7.33-7.50 52.3-73.0 7.4-79.1				
Alkaline reserve and acidity.	Torres Lopez (184).	1930	Van Slyke...	17 epileptics	10.5	19.1	32.8-71.0	...	...	.....	Abnormal values observed in 41 per cent of the cases.
Alkaline reserve and acidity.	Hopkins (72)..	1935	Hastings-Sendroy pH Van Slyke alkali reserve.	*Plasma pH* 7 epileptics 5 controls *Alkali reserve* 7 epileptics 5 controls	 .19 .24 1.58 2.08	 2.54 3.24 2.64 3.55					Plasma pH showed little variation from hour to hour. Alkali reserve of epileptics did not vary greatly from hour to hour.

TABLE 9—*Continued.*

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V.	Range.	S. D.	C. V.	Range.	
Alkaline reserve and acidity.	Simpson and Barker (175).	1938	Van Slyke-Cullen alkali reserve.	11 epileptics	4.9	8.9	48.6-61.7	...	...	.....	
Blood gases.	Lennox (91)...	1931	Van Slyke-Neill.	15 epileptics Int. jug. vein O_2 content O_2 capacity CO_2 content 4 controls Int. jug. vein O_2 content O_2 capacity CO_2 content	 2.6 3.5 3.8	 21.8 18.4 7.0	 4.9-15.4 12.0-24.7 48.6-60.2 8.2-12.7 15.1-20.0 54.2-59.8				
Blood gases.	Simpson and Barker (175).	1938	Van Slyke and Van Slyke-Cullen.	5 epileptics O_2 capacity 11 epileptics CO_2 content	 ... 4.9	 ... 8.9	 13.5-22.2 48.6-61.7				
Calcium and inorganic phosphorus.	Hamilton (63).	1925	Kramer calcium, Briggs inorganic phosphorus.	11 epileptics 15 epileptics	0.9 0.4	17.0 17.4	4.8-6.4 1.5-3.5	... ...	... ...	....	
Calcium and inorganic phosphorus.	Sachs (165)...	1926	.....	16 epileptics During interval During fit	 .74 .82	 5.8 6.0	 8.6-11.8 9.6-13.6				
Calcium and inorganic phosphorus.	Osnato *et al.* (143).	1927	Clark-Collip calcium, Benedict Theis inorganic phosphorus.	19 epileptics 11 epileptics	1.2 0.6	11.0 16.7	9.5-12.8 2.5-4.5	... ...	... ...	....	

TABLE 9—*Continued.*

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V.	Range.	S. D.	C. V.	Range.	
Calcium and inorganic phosphorus.	Mondio (132)..	1929	Briggs.....	73 epileptics.....	.50	15.6	1.8-4.5	...	...	.....	
				31 controls.....	.37	11.9	1.6-4.3	...	...	.....	
				38 epileptics Frequent fits.....	.57	16.8	2.3-4.5	...	...	.....	
				20 epileptics Few fits.....	.62	19.4	2.5-3.9	...	...	.....	
Calcium and inorganic phosphorus.	Di Renzo and Tomasino (41).	1930	.....	20 epileptics (31 measurements) During interval.....	.62	5.2	10.6-13.1	...	...	.....	
				20 epileptics (30 measurements) Within 4 hours after fit.....	1.05	8.3	10.6-14.1	...	...	.....	
				10 controls.....	.67	5.3	12.0-13.8	...	...	.....	
Calcium and inorganic phosphorus.	Madsen (109)..	1932	Kramer-Tisdall.	44 epileptics.....	.47	8.8	4.4-6.3	...	...	.....	(Expressed in mg. equivalents per 1000 cc.) much wider range of variability in epileptics than in controls. Greater individual variability, also in epileptics.
				33 controls.....	.63	11.5	5.0-6.2	...	...	.....	
Calcium and inorganic phosphorus.	Griffiths (61)..	1934	Kramer-Tisdall calcium, inorganic phosphorus.	48 epileptics (433 measurements)	1.10	10.8	.....	...	...	.....	Range of values for group and individual patients alike is wide and variable in serum calcium and serum phosphorus.
				48 epileptics (70 measurements)	1.28	29.5	1.8-11.4	...	...	.....	
Calcium and inorganic phosphorus.	Hopkins (72)..	1935	Clark-Collip serum calcium, Greenberg-Gunther dif. serum calcium, Fiske-Subbarow inorganic phosphorus.	7 epileptics (56 measurements) Serum calcium.....	.30	2.84	.....	...	...	.....	Separate inorganic phosphorus determinations for the epileptics covered a much wider range of variations than for the normal group, indicating possibly a disturbed phosphorus metabolism.
				Dif. serum calcium...	.19	3.48	.....	...	...	.....	
				Ser. inorg. phosph....	.41	10.35	.....	...	...	.....	
				5 controls (40 measurements) Serum calcium.....	.23	2.14	.....	...	...	.....	
				Dif. serum calcium...	.27	4.75	.....	...	...	.....	
				Ser. inorg. phosph....	.27	6.82	.....	...	...	.....	

TABLE 9—*Continued.*

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V.	Range.	S. D.	C. V.	Range.	
Calcium and inorganic phosphorus.	Fletcher and Peden (46).	1935	Jenner-Kay plasma phosphatose.	*Serum calcium* 32 epileptics During interval	1.24	12.5	8.4-11.4	...	...	.....	
				During fit	...	...	.....	3.06	29.8	8.6-11.7	
				Serum phosphate 30 epileptics During interval	1.97	68.4	1.9-4.0	...	...	.....	
				During fit	...	...	.....	2.25	75.0	2.2-4.5	
				Plasma phosphatose 30 epileptics During invertal	5.15	99.8	2.5-8.5	...	...	.....	
				During fit	...	...	.....	6.41	140.6	2.3-9.0	
Calcium and inorganic phosphorus.	Simpson and Barker (175).	1938	Fiske-Subbarow inorganic phosphorus.	11 epileptics	.45	13.4	2.40-4.10	...	...	.....	
Nitrogenous substances.	Wuth (196)	1922	Autenrieth creatinine, Thannhauser ser. NPN, Folin-Denis uric acid.	33 epileptics Creatinine	0.18	12.1	0.93-2.04	...	...	.....	Great variability of serum protein in epileptics.
				40 epileptics NPN	4.0	16.9	14.31-30.88	...	...	.....	
				Uric acid	0.79	26.1	1.50-5.40	...	...	.....	
				Serum albumin	...	...	7.24-10.23	...	...	.....	
				23 epileptics $\frac{\text{Globulin}}{\text{Albumin}}$ ratio	10.7	30.5	6.13-53.07	...	...	.....	
Nitrogenous substances.	Luck *et al.* (107).	1925	Luck-Seth ammonia.	10 epileptics During interval (22 measurements)	.105	74.5	.055-.470	...	...	.....	
				Before fit (10 measurements)	.134	63.8	.060-.450	...	...	.....	
				After fit (7 measurements)	...	...	.095-.355	...	...	.....	
				15 controls	.08	88.9	.02 -.37	...	...	.....	

TABLE 9—*Continued.*

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V.	Range.	S. D.	C. V.	Range.	
Nitrogenous substances.	Felsen (45)....	1930	Folin-Wu urea N, NPN, uric acid, creatinine.	55 epileptics Urea N..... NPN.....	... ...	... ...	14-20 20-40	... ...	... ...	....	
				51 epileptics Creatinine.....	...	...	1-2	...	...	.....	
				11 epileptics (after fit) Urea N..... NPN.....	1.9 6.9	13.1 24.7	11.5-19.0 12.8-38.0	... ...	... ...	....	
				8 epileptics (after fit) Creatinine.....	...	...	0.70-1.87	...	...	.....	
				3 epileptics (after fit) Uric acid.....	...	...	1.15-2.50	...	...	.....	
Nitrogenous substances.	McKenzie and McChesney (118).	1935	Greenberg serum protein.	29 epileptics Albumin per cent.... Globulin per cent....	... ...	... ...	3.88-4.83 1.49-2.56	... ...	... ...	....	
				10 controls Albumin per cent.... Globulin per cent....	... ...	... ...	4.35-4.92 1.54-1.97	... ...	... ...	....	
				10 epileptics During fit Albumin..... Globulin.....	... ...	... ...	....	.26 .31	5.8 14.5	4.11-4.90 1.50-2.51	
				10-12 hours after fit Albumin..... Globulin.....	.22 .22	4.9 11.6	4.15-4.76 1.60-2.24	... ...	... ...	....	
				5 epileptics 24-36 hours after fit Albumin..... Globulin.....	... ...	... ...	4.25-5.16 1.52-2.17	... ...	... ...	....	
Nitrogenous substances.	Fletcher and Peden (46).	1935	Total protein nitrogen.	24 epileptics During interval..... During fit.....	2.50 ...	30.9 ...	6.90-9.46	... 2.35	... 28.4	..... 6.74-9.40	
Nitrogenous substances.	Hopkins (72)..	1935	Greenberg serum protein.	7 epileptics (56 measurements) Albumin..... Globulin.....	.17 .17	3.02 7.08	.....	... ...	... ...	....	
				5 controls (40 measurements) Albumin..... Globulin.....	.07 .16	1.52 6.67	.....	... ...	... ...	....	

TABLE 9—*Continued.*

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V	Range.	S. D.	C. V.	Range.	
Nitrogenous substances.	Stone and Chor (176).	1937	Total protein nitrogen.	18 epileptics During interval.....	.51	7.1	6.0-8.0	...	...	.....	
				5 epileptics 24 or more hours before fit.....	...	...	6.3-7.5	...	...	.....	
				24 or more hours after fit.....	...	...	6.7-7.8	...	...	.....	
Nitrogenous substances.	Simpson and Barker (175).	1938	Howe-Wu Koch-McMeekin plasma protein.	11 epileptics.....	.89	11.5	6.09-9.00	...	...	.....	
Lipoidal substances.	Pighini (152)..	1911	Ludwig-von Lifschutz serum cholesterol and oxy-cholesterol.	19 epileptics Cholesterol.....	125	63.5	80-600	...	...	.....	
Lipoidal substances.	Osnato *et al.* (143).	1927	Myers-Wardell cholesterol.	22 epileptics.....	10.9	7.8	93-173	...	...	.....	
Lipoidal substances.	Jacobi (76)....	1927	Myers-cholesterol.	21 epileptics (44 measurements)	71.2	31.7	103-420	...	...	.....	
				8 epileptics During interval.....	...	...	130-450	...	...	.....	
				After fit.....	...	...	140-320	...	...	.....	
Lipoidal substances.	Gray and McGee (60).	1932	Bloor cholesterol.	149 epileptics (312 measurements)	.22	13.2	120-230	...	...	.....	
				29 controls.....	.22	11.5	160-230	...	...	.....	

TABLE 9—*Continued.*

Blood constituent.	Investigator.	Date.	Method.	Subject.	Interparoxysm.			Paroxysm.			Comments or conclusions.
					S. D.	C. V.	Range.	S. D.	C. V.	Range.	
Lipoidal substances.	Rosen *et al.* (164).	1933	Myers-Wardell cholesterol and Krasnow-Rosen lecithin.	47 epileptics Cholesterol Lecithin 27 controls Cholesterol Lecithin	 28.4 28.2 16.0 22.0	 16.0 11.6 9.4 9.1	 130-250 172-302				Greater variability in epileptics than in normals in cholesterol and in lecithin. In cholesterol 55 per cent of epileptic values abnormal and in lecithin 42 per cent abnormal.
Lipoidal substances.	McQuarrie *et al.* (121).	1933	Bloor.....	100 epileptics Cholesterol Lecithin Total fatty acid Lecithin/Cholesterol ratio 32 controls Cholesterol Lecithin Total fatty acid Lecithin/Cholesterol ratio	 43.0 60.0 214.0 0.30 34.5 43.6 57.2 0.23	 25.4 48.0 45.2 38.6 20.4 25.0 17.9 23.6	 82-313 26-283 228-1203 0.15-1.51 103-257 88-261 213-463 0.74-1.73				The variability of the epileptics in lecithin, total fatty acids and lecithin/cholesterol ratio was significantly greater than that of the controls.
Chlorides.....	Weeks *et al.* (188).	1923	.....	43 epileptics.....	19.9	3.5	536-626	...	...	.....	
Chlorides.....	Sherrill (174)..	1924	Whitehorn...	11 epileptics.....	33.0	5.5	552-650	...	...	.....	
Chlorides.....	Hamilton (63).	1925	Fiske-Lin....	13 epileptics (15 measurements)	3.2	3.2	95-103	...	...	.....	
Chlorides.....	Mazza (114)...	1932	.....	16 epileptics.....	27.6	4.5	565-680	...	...	.....	
Chlorides.....	Fletcher and Peden (46).	1935	.....	17 epileptics During interval During fit	 18.09 ...	 23.6 ...	 64.2-92.8	 ... 11.08	 ... 14.8	 ..... 64.30-80.10	Expressed as cc. 0.1 N per cent.

environment than control subjects. It is also evident that during and immediately before seizures, the epileptic is more variable biochemically than he is at other times.

Summary and Discussion.—A number of comments might be made, by way of summary, of the various studies which have been reviewed above. Very few clear cut, consistent trends have been observed in the biochemical studies of the blood which have been analyzed. One gains the impression that the epileptic has a disturbed physiological make-up. The objective evidence for this, however, is chiefly in the greater amount of variability than that found in the normal individual. The significance of this lack of stability in the internal environment is, in itself, difficult, if not impossible, to evaluate because of the large number of factors which might be involved.

It must be realized that the effects of the paroxysm are similar to those of any violent muscular exertion of fairly short duration. The picture secured from a biochemical study of the convulsion indicates that during, and as a result of, the fit, the blood sugar increases, the pH may or may not decrease slightly (depending upon the severity of the seizure) and the calcium increases. All of these reactions may be a result of the end-products of muscular metabolism. Although these trends appear to be commonly observed, they do not show statistical reliability as a rule because of the typically small number of subjects and the rather high degree of group and individual variability.

The muscular exertion during the seizure, as well as its emotional concomitants, may account for the increased blood sugar. It has been found that the rise of blood sugar during a seizure is roughly proportional to its severity. However, there appears to be no relationship between the blood sugar values and the type of epilepsy or the frequency of seizures since the amount of available glucose in the body at the time of the seizure will decidedly influence the concentration in the blood, seizure or no seizure. There is no evidence that blood sugar plays more than a passive rôle in the events associated with seizures (104). Although there is present in the inter-paroxysmal and pre-paroxysmal stages a slight tendency to decreased blood sugar there is no proof that the precipitating cause of the seizures is a temporary state of hypoglycemia or that the seizures are in any way etiologically similar to the hypoglycemic

convulsions of hyperinsulinism. Furthermore, the majority of the evidence points to a normal carbohydrate metabolism in epilepsy as shown by studies of glucose tolerance curves.

It has been found that a seizure has no apparent effect on the shape of the tolerance curve although there is a marked variability in the pre-paroxysmal response (99). Results which tend to relate epilepsy to a defective tolerance may in many cases be explained by the larger number of patients with marked emotional tension, or with particular mental or physical conditions, such as toxemia, endocrine dysfunction, menstrual disorders, etc. Instability of the sugar regulating mechanism, if it is present, may well be an expression of fluctuations in glandular activity, in the tone of the sympathetic system or in the acid-base balance of the blood. Thus, it is difficult to prove that a single factor is responsible for any change observed in the sugar level in relation to seizures. The factors mentioned above may distort the tolerance curves in one way or another, both in the epileptic or in normal subjects, and hence would vitiate any apparently reliable relationship between epilepsy and carbohydrate metabolism. At any rate, it must be remembered that with the exception of diabetes, the glucose-tolerance curve is never diagnostic *per se*.

In the interpretation of glucose-tolerance tests, the mere peak reached by the blood sugar is not as important as the time during which the higher levels are maintained, that is, the time required for the blood sugar to return to the pre-test level (83). Many studies of blood sugar curves in epileptics are difficult to interpret and of limited value because blood was taken for only two hours after glucose was ingested and no attempt was made to determine just when the blood sugar returned to the normal pre-test levels.

It is well established that the "seizure threshold" of the epileptic and the frequency of seizures can be increased or decreased by alterations in the acid-base equilibrium or in the fluid balance of the body, by changes in the oxygen tension in the blood or by alterations in the concentration of certain of the bio-chemical constituents of the body. "Changes in hydrogen ion concentration of the blood cause changes in other blood electrolytes. These, in turn, may influence the irritability of the brain" (144). Or it may be that "the abnormality in acid-base balance observed in certain cases of epilepsy is probably not the factor of chief importance in the

mechanism of seizures, but is an incidental accompaniment of a more basic disturbance in the physiology of the nerve cell" (123). While it is known that an induced alkalosis, such as that occasioned by ingestion of alkali or by hyperpnea, may precipitate seizures in the epileptic there is no evidence that the acid-base equilibrium of the epileptic differs to any great degree from the normal individual. No relation has been found between alkaline reserve and the time, occurrence, frequency or severity of convulsions. Studies of the hydrogen ion concentration curve have failed to show any definite characteristics of epilepsy or to show that the blood reaction has any relation to the epileptic manifestation. However, it is possible that there are acid-base changes in the tissues, especially in the central nervous system, which do not find expression in the blood (95). It is also very important to distinguish between those changes occurring during the epileptic convulsions that are the result of the seizures and those that are the cause of them.

The variability of the acid-base equilibrium in the epileptic has been observed frequently. There appears to be a greater than normal hour to hour range in pH and a larger daily variation in alkaline reserve.[11] It is difficult to determine the extent and significance of this variability. It may be connected intimately with an altered or defective neutralization regulation (13), or with a hypothetical rupture of the regulating mechanism of blood pH (11) which, in turn, may be predicated upon defective homeostatic regulators (22).

It would appear that the theories formerly in vogue which regarded the occurrence of convulsions as due to an "intoxication" with alkaline substances are no longer held. The evidence shows no such "intoxication." There is a slight, temporary acidosis found during and after the epileptic convulsion. This acidosis, which is the result of and not the cause of the fit, may be connected with the increased muscular metabolism associated with the convulsion. This would tend to neutralize any basic toxicity if it were present. The

[11] Lennox and Cobb (100) discussing this point state that "if we accept 7.30 to 7.50 as the limits of normal values of pH of the blood none of the dependable observations demonstrated an abnormal equilibrium. They do seem to show, however, that in epileptics there is within this normal limit, an unusual degree of fluctuation from day to day with a tendency to approach a more alkaline reaction."

proponents of the "intoxication" theory would have to explain the origin of the increased basicity as well as its continual recurrence since the fit would ordinarily bring about more nearly conditions of neutrality.

Attempts have been made to reconcile the normal pH which has been observed in the majority of epileptics with the theory of alkalosis as the precipitating factor in seizures. This has been explained by the hypothesis that non-convulsive epilepsy is accompanied by a normal pH while the convulsive types of epilepsy have a high pH (alkalosis). However, not all alkaloses in epileptics lead to seizures and not all seizures are preceded by alkaloses. "Because curves signifying acid-base changes in the blood and frequency of seizures did not maintain a constantly parallel course, associated changes in nerve cell, such as the tension of oxygen, the equilibrium of electrolytes, or the permeability of cell membranes may play a more fundamental part" (94).

Seizures may distinguish convulsive from non-convulsive disorders. However, "some epilepsies may have no convulsions at all in the ordinary acceptance of the term" (182). Also "many convulsive seizures that seem superficially to simulate an epilepsy, such as those occurring in uremia, diabetes, tetany, and many of the other toxemias, may possibly not be truly epileptic (182). Hence the presence or absence of seizures is no more of a guarantee of an epileptic or non-epileptic condition than is the presence or absence of alkalosis. It must be realized furthermore that "the motor cortical release which by reason of its dramatic intrusion startles the senses into its acceptance as the 'whole thing' has, by virtue of this self-advertisement, for ages dominated the conception of genuine epilepsy. It is probable for this reason that the comparatively smaller and less insistent gestures have escaped their true valuation" (182). It is quite possible that in the non-convulsive epilepsies abnormal metabolic processes produce biochemical variations from the norm.

There is evidence from various experiments that a close association exists between cerebral activity and the utilization of oxygen in the cortical cells. The importance of this association, however, in explaining morbid mental states and aberrations is by no means clear. Studies of the blood gases have revealed, in general, no evidence of a generalized cerebral anoxemia in epilepsy. Further-

more, induction of a generalized cerebral anoxemia (with consequent local anemia) is not a certain method of precipitating a seizure in an ordinary epileptic (92). The rather common finding of hypoxemia in the venous blood of the extremities of epileptics appears to be indicative of a laggard, peripheral circulation, defective pulmonary ventilation, poor posture, and poor physique. These factors may account for the stagnant physical and mental state accompanying epilepsy. However, they do not appear to be causally connected with a seizure.

Epilepsy is definitely not related to tetany except by the similarity in seizures. Tetany is marked by a disturbed function of the peripheral nerves, whereas, in epilepsy there is present a disturbed function of the central nervous tissues. Although tetanic convulsions are precipitated by decreased calcium values in acute cases of hypoparathyroidism, no relation has been found in the majority of cases between calcium values and convulsions, mental states, or other clinical phenomena in the epileptic. Many investigators have reported an increased blood calcium during and after the seizure. This increase is in all probability due to the muscular exertion or tension involved in the seizure. Several studies in normal subjects have indicated that calcium may increase 10-15 per cent with physical exercise.

Wide limits of individual variation of blood calcium in epileptics tested from time to time as well as a wide scattering of calcium values from group to group have been reported by several investigators. This greater than normal individual and group variability of epileptics is of considerable interest since it is known that calcium influences the excitability of the vegetative nervous system. With a fluctuating serum calcium, one might reasonably expect a fluctuating degree of excitability in the nervous tissues with consequent variations in irritability.[12]

Studies of the protein metabolism in epileptics have been for the most part negative. It has been reported that non-protein

[12] Lennox and Cobb (100) state that "though calcium constitutes only 3% of the total base in blood serum, its possible variation in epilepsy has interested many workers. This is because of the well known relationship between increased irritability of nerves and decreased concentration of blood calcium in tetany and the relation of calcium to the permeability of capillaries." It is now known that an increased tissue permeability may be related to the precipitation of seizures.

nitrogen, urea nitrogen, amino-acid nitrogen, uric acid, creatinine, ammonia, fibrin, and serum protein values bear no relationship either to the degree or duration of epilepsy nor to the occurrence, time, frequency or severity of convulsions. Thus far increased blood fibrinogen appears to be the only direct evidence of abnormal protein metabolism in epileptics. As in other constituents, an abnormally large variability has been noted in some of the nitrogenous substances. The changes vary widely from one patient to another and there is no evidence that the variations are related to the processes underlying the attack or specific to epilepsy.

The retention of nitrogen products has been the effect rather than the cause of the seizure. Increased urea, creatinine, non-protein nitrogen, uric acid, albumin, and so forth, have been reported by several investigators. These increases, however, were not always uniform in the same or in different individuals, nor were they specific for epilepsy or for the convulsive reaction. Here again all of these effects might be due to the effect of physical exertion (194).

There appears to be evidence of a disturbed lipoid metabolism in the epileptic. In general the majority of studies report low cholesterol values with a great degree of variability in the same epileptic at different times or in a group of epileptics at the same time. The cholesterol content appears to be more variable at about the time of the fit than during the attack-free period. The reasons for this are unknown. Some investigators have stated that there is no definite connection between the cholesterol content of the blood and the paroxysms while others believe that such a relationship may exist. Also there is some justification for the belief that the change in cholesterol is related to the emotional tone without relation to paroxysms and regardless of malady.

The fact that there is primarily a disturbance in lipoid metabolism is related to the reported beneficial effects of the ketogenic diet which induces an acidosis and thus acts as a sedative towards seizures. Other predisposing factors, however, such as a disturbed water balance, increased tissue permeability, anoxemia, and so forth, may aid in the tendency to seizures.[13]

[13] Patterson (149) in discussing this point states that ". . . . certain changes in the chemistry or in the physiology of the body . . . . may have influence for either good or ill on the tendency towards seizures. These changes are

The lack of clear-cut biochemical results, with the exception of the great variability,[14] points to the fact that the average collection of epileptics in a given investigation are usually both physiologically and biochemically a fairly heterogeneous lot. Whether or not this is due to faulty psychiatric diagnosis and classification or to the fact that individuals exhibiting the same mental disorder may differ greatly in their physiological abnormalities or to a defective homeostasis is open to question. In the light of the information now available, it seems "that to call a patient 'epileptic' means almost nothing in an explanatory sense—it is merely calling him a name" (182). It has been stated that "the subject with less severe epilepsy is not different from the normal person during the long intervals between attacks" (123).

It may well be that the only constant biochemical picture presented by the functional mental disorders is the inconstancy in the internal environment as discussed above. The degree of variability may be a reflection of the degree of instability of the sympathetic nervous system. It is possible that a biochemical profile of the separate variabilities of the different blood constituents under study may yield a picture that might differentiate the epileptic from the schizophrenic and the schizophrenic from the manic-depressive. "All these factors are liable to great variation, and they can be interpreted correctly only if the nature and sources of this variation are analyzed" (79).

It is obvious that in future studies greater accuracy must be exercised in the diagnosis and classification of patients, more control in the selection and in the adequacy of the number of subjects under

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not classed among the abnormalities because there is as yet no convincing evidence that epileptics are abnormal with respect to these conditions."

[14] Lennox and Cobb (100) feel that in the epileptic, "concerning abnormalities of the body elsewhere than in the brain, the most constant is the inconstancy of physiological processes. For example, there is a variation from day to day in such measurements as pH of the blood, oxygen consumption, blood sugar curves, activity of reflexes, vaso-motor reactions, leucocytic formula, excretion of ammonia, weight, and nitrogen balance." These may all be related by an unstable sympathetic nervous system. Disturbances in circulation and in general metabolism may arise as a result of such abnormalities of vaso-motor control. These abnormalities are intimately related to the precipitation and incidence of epileptic crises although it is difficult to state which is cause and which is effect.

investigation. In addition, greater care should be given to the dietary, physical, and mental factors which might distort the results. Also more care should be given to the periodic and long-section blood sampling over extended periods of time. Finally more precision should be obtained in the statistical treatment and interpretation of the obtained data.[15]

Both the physical and the psychological factors probably enter into the etiology of epilepsy. The relative importance of these two variables is as yet unknown. In some cases, the physical appears to be more important, in others the psychological stands out. It has become obvious that the psychological phases cannot be understood without studying the physiological ones. In the future, physiological studies[16] of the epileptic will continue, no doubt, to help clarify the picture of the psychological abnormalities just as one now follows particular mental and emotional states in attempting to determine their influence upon the organic constants in the internal environment.

BIBLIOGRAPHY.

1. Armstrong, E., and Hood, W.: The calcium content of serum in mental invalids. Jour. Ment. Sci., 73: 111-114, 1927.
2. Atkin, J.: Serum calcium in the psychoses. Lancet, 1: 439-440, 1937.
3. Ballif, L., and Gherscovici, I.: L'équilibre acido-basique dans les maladies nerveuses et mentales. Compt. rend. Soc. Biol., 108: 995-996, 1931.
4. Ballif, L., and Reznic, A.: La reserve alcaline chez les epileptiques. Compt. rend. Soc. Biol., 96: 1179-1180, 1927.
5. Barlocco, A.: Ricerche sul ricambio intermedio dell'N., indacano, NaCl, glucosio, calcio, colesterina e grassi nell' epilessia. Riforma Med., 38: 580-581, 1922.
6. Baudouin, A., Azérad, E., and Lewin, J.: Epreuve d'hypoglycémie insulinique chez les épileptiques. Compt. rend. Soc. Biol., 114: 902-903, 1933.

[15] Dr. E. M. Jellinek (personal communication) feels that the main task of biological workers "is to devise all their investigations in such a manner that it would be possible to distinguish the sources of variation and then apply such quantitative methods which will estimate the contributions of the sources efficiently."

[16] Lennox (92) feels that "our hope of learning the pathogenesis of epilepsy, so far as it concerns physiology and so far as it has the therapeutic approach, rests on finding the physico-chemical process involved in the electrical activity of the neurone."

7. Bigwood, E. J.: La carence du sang en ions calcium chez les épileptiques. Compt. rend. Soc. Biol., 90: 98-100, 1924.
8. Bigwood, E. J.: L'équilibre physico-chemique du sang dans l'épilepsie. L'équilibre acide-base. Ann. de Med., 15: 119-148, 1924.
9. Bigwood, E. J.: L'équilibre physico-chemique du sang dans l'épilepsie. L'ion calcium—I. partie. Jour. Physiol. et Path. Gen., *22*: 70-78, 1924.
10. Bigwood, E. J.: L'équilibre physico-chemique du sang dans l'épilepsie. L'ion calcium. II. partie. Jour. Physiol. et Path. Gen., *22*: 94-108, 1924.
11. Bigwood, E. J.: Perturbation de l'équilibre acide-base du sang dans l'épilepsie. Compt. rend. Soc. Biol., 89: 839-841, 1923.
12. Bisgaard, A., and Larsen, E. J.: Dérèglementation neutralisatrice. Compt. rend. Soc. Biol., 84: 1047-1048, 1921.
13. Bisgaard, A., and Nørvig, J.: Fortgesetzte Untersuchungen über die Neutralitätsregulation bei der genuinen Epilepsie. Ztschr. f. d. ges. Neurol. u. Psychiat., 83: 469-480, 1923.
14. Bisgaard, A., and Nørvig, J.: Recherches sur la réglementation neutralisatrice dans las cas d'èpilepsie proprement dite. Compt. rend. Soc. Biol., 84: 159-160, 1921.
15. Bisgaard, A., and Nørvig, J.: Recherches sur la réglementation neutralisatrice dans les cas d'épilepsie proprement dite. Compt. rend. Soc. Biol., 84: 318-322, 1921.
16. Blumgarten, A. S., and Rohdenburg, G. L.: Mineral salt content of the blood in disease. Arch. Int. Med., 39: 372-384, 1927.
17. Bornstein, A.: Über die Lecithinämie der Geisteskranken. Ztschr. f. d. ges. Neurol. u. Psychiat., 6: 605-608, 1911.
18. Bouttier, H., and Mestrezat, W.: Note sur les variations de l'azotémie dans un cas d'état de mal épileptique. Rev. Neurol., 36: 1224-1227, 1920.
19. Brain, R.: Discussion on epilepsy. Proc. Roy. Soc. Med., 20: 861-863, 1926-27.
20. Brühl, F.: Weitere blutchemische Untersuchungen zur Pathologie des epileptischen Krampfanfalles. Ztschr. f. d. ges. Neurol. u. Psychiat., 84: 642-651, 1923.
21. Brühl, F.: Weitere Untersuchungen über den Serum-Eiweisswert beim epileptischen Krampfanfall und seine Beziehungen zum Blutdruck. Ztschr. f. d. ges. Neurol. u. Psychiat., 83: 656-659, 1923.
22. Cannon, W. B.: The Wisdom of the Body, 312 pp. New York, W. W. Norton, 1932.
23. Cantarow, A., Calcium studies. VI. The effect of parathyroid extract on the diffusibility of calcium in human beings. Arch. Int. Med., 44: 834-842, 1929.
24. Cassaro, P., Chlorides in epileptics and their variation in relation to the convulsive attacks. Clin. d. mal. nerv. and emt., Palermo, 1932.
25. Caussade, L., Vérain, M. and Jacob, Study of the acid-base equilibrium of blood in epilepsy. Rev. med. de Nancy, 64: 311-316, 1936.

26. Charon, R., and Briche, E.: Recherches cliniques sur l'alcalescence du sang et les injections de solutions alcalines chez les épileptiques. Arch. de Neurol., 4: 465-485, 1897.
27. Clark, L. P.: The psychobiologic concept of essential epilepsy. In: Epilepsy and the Convulsive State, Ass. Research and Nerv. Ment. Dis., pp. 65-79, 1922.
28. Claude, H., Dublineau, J., and Bonnard: Les lipides du sang dans les maladies mentales. Biol. Med., 25: 94-114, 1935.
29. Claude, H., Targowla, R., and Badonnel. La cholestérine du sang dans les psychopathies. Compt. rend. Soc. Biol., 94: 102, 1926.
30. Cobb, S.: Review of neurology and psychiatry for 1935-1936. Arch. Int. Med., 58: 1111-1123, 1936.
31. Contini, M.: The plasma proteins in epilepsy and their variations in relation to attacks. Rassegna di studi psychiat., 25: 818-825, 1936.
32. Craig, R. N.: Blood-sugar curves in certain mental disorders. Lancet, 1: 925-927, 1927.
33. Cruickshank, J., and Tisdall, C. J.: The cholesterol content of the serum in mental diseases. Jour. Ment. Sci., 62: 168-173, 1916.
34. Daly, I. de B., Pryde, J. and Walker, J.: The blood sugar in cases of epilepsy. Brit. Med. Jour., 1: 232-233, 1924.
35. Dautrebande, L.: L'equilibre acide-base du sang dans l'épilepsie. Compt. rend. Soc. Biol., 94: 133-135, 1926.
36. de Crinis, M.: Die Beteiligung der Humoralen Lebensvorgänge des Menschlichen Organismus am Epileptischen Anfall. 80 pp., Berlin, Julius Springer, 1920.
37. de Crinis, M.: Über den Stoffwechsel beim epileptischen Symptomenkomplex. Ztschr. f. d. ges. Neurol. u. Psychiat., 99: 718-755, 1925.
38. Delaville, M., and Tcherniakofsky. Le chlore sanguin au cours de divers syndromes neuro-psychiatriques. Compt. rend. Soc. Biol., 100: 342-343, 1929.
39. Dide, M., Fages, G., and Baudouin, A.: Glycémie et glycorachie chez les aliénés. Presse Med., 32: 425-427, 1924.
40. di Renzo, F.: Ricerche sul comportamento della riserva alcalina del sangue e del pH urinario negli epilettici nei periodi intervallari e durante le crisi convulsive. Riv. de pat. nerv. e ment., 36: 549-572, 1930.
41. di Renzo, F., and Tomasino, A.: Ricerche sul comportamento del calcio e del potassio nel sangue degli epilettici nei periodi intervallari e durante gli accessi convulsivi. Rassegna di studi psychiat., 19: 503-518, 1930.
42. Drury, K. K., and Farran-Ridge, C.: Some observations on the types of blood-sugar curve found in different forms of insanity. Jour. Ment. Sci., 71: 8-29, 1925.
43. Dufour, H., and Semelaigne, G.: Sur un trouble humoral passager précédant la crise d'épilepsie. Variation de l'urée sanguine. Bull. et men. Soc. med. d'hop. de Paris, 44: 58-60, 1920.

44. Duncan, A. G.: The serum cholesterol in mental disorders. Jour. Ment. Sci., 76: 284-291, 1930.
45. Felsen, J.: Laboratory studies in epilepsy. Arch. Int. Med., 46: 180-217, 1930.
46. Fletcher, R. T., and Peden, O. D.: A note on the blood chemistry in epilepsy. Lancet, 1: 1382-1384, 1935.
47. Freudenberg, E.: Beitrag zur Epilepsiebehandlung. Ztschr. f. Kinderh., 41: 46-50, 1926.
48. Frigerio, A.: Ricerche sull 'azoto non coagulabile del sangue in alcune mallatie mentali. Riv. di patol. nerv., 26: 301-331, 1921.
49. Frisch, F., and Fried, E.: Die Serumeiweisskörper bei Epilepsie. Ztschr. f. d. ges. Exp. Med., 56: 766-777, 1927.
50. Frisch, F., and Fried, E.: Zur Frage der angeblichen Alkalose bei Epilepsie. Ztschr. f. d. ges. Exp. Med., 49: 462-470, 1926.
51. Frisch, F., and Walter, K.: Untersuchungen bei periodischer Epilepsie. I. Ztschr. f. d. ges. Neurol. u. Psychiat., 79: 366-384, 1922.
52. Frisch, F., and Weinberger, W.: Untersuchungen bei periodischer Epilepsie. II. Ztschr. f. d. ges. Neurol. u. Psychiat., 79: 576-588, 1922.
53. Genzel, A.: Über den Blutzuckergehalt bei Psychosen. Monatschr. f. Psychiat. u. Neurol., 55: 327-336, 1923-24.
54. Geyelin, H. R.: Relation between the acid and alkali of the blood in epilepsy. J. A. M. A., 81: 330, 1923.
55. Geyelin, H. R., Bigwood, E. J., and Wheatley, M. A.: The reaction of the blood in epilepsy. Proc. Soc. Exp. Biol. & Med., 21: 227-228, 1923-24.
56. Goebel, F.: Cholésterine du liquide céphalorachidien et du sang dans les maladies psychiques. Compt. rend. Soc. Biol., 90: 1191-1193, 1924.
57. Goodall, E.: Blood cholesterol during the menstrual and epileptic cycles. Lancet, 1: 384-386, 1929.
58. Gosden, M., Fox, J. T., and Brain, W. R.: The cholesterol of the blood plasma in epilepsy. Lancet, 2: 12-16, 1929.
59. Gozzano, M.: L'equilibrio acido-base negli epilettici. Riv. di pat. nerv. e. ment., 35: 128-131, 1930.
60. Gray, H., and McGee, L. C.: Cholesterol content of blood in epilepsy and in feeblemindedness. Arch. Neurol. Psychiat., 28: 357-369, 1932.
61. Griffiths, G. M.: Calcium in epilepsy. Jour. Neurol. Psychopath., 15: 29-38, 1934.
62. Guirdham, A., and Pettit, A. W.: The liver in epilepsy: a study in the application of the levulose tolerance test. Jour. Ment. Sci., 82: 119-130, 1936.
63. Hamilton, B.: A comparison of the concentrations of inorganic substances in serum and spinal fluid. Jour. Biol. Chem., 65: 101-115, 1925.
64. Hartenberg, M.: Urée sanguine constante d'Ambard, et règime hypoazoté chez les épileptiques. Encephale, 19: 539-540, 1924.

65. Haury, V. G., and Hirschfelder, A. D.: Some observations upon blood glucose in epilepsy. Proc. Soc. Exp. Biol. Med., 32: 1658-1659, 1934-35.
66. Heidema, S. T.: Blutzuckerbestimmungen bei psychiatrischen und neurologischen Patienten. Ztschr. f. d. ges. Neurol. u. Psychiat., 48: 111-129, 1919.
67. Hepburn, J. S., and Niebaum, A. H.: Blood calcium in mental diseases. Jour. Amer. Inst. Homeopathy, 24: 559-560, 1931.
68. Hirschfelder, A. D., and Haury, V. G.: Low plasma magnesium and high plasma potassium in essential epilepsy. Jour. Pharm. Exper. Therap., 57: 127, 1936.
69. Hirschfelder, A. D., and Haury, V. G.: Variations in plasma magnesium and potassium in epilepsy. Proc. Soc. Exp. Biol. & Med., 33: 40-42, 1935-36.
70. Hoeffel, G., and Moriarty, M.: The effect of fasting on the metabolism of epileptic children. Amer. Jour. Dis. Child., 28: 16-24, 1924.
71. Holmström, R.: Blutzuckerbestimmungen bei Epileptikern. Upsala Lakaref Forhand., 29: 17-56, 1924.
72. Hopkins, H.: Chemical Studies in the epileptic syndrome. II. Nocturnal and diurnal rhythm in blood chemistry. Amer. Jour. Psychiat., 92: 75-88, 1935.
73. Hopkins, H.: Chemical studies in the epileptic syndrome. I. Whole blood cholesterol. Jour. Nerv. Ment. Dis., 77: 601-616, 1933.
74. Jacchia, L., and Fattovich, F.: Contributo allo studio della patogenesi del' epilessia. Le variazioni glicemiche e gli equilibri del sistema glicoregolatore negli epilettici. Minerva Med., 23 (2): 517-526, 1932.
75. Jacchia, L., and Fattovich, G.: Le variazioni colesterinemiche e gli equilibri del ricambio lipoideo negli epilettici. Gior. di psichiat. e de neuropat., 62: 247-262, 1934.
76. Jacobi, W.: Cholesterinstoffwechsel und Konstitutionspathologie der Psychosen. Monatschr. f. Psychiat. u. Neurol., 62: 296-302, 1927.
77. Jacobi, J., and Koritter, H.: Über das Verhalten des Nüchternblutzuckers und der alimentären Blutzuckerkurven in der Neurologie und Psychiatrie. Psychiat-neurol. Wchnschr., 34: 1-7, 1932.
78. Jarloev, E.: Sur l'équilibre acido-basique du sang humain, étudié dans ses rapports avec diverses affections. Compt. rend. Soc. Biol., 84: 156-158, 1921.
79. Jellinek, E. M.: Some uses and abuses of statistical method in psychiatry. Biom. Bull., 1: 97-108, 1937.
80. Kanner, L.: Child Psychiatry. 527 pp., Springfield, Ill., C. C. Thomas, 1935.
81. Katzenelbogen, S.: The distribution of calcium between blood and fluid and the carbon dioxide content of the blood in epilepsy. Jour. Nerv. Ment. Dis., 74: 636-643, 1931.
82. Kersten, H.: Ergebnisse zur Frage des elementaren Krampfes. Ztschr. f. d. ges. Neurol. u. Psychiat., 63: 48-58, 1921.

83. Kilduffe, R. A.: The Clinical Interpretation of Blood Chemistry. 629 pp., 2 ed., Philadelphia, Lea & Febiger, 1931.
84. Koechig, I.: The calcium content of the blood in pathologic conditions. Jour. Lab. Clin. Med., 9: 679-685, 1923-24.
85. Kooy, F. H.: Blood-sugar and psychoses. (Psychiat. en Neurolog. Bladen, 1920, Nos. 3-4, May-August, p. 143) Jour. Nerv. Ment. Dis., 56: 177-178, 1922.
86. Kooy, F. H.: Hyperglycaemia in mental disorders. Brain, 42: 214-289, 1919-20 .
87. Kora, T.: On the phosphorus content of the blood serum of mental patients and of healthy individuals under special mental influences. Jap. Jour. Med. Sci., VIII, 1: 53-70, 1927.
88. Labin, B.: Recherches sur la glycémie chez les aliénés et dans quelques maladies nerveuses. Compt. rend. Soc. Biol., 96: 1172-1174, 1927.
89. Lennox, W. G.: Certain chemical and physiological conditions which may influence seizures. Epilepsy and the Convulsive State, Ass. Research Nerv. Ment. Dis., pp. 475-490, 1922.
90. Lennox, W. G.: Studies in the chemistry of the blood in epilepsy. Arch. Neurol. Psychiat., 10: 264, 1923.
91. Lennox, W. G.: The cerebral circulation. XV. The effect of mental work. Arch. Neurol. Psychiat., 26: 725-730, 1931.
92. Lennox, W. G.: The physiological pathogenesis of epilepsy. Brain, 59: 113-121, 1936.
93. Lennox, W. G.: The problem of epilepsy. Epilepsia, 1: 23-30, 1937.
94. Lennox, W. G.: The relation between seizures in epilepsy and the acid-base balance in the blood. Jour. Clin. Invest., 4: 429, 1927.
95. Lennox, W. G., and Allen, M. B.: Metabolism in Epilepsy. IV. The bicarbonate content of the blood. Arch. Neurol. Psychiat., 20: 155-161, 1928.
96. Lennox, W. G., and Allen, M. B.: Studies in epilepsy. XI. The calcium content of the blood and of the spinal fluid. Arch. Neurol. Psychiat., 24: 1199-1205, 1930.
97. Lennox, W. G., and Allen, M. B.: Studies in epilepsy. X. The chloride content of the blood and spinal fluid. Arch. Neurol. Psychiat., 23: 525-530, 1930.
98. Lennox, W. G., and Allen, M. B.: Studies in epilepsy. V. The fibrin content of the blood. Arch. Neurol. Psychiat., 20: 345-353, 1920.
99. Lennox, W. G., and Bellinger, M.: Studies of metabolism in epilepsy. III. The blood sugar curve. Arch. Neurol. Psychiat., 18: 395-413, 1927.
100. Lennox, W. G., and Cobb, S.: Epilepsy, from standpoint of physiology and treatment. Medicine, 7: 105-290, 1928.
101. Lennox, W. G., and Gibbs, E. L.: Oxygen saturation of blood draining the brain and the limbs of patients with epilepsy. Arch. Neurol. Psychiat., 36: 13-18, 1936.
102. Lennox, W. G., and Gibbs, E. L.: Oxygen saturation of the arterial blood in epilepsy. Arch. Neurol. Psychiat., 35: 1198-1202, 1936.

103. Lennox, W. G., O'Connor, M., and Bellinger, M.: Chemical changes in the blood during fasting in the human subject. Arch. Int. Med., 38: 553-565, 1926.
104. Lennox, W. G., O'Connor, M., and Bellinger, M.: Studies of metabolism in epilepsy. II. The sugar content of the blood. Arch. Neurol. Psychiat., 18: 383-394, 1927.
105. Lennox, W. G., O'Connor, M. F., and Wright, L. H.: Studies of the metabolism in epilepsy. I. The nonprotein nitrogenous constituents of the blood. Arch. Neurol. Psychiat., 11: 54-63, 1924.
106. Longo, V.: Sul contenuto in Ca e K Del siero di sangue di soggetti normali ed epilettici. Boll. Soc. Ital. Biol. Sper., 3: 112-114, 1928.
107. Luck, J. M., Thacker, G., and Marrack, J.: Ammonia in the blood of epileptics. Brit. Jour. Exp. Path., 6: 276-279, 1925.
108. Lui, A.: Sul comportarsi dell'alcalinita del sangue in alcune forme psicopatiche e nell'epilessia. Riv. sper. di freniat. et de med. leg., 24: 1-19, 1898.
109. Madsen, J.: On the calcium, potassium and sodium concentrations in the serum of epileptics. Acta Psychiat. Neurol., 7: 343-362, 1932.
110. MacKay, G. W. J., and Barbash, H.: The glucose tolerance curve in epilepsy. Jour. Ment. Sci., 77: 83-85, 1931.
111. Malerba, G. L.: Il tasso colesterinico del siero di sangue in varie forme morbose. Riforma Med., 37: 602-605, 1921.
112. Marrack, J., and Thacker, G.: The reaction of the blood in epilepsy, with a discussion of colorimetric methods of estimating the pH of plasma. Brit. Jour. Exp. Path., 7: 265-273, 1926.
113. Massazza, A.: La riserva alcalina e l'equilibrio acido-base negli epilettici. Riv. di pat. nerv. e ment., 35: 115-128, 1930.
114. Mazza, A.: Ricerche comparative die cloruri del siero di sangue in alcune malattie nervose e mentali. Riv. sper. Bi. freniat. e med. legale, 56: 744-751, 1932.
115. McFarland, R. A., and Goldstein, H.: Biochemistry of manic-depressive insanity: a review. Amer. Jour. Psychiat., 96: 21-58, 1939.
116. McFarland, R. A., and Goldstein, H.: Biochemistry of the psychoneuroses: a review. Amer. Jour. Psychiat., 93: 1073-1095, 1937.
117. McFarland, R. A., and Goldstein, H.: The biochemistry of dementia præcox: a review. Amer. Jour. Psychiat., 95: 509-552, 1938.
118. McKenzie, B. W., and McChesney, E. W.: Proteins of the blood serum in cases of essential epilepsy. Arch. Neurol. Psychiat., 34: 764-767, 1935.
119. McLaughlin, F. L., and Hurst, R. H.: Acid-base equilibrium of blood in epilepsy. Quart. Jour. Med., 2(N. S.): 419-429, 1933.
120. McLean, J. H.: The blood cholesterol in epilepsy in relation to treatment by dehydration and ketogenesis. Jour. Ment. Sci., 80: 82-86, 1934.
121. McQuarrie, I., Bloor, W. R., Husted, C., and Patterson, H. A.: The lipids of the blood plasma in epilepsy. I. A statistical study of single determinations in 100 epileptic and 32 "normal" subjects. Jour. Clin. Invest., 12: 247-254, 1933.

122. McQuarrie, I., Husted, C., and Bloor, W. R.: The lipids of the blood plasma in epilepsy. II. Variations of lipids in relation to occurrence of seizures. Jour. Clin. Invest., 12: 255-265, 1933.

123. McQuarrie, I., and Keith, H. M.: Experimental study of the acid-base equilibrium in children with idiopathic epilepsy. Amer. Jour. Dis. Child., 37: 261-277, 1929.

124. Mennato, de M.: L'equilibrio acido-base negli epilettici induzioni patogenetiche. Rassegna di studi psychiat., 24: 466-479, 1935.

125. Messina, B. G.: Calcium and potassium in blood of epileptics. Preliminary report. Riv. san. siciliana, 23: 33-35, 1935.

126. Metcalfe, G. E.: Induced hypoglycemic shock in cryptogenic epilepsy. Psychiat. Quart., 13: 348-356, 1939.

127. Meyer, M.: Serologische Studien zur Pathologie des epileptischen Krampfanfalls. Zentralbl. f. d. ges. Neurol. u. Psychiat., 41: 726-727, 1925.

128. Meyer, M., and Brühl, F.: Zur Pathologie des epileptischen Krampfanfalls. Ztschr. f. d. ges. Neurol. u. Psychiat., 75: 158-188, 1922.

129. Michaels, J. J., and Searle, O. M.: Calcium content of the cerebrospinal fluid, blood serum, and serum ultrofiltrate. Its relation to the clinical findings in 80 neuropsychiatric patients. Arch. Neurol. Psychiat., 33: 330-341, 1935.

130. Minchin, R. L. H.: The blood calcium in "idiopathic" epilepsy. Jour. Neurol. Psychopath., 17: 314-317, 1937.

131. Minchin, R. L. H.: The factor of hypoglycaemia in the aetiology of idiopathic epilepsy. Jour. Ment. Sci., 79: 659-676, 1933.

132. Modio, E.: Sul fosforo inorganico del sangue nei soggetti epilettici. Riv. di pat. nerv., 34: 524-533, 1929-30.

133. Mørdre, S. K.: Litt om alkalireservens forhold i organismen i nogen tilfaelder av epilepsi. Nørsk mag. Laegevid, 83: 603-610, 1922.

134. Mumford, P. B., and Parkin, G. G.: Some observations on fasting blood-sugar in certain mental states. Jour. Ment. Sci., 69: 330-339, 1923.

135. Munch-Petersen, C. J., and Schou, H. I.: Investigations into the sugar-metabolism in epileptics, especially the sugar threshold in adrenalin- and glucosis-hyperglycemia. Acta Psychiat. Neurol., 6: 545-563, 1931.

136. Neri, A.: La curva glicemica alimentare negli epilettici. Biochim. e terap. sper., 22: 162-168, 1935.

137. Nielsen, J. M.: Convulsions of undetermined etiology. Studies of the blood sugar. Arch. Neurol. Psychiat., 31: 1055-1062, 1934.

138. Nielsen, J. M.: Etiology of epileptiform convulsions. Med. Jour. Rec., 122: 338-340, 1925.

139. Nørvig, J.: Untersuchungen über Stoffwechselanomalien bei Psychosen. Die sogenannte genuine Epilepsie. Acta Med. Scand., 60: 211-275, 1924.

140. Notkin, J.: Greeff, J. G. W., Pike, F. H., and Killian, J. A.: Changes in the clinical signs and laboratory findings in various types of psychoses under the influence of subcutaneous administration of oxygen. Amer. Jour. Psychiat., 12: 1271-1303, 1933.

141. Obregia, and Urechia, C. I: Les variations de l'urea dans le sang, le liquide cephalo-rachidien et l'urine des epileptiques. Encephale, 17: 134-152, 1914-19.

142. Ornstein, L.: Sur la cholestérinémie chez les aliénés. Compt. rend. Soc. Biol., 93: 1622-1623, 1925.

143. Osnato, M., Killian, J. A., Garcia, T., and Mattice, M. R.: Comparative chemical studies of the blood and spinal fluid in epilepsy. Brain, 50: 581-600, 1927.

144. Page, I. H.: Chemistry of the brain. C. C. Thomas & Co., Baltimore, 1937, 444 pp.

145. Pagniez, P., and Salles, P.: Contribution à la pathogénie de l'épilepsie. Bull. Acad. Med., 113: 267-268, 1935.

146. Parhon, C., and Ornstein, J.: Sur la calcémie et la calcium-rachie chez les épileptiques ainsi que dans certaines psychoses. Bull. et men. Soc. med. d'hop. de Paris, 54: 742-744, 1930.

147. Parhon, C., Urechia, C., and Popea, A.: Note sur la lipoidhémie chez les aliénés. Ztschr. f. d. ges. Neurol. u. Psychiat., 7: 1021, 1913.

148. Parhon, C., and Werner, G.: Recherches sur la calcémie la potassémie et le rapport k: Ca dans les maladies mentales. Ann. méd. psychol., 2: 87-88, 1932.

149. Patterson, H. A.: Some observations on blood-calcium content in epilepsy. In: Epilepsy and the Convulsive State, Ass. Research Nerv. Ment. Dis., pp. 387-398, 1922.

150. Peters, J. P., and Van Slyke, D. D.: Quantitative Chemical Chemistry. Vol. I. Interpretations, 1264 pp. Baltimore, Williams and Wilkins, 1931.

151. Pezzali, G.: Ricerche sul contenuto del sangue in azoto (residuo) cloruri, colesterina, grassi, glucosi, indacano e calcio nell'epilessia. Riforma Med., 39: 433-437, 1923.

152. Pighini, G.: Über die Menge Cholesterins und Oxycholesterins des Serums bei verschiedenen Formen von Geisteskrankheiten. Ztschr. f. d. ges. Neurol. u. Psychiat., 4: 158-188, 1910-11.

153. Piotrowski, G.: La proteinemie dans les affections mentales. Schweiz. Med. Wchnschr., 68: 137-138, 1938.

154. Poli, G.: Il valore delle oscillazioni nell'equilibrio acido-basico per lo scoppio dell'accesso convulsivo epilettico. Riv. di pat. nerv. e ment., 39: 100-110, 1932.

155. Pollock, L. J., and Boshes, B.: Carbohydrate metabolism in epilepsy. Arch. Int. Med., 59: 1000-1023, 1937.

156. Popea, A., and Vicol, A.: Les variations de la cholestérinèmie durant l'accès épileptique. Compt. rend. Soc. Biol., 93: 749-750, 1925.

157. Prior, G. P. U., and Jones, S. E.: Calcium and epilepsy: A preliminary report. Med. Jour. Australia, 3 (1): 199-203, 1916.

158. Pugh, R.: On certain blood changes in idiopathic epilepsy. Brain, 25: 501-539, 1902.
159. Pugh, R.: The alkalinity of the blood in mental diseases. Jour. Ment. Sci., 49: 71-81, 1903.
160. Rampini, M.: Cholesteremia in convulsive epilepsy of infancy and in spasmophilia. Riv. di Clin. pediat., 32: 1281-1309, 1934.
161. Reid, B.: A biochemical study of the blood and urine in mental disorders. Jour. Ment. Sci., 73: 254-257, 1927.
162. Reiter, P. J.: Recherches sur le calcium total du sérum chez les épileptiques au sens propre et dans les etats voisins, Compt. rend. Soc. Biol., 92: 1325-1326, 1925.
163. Robinson, S. H. G., Brain, W. R., and Kay, H. D.: The association of low blood cholesterol with the occurrence of fits in epileptics. Lancet, 2: 325-326, 1927.
164. Rosen, I., Krasnow, F., and Notkin, J.: Cholesterol and lecithin content of the blood in cryptogenic epilepsy. Arch. Neurol. Psychiat., 29: 154-156, 1933.
165. Sachs, I.: Besteht ein Zusammenhang zwischen Epilepsie und Tetanie? Deutsch. z. Nervenh., 92: 304-310, 1926.
166. Salvesen, H. A.: Blodkalken under normale og under visse patologiske tilstande. Norsk. mag. Laegevid., 84: 1047-1052, 1923.
167. Schou, H. J.: Epilepsy and alimentary hypoglycemia (preliminary communication). Acta psychiat. et neurol., 12: 533-543, 1937.
168. Schryver, D.: Blutzucker und Schizophrenie. Ztschr. f. d. ges. Neurol. u. Psychiat., 89: 179-196, 1923.
169. Schultz, J. H.: Ueber das Verhalten der Alkaleszenz des Blutes und der weissen und roten Blutkörperchen bei Nerven-und Geisteskranken. I. Ueber alkaleszenz des Blutes bei Nerven-und Geisteskranken. Monatschr. f. Psychiat. u. Neurol., 22: 21-61, 1907.
170. Schwab, S. I.: Diagnostic value of blood sugar curves in neurology. Arch. Neurol. Psychiat., 8: 401-411, 1922.
171. Scott, M., and Pigott, A. W.: Calcium content of the blood serum during an epileptic convulsion. Arch. Neurol. Psychiat., 36: 590-592, 1936.
172. Shaw, E. B., and Moriarty, M.: Hypoglycemia and acidosis in fasting children with idiopathic epilepsy. Amer. Jour. Dis. Child., 28: 553-567, 1927.
173. Shera, G.: Investigation of the acid-base balance in mental cases, with special reference to epilepsy. Jour. Ment. Sci., 74: 454-459, 1928.
174. Sherrill, J. W.: Metabolic observations in psychiatric cases. Jour. Metabolic Research, 5: 129-143, 1924.
175. Simpson, T., and Barker, M. H.: Rôle of anoxia in production of epileptiform seizures. Arch. Int. Med., 61: 208-222, 1938.
176. Stone, T. T., and Chor, H.: Water metabolism in relation to convulsions. Arch. Neurol. Psychiat., 38: 798-817, 1937.
177. Ström-Olsen, R.: The blood sugar curve in mental cases. Lancet, 1: 128-130, 1932.

178. Talbot, F. B.: Treatment of epilepsy. 308 pp. New York, MacMillan, 1930.
179. Talbot, F. B., Metcalf, K. M., and Moriarty, M. E.: Epilepsy: chemical investigations of rational treatment by production of ketosis. Amer. Jour. Dis. Child., 33: 218-225, 1927.
180. Targowla, R., Badonnel, M., and Berman, A.: Sur la cholestérinèmie dans les maladies mentales. Encephale, 18: 138-140, 1923.
181. Telatin, L.: Carbohydrate metabolism in mental diseases; comparative behavior of glycemic and glycorrhachic curves after dextrose injections in epilepsy and dementia præcox. Gior. di psichiat. e de neuropat., 63: 151-173, 1935.
182. Timme, W.: Introduction. Presidential Address. In: Epilepsy and the Convulsive State. Ass. Research Nerv. Ment. Dis., pp. 1-4, 1922.
183. Tingey, A. H., and Hardwick, S. W.: Serum calcium and sodium in some psychotic disorders. Jour. Ment. Sci. 81: 528-532. 1935.
184. Torres Lopez, A. J.: Las perturbaciones del equilibrio acido-basico y su relacion con la epilepsia. Archivos de Neurobiol., 10: 95-113, 1930.
185. Tsuchiya, S.: Über den Verlauf der Blutzuckertoleranz bei Psychosen. Ztschr. f. d. ges. Neurol. u. Psychiat., 91: 23-25, 1924.
186. Tyson, G. N., Jr., Otis, L., and Joyce, T. F.: A study of blood sugar of epileptics. Amer. Jour. Med. Sci., 190: 164-169, 1935.
187. Vollmer, H.: Zur Pathogenese der genuinen Epilepsie. Ztschr. f. d. ges. Neurol. u. Psychiat., 84: 546-560, 1923.
188. Weeks, D. F., Renner, D. S., Allen, F. M., and Wishart, M. B.: Observations on fasting and diets in the treatment of epilepsy. Jour. Metabolic Research, 3: 317-364, 1923.
189. Weil, A., and Liebert, E.: Phosphorus content of the blood serum during the epileptic seizure. Arch. Neurol. Psychiat., 37: 584-588, 1937.
190. Weiss, S., and Lennox, W. G.: The cerebral circulation. XVII. Cerebral blood flow and the vasomotor response of the minute vessels of the human brain to histamine. Arch. Neurol. Psychiat., 26: 737-744, 1931.
191. Weston, P. G.: Analyses of blood of insane patients. Arch. Neurol. Psychiat., 3: 147-150, 1920.
192. Weston, P. G.: Sugar content of the blood and spinal fluid of insane subjects. Jour. Medical Research, 35: 199-208, 1916-17.
193. Wladyczko, S.: La cure sucrée dans l'épilepsie. Presse Med., 33: 1475-1477, 1925.
194. Wuth, O.: Blood changes in convulsions, especially in epilepsy. Bull. Johns Hopkins Hosp., 38: 389-408, 1926.
195. Wuth, O.: Der Blutzucker bei Psychosen. Ztschr. f. d. ges. Neurol. u. Psychiat., 64: 83-95, 1921.
196. Wuth, O.: Untersuchungen über die Körperlichen Störungen bei Geisteskranken. 113 pp. Berlin, Julius Springer, 1922.
197. Ziskind, E., and Bolton, R.: Insulin hypoglycemia in epilepsy. Arch. Neurol. Psychiat., 36: 331-341, 1936.

EVALUATION OF THE ELECTROENCEPHALOGRAMS OF SCHIZOPHRENIC PATIENTS.*

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Introduction.

Schizophrenia is a term which labels a very high percentage of mental patients in hospitals. The sub-classifications made on the basis of the psychiatric picture appear to involve behavior, mental content and their relationships. Routine physical and neurological examinations are usually made on these patients when they are admitted to a mental hospital, but the findings have not led to a real insight into the schizophrenic condition, and neither have current methods of treatment yet told us what made the schizophrenic patient ill or what underlies his behavior. Some patients get well spontaneously, and they have little understanding or insight as to why they became ill or why they got well.

In the last five years in the United States, electroencephalography has been developing and is giving us new leads and a more objective tool for studying the behavior of the human brain. Electroencephalograms are electrical records of the physiological activity of the cortex, which can be amplified and recorded automatically on a tape, like electrocardiograms. The technique is such that the patient is not hurt or uncomfortable in any way, when recorded.

Comparison of EEG's of Schizophrenic and Normal Subjects.

At the Harvard Medical School we have been making an intensive study of people selected on the basis of having a normal history from birth; absence of any history of accidents involving the central nervous system, or psychiatric difficulties requiring advice

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or help; and whose daily life and behavior over a period of at least five years have been normal as far as can be ascertained. Our series of the records of normal individuals is being constantly reanalyzed and further refined in the light of our increasing understanding of variations in the patterns. For instance, there have been a few individuals in classifications of our "normal" group whose records are clearly different from the majority of the group of patterns. Their records are dysrhythmic at times and are similar to the patterns of known epileptics, yet these individuals have never had an epileptic seizure. Inquiry into the heredity of some of these people revealed the presence of epilepsy or migraine headaches. Consequently in order to extend the study of these normal people so that our baseline can be even more stable, a sub-classification including people whose family history is at least indicative of nervous system disturbances is being made. The records of these individuals remain, however, in the group of "normals."

Such a baseline of reference for studying special groups such as a mental hospital group have revealed these facts:[3]

The patient in a mental hospital cannot be distinguished from the "normal" by his EEG. The EEG's of patients in a mental hospital as a group, however, are less stable than those of "normal" individuals who constitute our baseline group. The relatively slight variabilities seen in some of the records of our normal group of EEG's are highly exaggerated in the mental patients. There is no sharp line, but the trend is toward a striking increase in abnormal features, a decrease in stability and synchronization of waves, as well as a breakdown in the normal distribution and relationships of the rhythms from one part of the brain to another.[7, 13, 8, 3]

The EEG's of 132 schizophrenic patients at McLean Hospital and Metropolitan State Hospital have been recorded. The histories at McLean Hospital are very comprehensive and have made possible a study of the early behavior of the schizophrenic individual. The importance of this will be discussed.

Of the 66 patients in this series at McLean Hospital, 42 have histories of mental illness in the parents' or grandparents' generations.

The 66 patients at the Metropolitan State Hospital are chronic patients. The histories are not adequate for intensive study, but two sets of EEG's on each patient, taken 6 months apart, were found to be more normal and less variable than the group at McLean Hospital. The reason for this may be that the Metropolitan State Hospital admits only chronic patients who are not subject to continually disturbed behavior and who are amenable to ward routine. Thus there is a definite selection of the type of patient.

Dividing the schizophrenics into their sub-groups as classified in the hospitals, the types are listed as simple, paranoid, catatonic, hebephrenic, so-called "other types" and "mixed types." An attempt was made to find a correlation between the EEG and these sub-groups. This was difficult because some patients after a year may be rediagnosed, so that a manic-depressive is later called a schizophrenic, and a catatonic a hebephrenic or vice versa.

Classification of the EEG's of Schizophrenic Patients.

On the basis of repeated and extensive EEG studies of these patients, the EEG patterns fall into three main groups:

Group I.—Those patterns which appear to be essentially normal (Fig. 1).

Group II.—Dysrhythmic patterns which are indistinguishable from the records of patients not in mental hospitals but known to be suffering from convulsive disorders such as epilepsy, its variants or equivalents (Fig. 2).

Group III.—"Choppy" patterns which suggest the possibility of localized or generalized irritative lesions, cysts, atrophy and other pathological conditions which cannot yet be adequately defined (Fig. 3).

A fourth group of brain patterns might well be added, representing a mixed group showing features of both Groups II and III, where there is a good deal of overlap.

Group I includes the normal patterns. The histories of the majority of patients of this group indicate a type of schizophrenia which has a very gradual onset and often may not be clearly revealed until adolescence. In this group, more than in Group II, are patients whose history and heredity give little definite evidence of a schizophrenic tendency. In the hospital, the behavior of many

of these patients tends to become stable and the patients do not *appear* to show rapid deterioration. They may become withdrawn. They do not make much trouble and settle down to the routine and

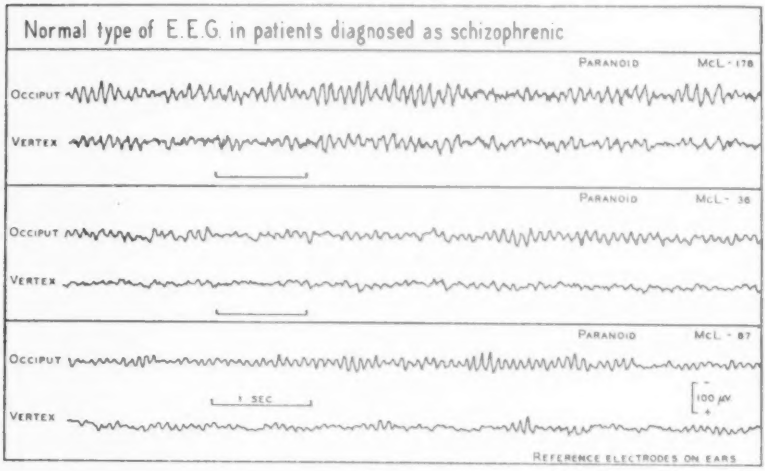


FIG. 1.

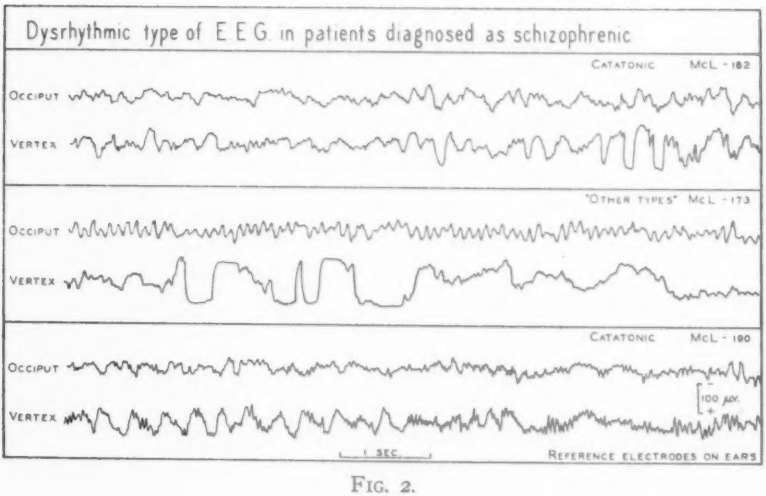


FIG. 2.

remain for years essentially the same. They are the ones who perhaps have previously received less specific attention from nurses and doctors.

We have had the impression from the beginning of our studies of mental patients that the most normal records among the schizophrenics have been those from patients carrying the diagnosis of "paranoid" schizophrenia. For example, we selected the three records for Fig. 1 on the basis of normality and later discovered that the diagnosis was "paranoid" type in each case.

Group III is at present the smallest group of this series and will be discussed next. The main feature of the records in this group is described as "choppy," for want of a better term. The EEG usually represents a diffuse rather than localized abnormal activity, though both are seen. Until more pneumoencephalograms, biochemical studies and autopsies can be obtained on mental patients, we cannot adequately identify the conditions associated with many of these records. In this third group are records which indicate some brain lesion or abnormality which pneumoencephalograms might help to evaluate. For example, the patient McL-171 in Fig. 3 has been regarded as schizophrenic for nearly 10 years and was in a hospital for more than 7 years. The onset was gradual, his heredity negative, the eight siblings normal. His abnormal response to metrazol therapy led to further inquiry into his neurological condition, and from a skull plate he was diagnosed as having a hemangioma in the right occipital bone. Then a pneumoencephalogram was done, and the condition was found to be a large inoperable porencephalic cyst in the right occipital lobe. The rest of the brain of this patient showed lesser, but diffuse, changes. There was an old fracture of the inner table of the skull in the region of the right occiput. His history states that when 7 months of age he was dropped 15 feet, remained unconscious for a week, and had to wear an ice-cap on his head for a year.

A second case, McL-175 (Fig. 3) shows a similar "choppy" pattern in one part of the brain. The history is more obscure except that the onset was sudden and deterioration started at once, then her condition became stationary and has remained essentially unchanged for 20 years. Skull plates of this patient taken the same month as the brain record showed hyperostoses, extensive calcification of the falx, and very deep Pacchionian depressions in the parietal regions. The first patient cited had a history of severe trauma. The second patient cited had no history of trauma. Both brains, however, show similar "choppy" records.

The third patient, McL-174 (Fig. 3) has remained essentially in the same condition for years. His pneumoencephalogram showed some displacement of ventricles to the right, probably due to rotation, and "no definite disease was demonstrated." This pneumoencephalogram is inconclusive, but it gives no clear explanation for the "choppy" appearance of the electrical record.

Other authors have published reproductions or descriptions of records which apparently belong to our "choppy" classification and which were obtained in cases of known cerebral lesions, such as

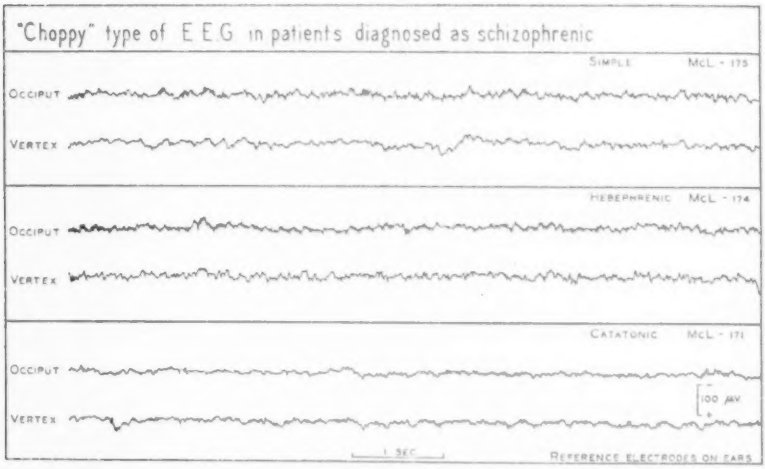


FIG. 3.

general paresis,[1] cerebral softening due to vascular thrombosis,[10, 6] porencephaly,[6] and cerebral tumor.[14] The importance of the "choppy" record as a possible clue to unsuspected lesions is indicated by the fact that in many of the instances cited, as in one of our own (McL-171) the "choppy" type of record was obtained only or most clearly over the region where the lesion was known to be localized. The prominent irregular fast waves of variable voltage apparently represent an irritative but unsynchronized type of cortical activity.

The "choppy" type of record has apparently been recorded from schizophrenic patients by Jasper, Fitzpatrick and Solomon,[8] who speak of "continuous high-frequency" records. They group such records with other "low-alpha" records which they term "flat."

It is true that a fair percentage of schizophrenic patients yield rather low-voltage records with few or poorly developed alpha waves,[9, 11, 12, 8, 3] but *mere* absence of alpha waves is perfectly normal.[2] We believe that it is important to distinguish within the "low-alpha" group the *abnormal* "choppy" record with its irregular fast waves of at least 5 or 10 μV. The "choppiness" is an abnormal disturbance which may appear in a high-alpha type as well as a low-alpha type of individual. In McL-175 there are parts of the brain showing enough alpha rhythm in her total record to exclude her from the "low-alpha" or "flat" classification.

Group II is the largest group and the patterns give evidence that many schizophrenics have a disorder of brain function which is the same as that of people suffering from convulsive disorders (*cf.* also Gibbs, Gibbs and Lennox,[5] and Jasper, Fitzpatrick and Solomon[8]). Records of the physiological behavior of the brain seem to show that schizophrenia and epilepsy are by no means mutually exclusive. The difference appears to be one of degree, in that the schizophrenic patient having such a pattern may suffer from episodes of excessive motor activity and confusion rather than major convulsions. The presence of dysrhythmias in the EEG does not imply that the patient is not schizophrenic, but it does imply that he may also have some variant of the convulsive disorders coexisting with his schizophrenia.

In the histories of these patients there is very frequently the syndrome so common in epileptic patients, namely, bed-wetting, night terrors, temper tantrums, periods of destructive, unreasonable, sulky, violent or excitable behavior in childhood and later. It is interesting that the patients whose brain records clearly fall in Group II are either diagnosed as "catatonic" schizophrenia, or else have a history of catatonic stupors and excitements in the hospital. They are the patients whose behavior on the hospital ward is unpredictable and who have catatonic episodes which psychotherapy, insulin and metrazol have failed to eliminate.

Responses of Schizophrenics and Their EEG's to Treatment.

This brings up a consideration of the responses of the three groups of schizophrenics to treatment. The treatments at McLean Hospital and Metropolitan State Hospital have been metrazol,

insulin, a combination of the two, and the "total-push" therapy. The human factor of attention is a constant factor in all these treatments at McLean Hospital. The patients have extra attention, special nurses upon whom much responsibility rests during and between treatments, and constant care from the medical staff.

The "total-push" treatment as used at McLean Hospital is very intensive. Personal nurses act as companions to the patients and bring them into natural associations of a constructive type and happy social relationship with others. The patients under this treatment are a good control group for the others under metrazol and insulin therapy. There are 16 patients under pharmacological treatment upon whom EEG studies are being made. All three types of EEG, the normal, dysrhythmic and "choppy," are well represented in the patients under each form of therapy. Records have been made before, during and after shock therapy. Follow-ups a month, six months and a year later have been made on almost all.

The EEG patterns of these patients, if normal before treatment, became abnormal during and following the termination of the pharmacological shock.[4] Patterns abnormal before treatment remained abnormal, and the abnormalities increased during and following termination of treatments.

Three patients whose EEG's are classified under Group III and are suggestive of lesions of the brain reacted so atypically that treatment had to be discontinued. The EEG during a treatment did not show the same sequence or quality of changes [4] as the EEG's in Group I, whose patterns were essentially normal.

Many of the patients on "total-push" therapy who had not received pharmacological treatment previously have improved markedly both physically and in mental alertness. Their schizophrenic symptoms have not disappeared, nor have their personalities changed, but as human beings they have been stepped up to a higher level of coordinated activity and awareness of their surroundings. Their EEG's have not become abnormal; they remain the same.

The brain pattern changes only when the physiology of the brain is modified. If damage is done, the EEG becomes abnormal. In view of these findings, this new approach to the problem of schizophrenia seems to be worth while. If all mental patients on

admission to the hospital could be thoroughly examined, physically and medically, and given a complete thorough neurological examination (which would include skull plates, pneumoencephalograms, electroencephalographic studies), as well as a thorough inquiry into their heredity and psychiatric history, one would be in a better position to evaluate the relationships of these many factors to one another. Autopsies on every patient dying are increasingly essential for the sake of the living patients.

Discussion.

This paper is only a general outline of work still in progress, and it is therefore too early to attempt elaborate discussion or to draw general conclusions. Three obvious suggestions emerge, nevertheless.

First, if the EEG shows patterns suggestive of the convulsive disorders in some schizophrenic patients it would seem rational to study this group separately from the clinical point of view and to institute treatment appropriate to epileptics. It is well known that many epileptics who are kept busy mentally and physically are less likely to have seizures. The "total-push" treatment would therefore be indicated for this group. Also the drugs which have proved useful in the convulsive disorders could be tried and the results studied both clinically and electroencephalographically.

Second, since the EEG becomes more abnormal following shock therapy and remains unchanged during "total-push," the clinical results of the two methods should be carefully compared, and unless shock therapy is clearly more successful, preference should be given to the method which shows least evidence of temporary or possibly permanent damage to the brain, at least until further research throws some light on the mechanisms involved in schizophrenia.

Third, the EEG may prove to be of practical value in identifying certain types of cases, as those of Group III and perhaps also Group II, for which shock therapy might be definitely contraindicated.

Summary.

A study of 132 schizophrenic patients at Metropolitan State Hospital and McLean Hospital is in progress.

The EEG's of these schizophrenics fall into three groups:

Group I: Essentially *normal.*

Group II: Dysrhythmic type, which is indistinguishable from EEG's of individuals known to have convulsive disorders.

Group III: "Choppy" type, which suggests the possibility of a pathological condition in the brain.

Patients in Group II frequently present in their histories the same syndrome as found in epileptics. These patients are either diagnosed as "catatonic schizophrenia" or give a history on the hospital ward of catatonic stupors and excitements.

BIBLIOGRAPHY.

1. Berger, H.: Über das Elektrenkephalogramm des Menschen. V. Arch. f. Psychiat., 98: 231-254, 1933.
2. Davis, H., and Davis, P. A.: Action potentials of the brain in normal persons and in normal states of cerebral activity. Arch. Neurol. and Psychiat., 36: 1214-1224, 1936.
3. Davis, P. A., and Davis, H.: The electroencephalograms of psychotic patients. Am. J. Psychiat., 95: 1007-1025, 1939.
4. Davis, P. A., and Sulzbach, W.: Changes in the electroencephalogram during metrazol therapy. Arch. Neurol. and Psychiat. (in press).
5. Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: The likeness of the cortical dysrhythmias of schizophrenia and psychomotor epilepsy. Am. J. Psychiat., 95: 255-269, 1938.
6. Janzen, R., and Kornmüller, A. E.: Örtliche Unterschiede hirnbioelektrische Erscheinungen von kranken Menschen bei Ableitung durch die Kopfschwarte. Arch. f. Psychiat., 109: 247-263, 1939.
7. Jasper, H. H., and Andrews, H. L.: Electro-encephalography: III. Normal differentiation of occipital and precentral regions in man. Arch. Neurol. and Psychiat., 39: 96-115, 1938.
8. Jasper, H. H., Fitzpatrick, C. P., and Solomon, P.: Analogies and opposites in schizophrenia and epilepsy. Am. J. Psychiat., 95: 835-851, 1939.
9. Lemere, F.: The significance of individual differences in the Berger rhythm. Brain, 59: 366-375, 1936.
10. Lemere, F.: Berger's α rhythm in organic lesions of the brain. Brain, 60: 118-125, 1937.
11. Lemere, F.: Effects on electroencephalogram of various agents used in treating schizophrenia. J. Neurophysiol., 1: 590-595, 1938.
12. Lemere, F.: Electroencephalography. Psychiat. Quart., 13: 5-15, 1939.
13. Rubin, M. A.: The distribution of the alpha rhythm over the cerebral cortex of normal man. J. Neurophysiol., 1: 313-323, 1938.
14. Schwab, R. S.: The electroencephalogram in a general hospital. A three-year report. J. Am. Med. Assoc. (in press).

ELECTROENCEPHALOGRAPHY IN THE PSYCHOSES.

Localization of Cerebral Atrophy.*

By MORTON A. RUBIN, Ph. D.

Electroencephalography has been used with much success in the localization of cerebral lesions (Walter,[1] Williams and Gibbs,[2] and Case and Bucy[3]). The criterion employed by these authors is the presence of "pathologic" delta waves, confined to the region of the lesion. From a survey of the literature, it appeared that cerebral atrophy was rarely described in association with delta waves. Preliminary investigation (Rubin[4]) still further demonstrated the general absence of foci of delta-wave activity in cases of cerebral atrophy. Lemere[5] has reported a difference in the amount of "normal" 10-per-second electrical activity (alpha rhythm) from an atrophied region of one cerebral hemisphere as compared with the same region in the unaffected hemisphere. However, considerable differences in amount of alpha activity may exist in comparable regions of the two hemispheres of normal persons (Rubin[6]). Furthermore, the amount of alpha activity varies to some extent (Rubin[7]), so that simultaneous recording from two regions must be employed if a genuine comparison is to be made. Lemere did not take simultaneous records from the affected and unaffected regions.

It seems reasonable that an atrophied region of the cerebrum should exhibit a different amount of alpha activity relative to neighboring intact tissue in the same hemisphere than that found in the corresponding, but not atrophied, region in the opposite hemisphere. Atrophied tissue must contain fewer neural elements capable of autonomous electrical activity, and it probably does not react to other nervous influences playing upon it in the same manner as does normal tissue. It is with the data obtained by a method based on this reasoning that the present report is concerned.

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METHODS.

Electrodes.—The electrodes consisted of flattened pellets of lead solder approximately 2 mm. in diameter. A small amount of Sanborn electrode paste was applied to the scalp directly underneath the electrode to insure good electrical contact, and the electrode itself was fixed to the scalp with a drop of collodion.

Monopolar leads were employed, *i.e.*, potential differences between an active (grid) electrode, which was placed over cortical tissue, and an earthed (reference) electrode placed on the mastoid processes were recorded.

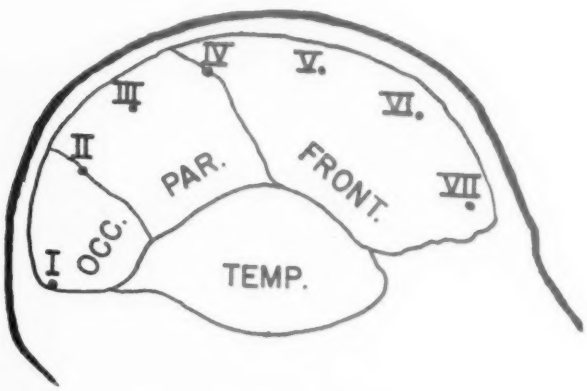


FIG. 1.—See text for description.

Electrode Placement.—The electrodes were arranged in two rows from back to front of the head over both cerebral hemispheres about 3 cm. to either side of the midline. Seven electrodes were fixed to the scalp on each side of the head and potentials from the corresponding leads on both sides were recorded simultaneously.

Fig. 1 is a diagrammatic representation of one cerebral hemisphere. Roughly, electrode I is over the posterior tip of the occipital lobe; II, over the occipito-parietal fissure; III, mid-parietal; IV, central fissure (fissure of Rolando); V, VI and VII divide the frontal lobe into three approximately equal parts.

Apparatus.—Two independent, well-matched amplifiers and ink-writing undulators built by Mr. A. M. Grass were used to record the potential changes. In addition, two filter systems tuned to 10 cycles per second were used to make analysis easier. The amplifi-

cation was kept constant throughout the experiments, a 5 mm. excursion of the writing pen corresponding to 50 microvolts.

Procedure.—The subject reclined, with eyes closed, on a bed in a semi-darkened, quiet, shielded room. As a precaution against his falling asleep, he was asked to open his eyes for a minute or so at frequent intervals during the experiment. The recording took one-half to three-quarters of an hour in all.

Two meters of filtered and two meters of unfiltered record were obtained from the various electrode combinations, and the distribution curves of the amount of alpha rhythm over the head were plotted. The amount of alpha activity is quantitatively given by the *per cent time alpha.* At least 3 consecutive alpha waves with amplitudes of 7 microvolts or more were arbitrarily regarded as constituting an alpha rhythm. The percentage of 2 consecutive meters of record occupied by the alpha rhythm gave the *per cent time alpha.*

Complete data were obtained on two or more different occasions on 14 schizophrenics, two manic-depressives and on one patient with a traumatic psychosis. The results were essentially the same in the individual cases with each repetition.

RESULTS.

A set of distribution curves of *per cent time alpha* characteristic for schizophrenics with no detectable cerebral pathology is given in Fig. 2. The curves for both cerebral hemispheres run quite parallel. In other words, each pair of simultaneously recorded points, representing the amount of alpha activity of corresponding regions in the two hemispheres, increases or decreases in value relative to the preceding pair of points. The absolute values of a pair of points may differ, but they change in the same direction as one records anteriorly along the head. The same relationships have been observed in non-psychotic individuals (Rubin [6]).

In contrast to the similarity of the distribution of alpha-wave activity from the two sides of the head in a case of non-atrophy, there are unmistakable differences in the curves of another patient which are reproduced in Fig. 3. There is a marked difference in the curves in the region between electrodes II and VI. On the basis of this dissimilarity, a diagnosis of atrophy involving the parietal region and extending anteriorly through the posterior third of the

frontal area was made. This localization was subsequently confirmed by pneumoencephalography.

The 14 schizophrenic patients studied were, in the main, markedly deteriorated. They had been selected by Dr. A. Angyal for another investigation on the basis of certain subjective symptoms which could be observed in them (Angyal [8]). In brief, the symptoms were, so-called loss of ego-reference, experience of motor

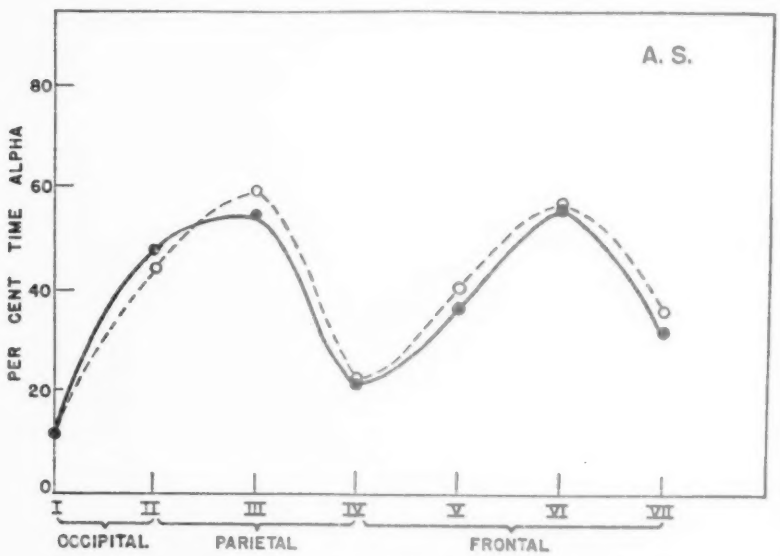


FIG. 2.—Distribution curves of *per cent time alpha* over the two cerebral hemispheres of schizophrenic patient A. S., illustrating the similarity in shape of the two curves. Open circles for left, and solid circles for right hemisphere.

influences, disturbances of the body image, distractibility, indecision and lack of initiative. Very few of the patients exhibited neurologic signs indicative of central nervous pathology, and the present investigator was not informed as to the nature of the symptoms exhibited by the patients until the electroencephalographic examination and diagnosis were completed. Pneumoencephalograms were then taken and the electroencephalographic findings compared with them. Such a procedure eliminated the possibility of bias entering into the interpretation of the electrograms.

The following are the relevant data on 9 schizophrenics, on whom pneumoencephalographic confirmations were subsequently made available.

PATIENT H. A.—White male, 31 years of age.

Type of Schizophrenia.—Hebephrenic; five years' duration.

History of Head Trauma.—Head injury at age of 17.

Neurological Examination.—Pathological findings were past-pointing, slight polyesthesia, and tendency to irradiation of tactile sensation.

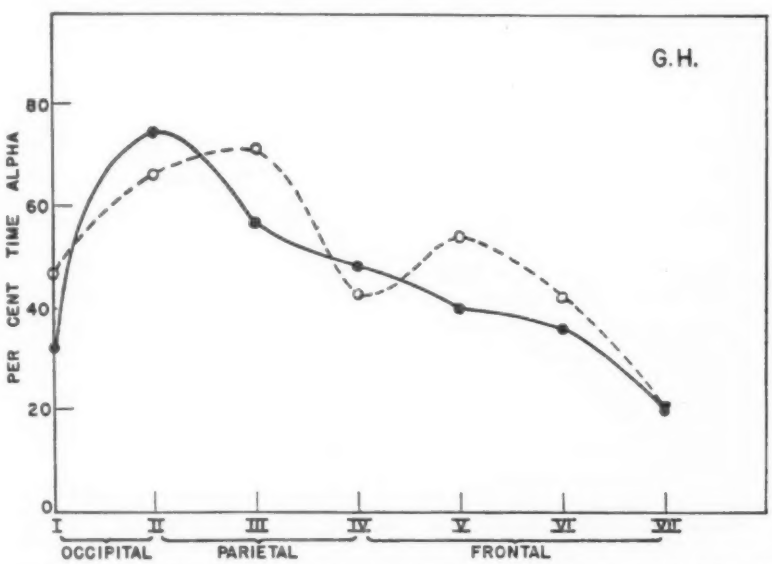


FIG. 3.—Distribution curves of *per cent time alpha* over the cerebral hemispheres of a schizophrenic patient with cerebral atrophy, illustrating dissimilarities in shape of the two curves in the region between electrodes II and V. Left hemisphere represented by the open-circle curve, and the right by the solid-circle curve.

Pneumoencephalogram.—Generalized cerebral atrophy, with cortical atrophy most marked in posterior part of parietal lobes; ventricular enlargement.

Electroencephalogram.—Dissimilarity of *per cent time alpha* curves extending from posterior parietal to anterior frontal region; occasional delta-wave discharges.

PATIENT C. B.—White male, 24 years of age.

Type of Schizophrenia.—Unclassified, early; six months' duration.

History of Head Trauma.—Trauma followed by two hours of unconsciousness.

Neurological Examination.—Pathological findings consisted of past-pointing, gross errors in localization of tactual stimuli, polyesthesia, swaying (when eyes closed), and positive Barré sign on left side.

Pneumoencephalogram.—Moderate deep cerebral atrophy, characteristic of post-traumatic case; moderate equilateral ventricular atrophy, including third ventricle; no evidence of cortical atrophy.

Electroencephalogram.—Dissimilarity of curves in posterior half of frontal region.

PATIENT A. B.—White male, 31 years of age.

Type of Schizophrenia.—Hebephrenic; nine years' duration.

History of Head Trauma.—None.

Neurological Examination.—No pathological findings.

Pneumoencephalogram.—Diffuse cortical atrophy, most predominant in right parietal lobe.

Electroencephalogram.—Dissimilarity of curves in frontal region; also delta waves.

PATIENT I. B.—White male, 35 years of age.

Type of Schizophrenia.—Paranoid; two months' duration.

History of Head Trauma.—Received severe face injury at age of 12.

Neurological Examination.—Pathological findings were past-pointing, swaying (when eyes closed) and faulty two-point discrimination.

Pneumoencephalogram.—Questionable, slight parietal atrophy.

Electroencephalogram.—Normal.

PATIENT M. C.—White male, 37 years of age.

Type of Schizophrenia.—Late indeterminate; duration of illness 19 years.

History of Head Trauma.—None.

Neurological Examination.—No pathological findings.

Pneumoencephalogram.—Normal.

Electroencephalogram.—Normal.

PATIENT A. D.—White male, 33 years of age.

Type of Schizophrenia.—Hebephrenic; nine years' duration.

History of Head Trauma.—None.

Neurological Examination.—No pathological findings.

Pneumoencephalogram.—Normal.

Electroencephalogram.—Normal.

PATIENT G. H.—White male, 35 years of age.

Type of Schizophrenia.—Paranoid; three years' duration.

History of Head Trauma.—Accidentally shot in right eye and forehead.

Neurological Examination.—No pathological findings.

Pneumoencephalogram.—Gross atrophy of cerebral cortex, extending from frontal to occipital region; most predominant in posterior half of parietal lobes on both sides.

Electroencephalogram.—Dissimilarity of curves from the occipito-parietal fissure through posterior third of frontal areas.

PATIENT B. M.—White male, 25 years of age.
Type of Schizophrenia.—Hebephrenic; 10 years' duration.
History of Head Trauma.—None.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Doubtful evidence of very slight atrophy in left hemisphere; no cortical atrophy.
Electroencephalogram.—Normal.

PATIENT T. S.—White male, 32 years of age.
Type of Schizophrenia.—Catatonic; 1½ years' duration.
History of Head Trauma.—None.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Normal.
Electroencephalogram.—Normal.

Pneumoencephalograms have not yet been taken on the remaining five schizophrenic patients. Electroencephalographically four of the five do not show dissimilarities in their *per cent time alpha* curves, and one does. The five patients were:

PATIENT J. B.—White male, 29 years of age.
Type of Schizophrenia.—Hebephrenic; four years' duration.
History of Head Trauma.—None.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Not taken.
Electroencephalogram.—Normal.

PATIENT A. L.—White male, 23 years of age.
Type of Schizophrenia.—Hebephrenic; one year's duration.
History of Head Trauma.—Fell and knocked unconscious at age of 20.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Not taken.
Electroencephalogram.—Normal.

PATIENT E. L.—White male, 29 years of age.
Type of Schizophrenia.—Unclassified; 1 year's duration.
History of Head Trauma.—None.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Not taken.
Electroencephalogram.—Dissimilarity of distribution curves in posterior frontal region.

PATIENT H. M.—White male, 22 years of age.
Type of Schizophrenia.—Hebephrenic; six months' duration.
History of Head Trauma.—None.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Not taken.
Electroencephalogram.—Normal.

PATIENT A. S.—White male, 31 years of age.
Type of Schizophrenia.—Paranoid; six months' duration.
History of Head Trauma.—None.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Not taken.
Electroencephalogram.—Normal.

The following two manic-depressive patients were examined electroencephalographically:

PATIENT E. D.—White male, 38 years of age.
Psychosis.—Manic-depressive, depressive type; six months' duration.
History of Head Trauma.—Automobile accident six months previous, followed by depression.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Diffuse cortical atrophy.
Electroencephalogram.—Dissimilarity of *per cent time alpha* curves in the parietal region.

PATIENT A. LU.—White male, 24 years of age.
Psychosis.—Manic-depressive, manic type; seven years' duration.
History of Head Trauma.—Depressed fracture of skull in right frontal region at age of 5.
Neurological Examination.—No pathological findings.
Pneumoencephalogram.—Cortical atrophy in right frontal lobe; enlargement of right lateral ventricle.
Electroencephalogram.—Dissimilarity in frontal region; also activity from forehead of type found in "psychomotor" epilepsy (see Fig. 4).

From the preceding cases, it appears that dissimilarities of the *per cent time alpha* distribution curves are associated with cerebral atrophy. All six cases exhibiting dissimilarity in their curves were shown pneumoencephalographically to have cerebral atrophy. The absence of atrophy was confirmed by air injections in four of the five cases with parallel distribution curves. The one exception was a patient whose pneumoencephalogram was interpreted as showing very doubtful evidence of atrophy. His *per cent time alpha* distribution curves were parallel, indicating the absence of pathology.

In addition to evidence of cerebral atrophy, electrical discharges of the type associated with a "psychomotor" equivalent of an epileptic attack (Gibbs, Gibbs and Lennox [9]) were recorded from the forehead, bilaterally, of Patient A. Lu. This psychomotor seizure is shown in Fig. 4.

Patient A. Lu. had no past history of seizures. However, during the 20 seconds or so that the "electrical seizure" lasted, the patient did not respond to a command to open his eyes. Shortly following this period he opened his eyes immediately when that request was made.

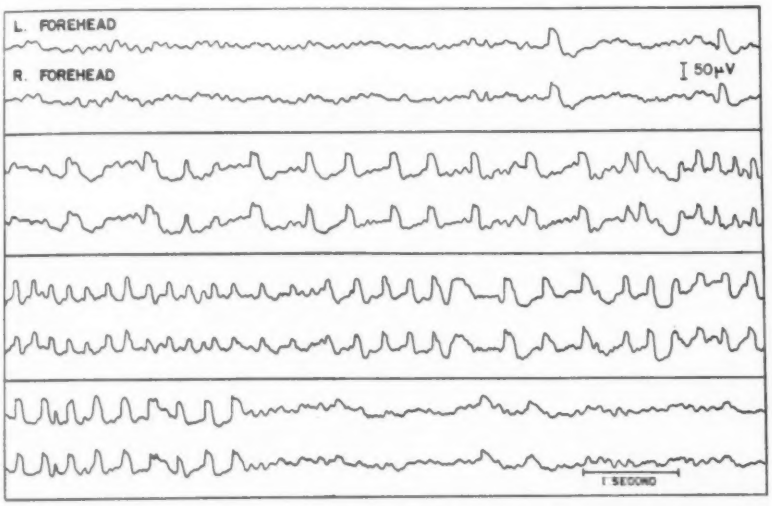


FIG. 4.—A continuous record from the forehead of a manic-depressive patient (A. Lu.) before, during and after a "Psychomotor" seizure. The seizure starts at the end of the first (top) strip of record and is over after the first third of the last (bottom) strip. Amplification constant throughout.

One patient with a traumatic psychosis was also examined.

PATIENT H. S.—White male, 39 years of age.

Psychosis.—Traumatic psychosis; one year's duration.

History of Head Trauma.—Fell from ladder at age of 36.

Neurological Examination.—Impairment of joint-position sense on left side.

Pneumoencephalogram.—Generalized, moderate deep cerebral atrophy; atrophy more marked in cortex, especially in parietal regions near vertex.

Electroencephalogram.—No alpha activity from any region of the head.

It was not possible to diagnose cerebral pathology in Patient H. S., since no alpha activity was found anywhere over the head. Some faster activity at 18-20 per second was observed under the frontal leads. The patient was allowed to fall asleep, and electro-

grams were obtained for one-half an hour or so. Almost from the start of sleep, the patient snored deeply and appeared to be fast asleep. It was rather difficult to arouse him at this time. Fig. 5 is an illustrative sample of record from the vertex of this patient when awake and during sleep. The pattern of the sleep record corresponds to the "C stage" of Loomis, Harvey and Hobart [10] and the "alpha+delta+14 per second" stage of Blake, Gerard and

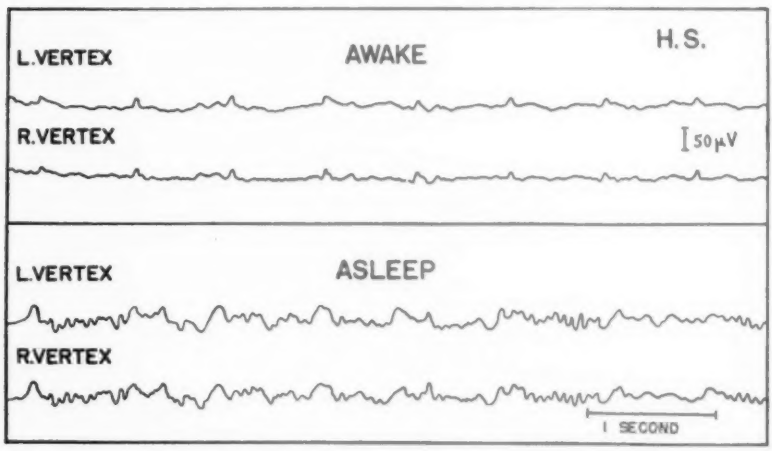


FIG. 5.—Sample tracings from the vertex of a patient (A. S.) with extensive cerebral atrophy, illustrating the appearance of characteristic potential changes during sleep in spite of the almost complete absence of electrical activity when awake. The larger potential changes in the "awake" record, occurring about once every three-quarters of a second, are artifacts due to the heart beat. The amplification is the same in both upper and lower records.

Kleitman.[11] Normal individuals who exhibit no alpha activity in the waking state also show this typical sleep pattern (Davis *et al.*[12]). The interesting point in this case is that a patient with extensive marked cerebral atrophy, both deep and superficial, shows brain potential changes typical of those observed in the sleep of individuals with a presumably normal cerebrum.

DISCUSSION.

The electroencephalographic method of localizing cortical atrophy has a distinct advantage over other methods in that it measures

activity without itself altering the condition of the patient or of his central nervous system. The method is based essentially on the detection of disturbances in the "normal" integrated electrical activity of the brain, which an area of atrophy might cause. It should be emphasized that it is not the crossing of the *per cent time alpha* curves that is taken as the localizing sign of atrophy. In itself crossing may not be employed as a criterion, since it occurs in patients without cerebral atrophy. In such cases of crossing, the curves both proceed in the same direction, and are due to a sharper increase or decrease, as the case may be, in one hemisphere than in the other. Atrophy may or may not be accompanied by crossing. *The real criterion for atrophy is the occurrence of marked differences in the change of slope of the curves between two successive pairs of electrodes* (*i. e.,* the curve rises sharply in one hemisphere and falls in the other).

A limitation of the electroencephalographic method at the present time is the fact that one cannot decide whether the atrophy is unilateral or bilateral. To do this, it would be necessary to know what shape of *per cent time alpha* curve is "normal." To know this may not be possible, even if a large number of normal individuals were studied, since there may be an almost unlimited number of "normal" curves. This has already been found true for the distribution curves for the frontal lobes (Rubin [6]). The other limitation of the method is that it cannot be used for individuals with extremely low or entirely absent alpha activity.

An interesting point may be raised in connection with "normal" and "pathologic" potential changes. We have seen that it is possible to localize brain atrophy by alterations in the distribution of so-called normal (alpha-wave) electrical activity. Slow (delta-wave) activity is used as the criterion for the presence of brain tumors, and yet it is characteristic of everyday sleep. It would seem, then, inadvisable to call any potential change pathologic unless it occurs in a known instance of pathology. It is for this reason, primarily, that we feel that the electroencephalogram should not be employed as the sole test for cerebral pathology.

The present data revealed no consistent relationship between the presence or absence of cerebral atrophy and type of psychosis, duration of illness, history of head trauma or neurologic symptoms of central nervous pathology. Routine electroencephalographic

examination for cerebral atrophy should be of value in uncovering pathology, not easily detected by ordinary examination, which may aid in the evaluation of some aspects of psychotic behavior and in prognosis.

SUMMARY.

The bilateral distribution of the amount of alpha-wave activity (*per cent time alpha*) from the cerebrum of 14 schizophrenics, two manic-depressives and one case of traumatic psychosis was determined.

From differences in the distribution of *per cent time alpha* over the two cerebral hemispheres, it was found possible to localize cerebral atrophy. The electroencephalographic diagnoses were confirmed pneumoencephalographically in eight of the nine schizophrenics and in both manic-depressive patients. Air injections have not yet been done on the other five schizophrenics.

In two of the schizophrenics with cerebral atrophy delta-wave discharges were occasionally observed. Epileptiform discharges were recorded from the forehead of one of the manic-depressive patients. The patient with traumatic psychosis had no detectable alpha rhythm, so that it was not possible to localize the extensive, marked cerebral atrophy, both deep and superficial, which were subsequently found pneumoencephalographically. However, potential changes typical of normal sleep were obtained while this patient was asleep.

BIBLIOGRAPHY.

1. Walter, W. G.: The electroencephalogram in cases of cerebral tumor. Proc. Roy. Soc. Med., 30: 579-598, 1937.
2. Williams, D., and Gibbs, F. A.: The localization of intracranial lesions by electroencephalography. N. E. J. Med., 218: 998-1002, 1938.
3. Case, T. J., and Bucy, P. C.: Localization of cerebral lesions by electroencephalography. J. Neurophysiol., 1: 245-261, 1938.
4. Rubin, M. A.: Electroencephalographic localization of atrophy in the cerebral cortex of man. Proc. Soc. Exp. Biol. Med., 40: 153-154, 1939.
5. Lemere, F.: Berger's α rhythm in organic lesions of the brain. Brain, 60: 118-125, 1937.
6. Rubin, M. A.: The distribution of the alpha rhythm over the cerebral cortex of normal man. J. Neurophysiol., 1: 313-323, 1938.

7. Rubin, M. A.: A variability study of the normal and schizophrenic occipital alpha rhythm. I. J. Psychol., 6: 325-334, 1938.
8. Angyal, A.: Disturbances of activity in a case of schizophrenia. Arch. Neurol. and Psychiat., 38: 1047-1054, 1937.
9. Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: Epilepsy; a paroxysmal cerebral dysrhythmia. Brain, 60: 377-388, 1937.
10. Loomis, A. L., Harvey, E. M., and Hobart, G.: Cerebral states during sleep, as studied by human brain potentials. J. Exp. Psychol., 21: 127-144, 1937.
11. Blake, H., Gerard, R. W., and Kleitman, N.: Factors influencinig brain potentials during sleep. J. Neurophysiol., 2: 48-60, 1939.
12. Davis, H., Davis, P. A., Loomis, A. L., Harvey, E. N., and Hobart, G.: Human brain potentials during the onset of sleep. J. Neurophysiol., 1: 24-38, 1938.

DISCUSSION.

DR. WILLIAM G. LENNOX (Boston, Mass.).—I thing Dr. Rubin deserves a great deal of credit for being the first one to make a careful study of the alpha rhythm in psychotic cases, comparing various localities in one hemisphere and the homologous areas of the two hemispheres. That certainly is work which needed to be done.

My criticism, I think, would be mainly with his use of the word "localization" in the title. It is true that there are differences shown in the top when compared with the front and back of the head, but as he says, there are no lateralizing values in the test. I wonder a little in saying that, because offhand you would think that the higher the percentage of alpha rhythm, which is the normal rhythm, the more normal that side would be, and in comparing the two sides, you would give the credit to the one with the higher alpha rhythm for being the normal. Of course, you might say that there is a good deal of variety in the two sides in normal individuals, so that that might not hold.

I think a more serious criticism is in the suggestion that these curves go along with the atrophy. He didn't show us the x-ray films on which the diagnosis of atrophy was based. Of course these cortical potentials are measuring the cortex only. It would show, perhaps, evidence of cortical atrophy, but not of deep atrophy, which a pneumoencephalogram is likely to demonstrate.

It isn't necessarily air over the cortex. I don't know how he can say this was cortical atrophy in comparing the cortical potentials with what he sees in the pneumoencephalogram.

A third point on localization is this business of talking about atrophied cells, because those cells are dead cells and are not discharging, and therefore he is speaking about active cells contrasted with dead cells, which are presumably in that same region, but there is no demonstration that this is true. They may all be dead in the atrophied region and not active at all, so I would be much happier if he would make this simply as a study of the electrical potentials which are to be found in various areas of the cortex of patients with schizophrenia.

Certainly there are things to be seen besides the alpha rhythm. He shows a patient with the upside down psychic seizure type of wave. That is not at all strange, because many schizophrenics do show types of waves indistinguishable from those shown by epileptic patients subject to psychic seizures.

I am sure we all need more of these studies.

DR. OSKAR DIETHELM (New York, N. Y.).—I would like to ask a few questions. First, it seems to me it is important in the presentation of such material that you give the age of the patients; otherwise I do not see how you can evaluate schizophrenics.

The second question I would like to ask, is with regard to psychopathology. The psychopathologic findings were given in a language with which at least I am not familiar; it seems to me we should have the privilege of having a clear picture of the patient, otherwise we come down to laboratory studies which do not allow us to really evaluate what schizophrenia means.

DR. IRA C. NICHOLS (Providence, R. I.).—I want to ask Dr. Rubin a couple of questions. He said in atrophy these alpha waves do not appear. How do you distinguish that from lack of conduction, say, from the center, or complete cancelling out? The elements that give the alpha rhythm cancel out, the potentials equal each other, and you get a straight line.

The other question is about pneumo-encephalography which check with the changes definitely and readily interpretable. I have had some experience with pneumo-encephalography, and it seems to me a rather indefinite method. Sometimes the air will go one way and sometimes it will go in another direction, and it is not under your control. I wonder if you could tell us a few details about that.

A third question is, if there is atrophy, why haven't the pathologists found it in definite cases, perhaps not in the weighing of the brain, but looking at sections? Wouldn't this atrophy appear as a definite falling out of cells, as is described in many conditions?

DR. RUBIN.—I am really indebted for the criticism that I have received.

I probably should have made it clear, in answer to Dr. Lennox's criticism as to the title of the paper, that the title has been changed since he received a copy. Although the "localization" is still in, the "cortical" part is left out; it is "cerebral."

I agree with him that localization is only front and back of the head within a hemisphere, and not laterally. The agreement there within the limits of reading an air injection in the pneumo-encephalogram are really within a fraction of a centimeter.

The pneumo-encephalograms were taken at the Worcester State Hospital and were read by Dr. Van Storch of the Boston City Hospital.

Another point that Dr. Lennox referred to was the dead cells, the atrophied cells, being unable to produce autonomous electrical activity, and that is well taken. I don't know how you could decide whether the activity

should be less or greater. Theoretically you would say there are fewer cells, therefore less activity. However, dead tissue in many instances is found to be a good conducting medium and would conduct through the activity from adjacent normal regions. Lemere has found that the activity might be higher or lower in the atrophied regions. The main criterion here is that if there are cells incapable of producing activity on their own, any activity recorded from that region is not the same as it would be, compared especially with the other side, if those cells were capable of activity. Therefore it might be higher or lower, but not in the same direction as the other side, and that dissimilarity of the curves, or the differences in slope, have been taken as the criterion.

I certainly agree that more study is necessary.

In answer to Dr. Diethelm, the ages ranged from 22 to 39 years, with a mean of 30.

Dr. Nichols, if I understand your first question correctly about the cancelation out and obtaining a flat base line, that wouldn't be very probable with the method used here. We purposely used monopolar leads; that is, an active lead and an earthed lead. If you use bipolar leads, where both electrodes are over active tissue, you might readily get cancelation and a flat base line.

The pneumo-encephalographic changes were just as definite or marked as they could be. All the cases in which I said there was agreement were cases of very marked, extensive atrophy.

Pathologists have found atrophy, and I think the reason why atrophy hasn't been found more in the past is that methods were not adequately developed for detecting atrophy histologically.

A FIVE TO TEN YEAR FOLLOW-UP STUDY OF 641 SCHIZOPHRENIC CASES.*

By CHARLES RUPP, M. D.,

AND

ELIZABETH K. FLETCHER, M. Soc. Serv.,

Howard, R. I.

The possibility of inducing remissions in cases of schizophrenia by the use of pharmacological agents has aroused interest in and revealed the paucity of satisfactory data regarding the prognosis of the disorder in cases not receiving any so-called "specific" treatment. In an attempt to determine the prognostic outlook in a sufficiently large group of cases followed over a long period, a follow-up study of 641 cases clinically diagnosed schizophrenia was undertaken at the Rhode Island State Hospital for Mental Diseases with a four and a half to ten year follow-up. Recently similar studies have appeared,[1-5] based mainly on the more selected and acute case material of private and psychopathic hospitals; but few adequate follow-up studies of unselected state hospital cases are to be found in the literature.[6]

MATERIAL.

The 641 cases utilized represent essentially all the clinically diagnosed cases of schizophrenia admitted for the first time to the Rhode Island State Hospital for Mental Diseases between 1929 and 1934. A period of at least four and a half years had therefore elapsed between the time of admission and the time of this study. Adequate records were available in all cases. The diagnosis in each case had been made by the staff after presentation at the clinical conference. Most of the cases were seen again in conference prior to leaving the hospital and in a few instances in light of the subsequent course, the official diagnosis was changed. All such doubtful cases were excluded from the study. The criteria deter-

* From the Adolf Meyer Clinic and Social Service Department of the Rhode Island State Hospital for Mental Diseases, Howard, R. I.

mining the diagnosis were essentially those outlined in the Statistical Manual of the National Committee for Mental Hygiene.

The Rhode Island State Hospital for Mental Diseases at Howard, R. I., being the only state hospital in Rhode Island, receives an unselected group of mental patients. With hospital facilities for private and acute cases available at Butler Hospital and in the psychopathic department of the Charles V. Chapin Hospital, many of the cases later transferred from these hospitals represent a less favorable prognostic group. Acute and recent cases never hospitalized previously were, however, received from all parts of the state, although at times exigencies of overcrowding necessitated some delay in the admission of mildly ill cases whose immediate hospitalization was not imperative.

During the period 1929-1934, with a large hospital population and a limited staff, intensive therapy was impossible. All patients were admitted to a reception service where a complete anamnestic study, physical, neurological, psychiatric and routine laboratory examinations were made. More intensive laboratory tests and psychometric examinations were performed, if indicated. Hydrotherapy, physiotherapy, occupational, industrial and recreational therapy were available and frequent brief interviews were held with the physician. Patients continued to reside on the reception service four to six weeks, at the end of which those failing to show evidence of improvement and whose prognosis appeared poor, were transferred to the continued treatment service. All of the cases, therefore, received the average therapeutic opportunities available at a state hospital.

PROCEDURE.

An attempt was made to secure adequate information regarding the present whereabouts and condition (as of June 1, 1939) of all the 641 cases included in the study. For the patients still in the hospital current records were available, as well as the results of recent examinations by the staff physicians. Abstracts were obtained on patients transferred to other hospitals. Many of the patients outside the hospital continued to report at the extramural clinic at frequent intervals and examination by a physician was possible. For the remaining cases visits to the patient's last known address were made by experienced psychiatric social work-

ers who endeavored to see the patient and obtain from the family reliable data regarding the patient's physical and mental condition, work and social adjustment, as well as his freedom from psychotic symptoms. While it is realized that an examination by a physician in all cases was preferable, this, unfortunately, was not possible. In some instances, it was impossible to interview the patient directly because of inaccessibility, or unwillingness of the family to cooperate. In all such cases the information given by the relatives was checked, so far as possible, by information from physicians, hospitals, interested social agencies and neighbors. Cases in which the available information could not be considered reliable were classified under "no satisfactory follow-up information available." In 394 cases personal examination was possible. In 125 cases, including 28 in other hospitals, information only was available. 89 patients were dead and 33 were lost or satisfactory follow-up information was unavailable. On those cases where adequate follow-up data were available the duration of the follow-up period in 77 patients was from 4½ to 5 years; in 73, 5 to 6 years; in 81, 6 to 7 years; in 104, 7 to 8 years; in 87, 8 to 9 years; in 85, 9 to 10 years and in 12 slightly more than 10 years.

RESULTS.

Of the 641 patients, 340 were male and 301 were female. On June 1, 1939, their situation was as follows:

	Male.	Female.	Total.	Per cent.
In the community	85	91	176	27.5
In mental hospitals	180	163	343	53.5
Dead	52	37	89	13.9
No adequate information	23	10	33	5.1

Adequate follow-up data were, therefore, available for 608 patients. Since patients were frequently allowed to leave the hospital in the custody of relatives because they were considered harmless to themselves and others, rather than because of any essential degree of improvement in their condition; they were, therefore, classified as being either much improved, improved or unimproved.

The group classified as much improved includes those patients able to make a good social and work adjustment to the outside community at a level as high as before their illness and who are

entirely free of any residual symptoms, aside from a slight affective blunting. The improved group includes those patients able to make some sort of a social and work adjustment in the outside community, although the level of adjustment may be lower than before their illness, and whose psychotic symptoms are ameliorated, although still present. The unimproved group includes those patients showing no abatement of psychotic symptoms and who continue to require close supervision, either in or outside of a mental hospital.

The results in terms of such a classification follow:

	Male.	Female.	Total.	Per cent.
Much improved	15	25	40	6.6
Improved	46	47	93	15.3
Unimproved	204	182	386	63.5
Dead	52	37	89	14.6

It appears, therefore, that from 4½ to 10 years after admission 21.9 per cent of the patients were improved, 63.5 per cent were unimproved and 14.6 per cent were dead.

Further analysis of the cases with respect to the influence of diagnostic subtype, age of onset, duration of illness prior to hospitalization and duration of hospital residence was made.

(A) Subtype.

Classification of the original group on the basis of diagnostic subtype is shown in the following table:

	Male.	Female.	Total.	Per cent.
Simple	16	6	22	3.4
Hebephrenic	101	89	190	29.6
Catatonic	50	66	116	18.1
Paranoid	136	96	232	36.2
"Other types"	37	44	81	12.7

Excluding those cases for which adequate follow-up information was not available, the results are as follows:

	Simple.		Hebephrenic.		Catatonic.		Paranoid.		Others.	
	No.	%	No.	%	No.	%	No.	%	No.	%
Much improved	2	10.0	8	4.4	13	11.5	5	2.3	12	16.2
Improved	5	25.0	22	12.0	21	18.5	30	13.7	15	20.2
Unimproved	10	50.0	129	70.9	57	50.5	154	70.3	36	48.7
Dead	3	15.0	23	12.6	22	19.5	30	13.7	11	14.8

Therefore, 35 per cent of the simple, 16.0 per cent of the hebephrenic, 30 per cent of the catatonic, 16 per cent of the paranoid and 36.4 per cent of the "other" group were either improved or much improved. The greatest percentage benefited thus appears to be in the heterogeneous cases classified in the "other" group. It is of interest that in reviewing the individual cases classified in this group, many presented marked affective features, characteristic of the so-called schizo-affective psychoses. Hunt and Appel [8] have called attention to the better prognosis of such a group. Disregarding the simple group, because of the small number of cases, the next best results were obtained in the catatonic group, while the paranoid and hebephrenic group did poorest.

(B) Age of Onset.

Of the entire group of patients studied, the age of onset of the first detectable symptoms of mental illness was as follows:

Under 15	7 cases	45-54	42 cases
15-24	190 cases	Over 55	4 cases
25-34	224 cases	Unknown	28 cases
35-44	146 cases		

In the various subtypes, the maximum incidence of onset of symptoms occurred in the simple, hebephrenic and catatonic groups between 15 and 34, in the paranoid between 25 and 44, and in the "other" group between 15 and 44.

The status in relation to age of onset (excluding cases with inadequate follow-up information) follows:

	Under 15.		15-24.		25-34.		35-44.		Over 45.		Unknown.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Much improved ...	1	16.6	15	8.2	16	7.5	6	4.4	2	4.3	0	0
Improved	2	33.3	31	17.1	28	13.0	28	20.5	2	4.3	2	8.7
Unimproved	3	50.0	117	64.6	142	66.0	78	56.9	31	67.3	15	65.2
Dead	0	0.0	18	10.0	29	13.5	25	18.1	11	23.9	6	26.1

Leaving out the group under 15 and the group with the age of onset unknown, because of the small number of cases available, 25.3 per cent of the group between 15-24, 20.5 per cent of the group from 25 to 34, 24.9 per cent of the group from 35-44 and 8.6 per cent of the group over 45 were much improved or improved,

indicating that the age of onset of symptoms has little bearing on the prognosis, except that those cases having an onset of symptoms at age 45, or older, do less well than those where the onset occurs at a younger age.

(C) Duration of Symptoms.

The duration of symptoms prior to hospitalization for the entire group follows:

Less than 6 months	199	2 to 3 years	57
6 to 11 months	96	3 to 4 years	48
12 to 17 months	62	4 to 5 years	37
18 to 23 months	19	5 to 6 years	21
		6 to 7 years	15
		Over 7 years	60
		Unknown	27

Omitting the dead and those for whom adequate follow-up information was not available, the outcome in relation to the duration of symptoms prior to hospitalization for the group follows:

Duration of symptoms prior to hospitalization:

	Less than 6 months.		6 to 11 months.		12 to 23 months.		Over 2 years.	
	No.	%	No.	%	No.	%	No.	%
Much improved	20	11.5	9	11.5	5	8.2	6	2.9
Improved	40	23.0	14	17.9	12	19.6	27	13.1
Unimproved	114	65.5	55	70.6	44	72.1	173	83.9

The percentage of cases much improved or improved was 34.5 for those with symptoms of less than 6 months duration, 29.4 for those with symptoms of 6 to 11 months duration, 27.8 for those with symptoms of 12 to 23 months duration and only 16 for those with symptoms of more than 2 years duration. The outcome in this series, therefore, is slightly more favorable, the shorter the duration of symptoms prior to hospitalization, with a noticeable falling off in the percentage of cases benefited in those showing symptoms for 2 or more years before hospitalization.

(D) Period of Time During Which Hospitalization Was Necessary.

The duration of hospital residence for the original group of patients follows:

Less than 6 months	98 cases	4 to 5 years	67 cases
6 to 11 months	64 cases	5 to 6 years	67 cases
12 to 17 months	31 cases	6 to 7 years	61 cases
18 to 23 months	25 cases	7 to 8 years	64 years
2 to 4 years	26 cases	8 to 9 years	56 cases
3 to 4 years	32 cases	9 to 10 years	46 cases
		10 or more years	4 cases

The outcome correlated with the period of hospital residence, excluding those cases dead or for whom adequate follow-up information was not available follows:

Duration of Hospitalization.

	Less than 6 months.		6 months to 1 year.		1 to 2 years.		2 to 5 years.		Over 5 years.	
	No.	%	No.	%	No.	%	No.	%	No.	%
Much improved	14	20.3	9	20.5	11	26.8	5	5.3	1	0.4
Improved	38	55.0	20	45.5	17	41.4	8	8.6	10	3.8
Unimproved	17	24.6	15	34.0	13	31.7	80	86.0	261	95.7

Duration of required hospitalizations.	Per cent benefited.
6 months or less	75.3
6 months to 1 year	66.0
1 year to 2 years	68.2
2 years to 5 years	13.9
Over 5 years	4.2

It appears, therefore, that those patients requiring hospitalization for more than two years are much less likely to show improvement than those who require hospitalization for less than 2 years.

An analysis of the cases correlated with the subtype in relation to the duration of hospital residence follows:

DURATION OF HOSPITAL RESIDENCE.

	Less than 6 months.		6 months. to 1 year.		1 to 2 years.		2 to 5 years.		Over 5 years.	
	No.	%	No.	%	No.	%	No.	%	No.	%
Simple	8	36.3	1	4.5	3	13.6	3	13.6	7	31.8
Hebephrenic	24	12.6	17	8.9	12	6.3	39	20.5	98	52.6
Catatonic	16	13.8	16	13.8	11	9.5	26	22.4	47	40.5
Paranoid	29	12.5	21	8.7	18	7.8	44	19.0	120	51.9
"Other"	21	25.9	9	11.1	12	14.8	13	16.0	26	32.1

Of the simple group, 40.8 per cent are out of the hospital after 1 year.
Of the hebephrenic group, 21.5 per cent are out of the hospital after 1 year.
Of the catatonic group, 27.6 per cent are out of the hospital after 1 year.
Of the paranoid group, 21.7 per cent are out of the hospital after 1 year.
Of the "other" group, 37.0 per cent are out of the hospital after 1 year.

The simple and "other" groups have the highest percentage of patients out of the hospital at the end of one year; the hebephrenic and paranoid the least. The two latter groups show the highest percentage of cases remaining in the hospital after five years.

The following table shows the number of patients in the entire group in the community at increasing intervals after admission:

Time elapsing since admission.	Number of cases in community.	Increase.
6 months	68	68
1 year	110	42
1½ years	128	18
2 years	147	19
3 years	157	10
4 years	165	8
5 years	171	6
6 years	175	4
10 years	176	1

It is to be noted that the rate of increase in the number of patients in the community declines rapidly as the time elapsing since admission increases. While 68 patients are in the community 6 months after admission and 42 more in the next 6 months, only 6 patients left the hospital between the fourth and fifth year after admission and of those in the hospital for from 5 to 10 years only 5 left the hospital in the 5-year period.

READMISSIONS.

Excluding the dead and those cases for which no adequate follow-up information is available, 259 or approximately 50 per cent of the 519 remaining patients were out of the hospital at some time or other for a period of at least one month, while 260 have not left the hospital; of those leaving the hospital, 129 have not returned, 47 returned, but later were again able to leave, 83 returned and continued as permanent hospital residents. The relationship of the degree of improvement and return to the hospital is shown in the following table:

	No. of cases leaving hospital.	Per cent of total no. of cases.
Much improved	40	100
Improved	93	100
Unimproved	126	32.6
Total	259	50.0

	Not returning to hospital.	Per cent of cases leaving hospital.	Temporary return.		Permanent return.	
			No.	%	No.	%
Much improved	33	82.5	7	17.5	0	0
Improved	63	67.7	21	22.6	9	9.7
Unimproved	33	26.2	19	15.0	74	58.7
Total	129	49.8	47	18.0	83	32.1

As might be anticipated, all the much improved and improved groups have left the hospital at some time or other for longer than one month. A higher percentage of the much improved group have not had to return to the hospital. Slightly less than 10 per cent of the improved group have returned, apparently permanently, to the hospital, mainly because of a lack of suitable economic and environmental opportunities. 126, or 32.6 per cent, of the unimproved group left the hospital for more than 1 month, but 74 returned permanently.

The following table shows the relationship between the various diagnostic subtypes and readmissions to the hospital:

	Total.	Number leaving hospital for longer than 1 mo.		Not returned to hospital.		Temporary return.	
		No.	%	No.	%	No.	%
Simple	17	11	64.7	5	29.4	4	23.5
Hebephrenic	159	72	45.2	37	23.2	9	5.6
Catatonic	91	52	57.1	29	31.8	10	10.9
Paranoid	189	79	41.8	33	17.4	15	7.9
"Other"	63	45	71.4	25	39.6	9	14.3

	Permanent return.	
	No.	%
Simple	2	11.8
Hebephrenic	26	16.3
Catatonic	13	14.3
Paranoid	31	16.4
"Other"	11	17.6

The "other" group shows the highest percentage of cases leaving the hospital for one month, as well as the highest percentage of cases with no return to the hospital. In order follow the simple, catatonic and hebephrenic groups. The paranoid group shows the lowest percentage of cases leaving the hospital and the lowest percentage with no return to the hospital and the "other" group the highest percentage of permanent returns to the hospital.

CAUSES OF DEATH.

Of the 89 dead, 52 were males and 37 females. Ages at death were as follows:

15-24	9 cases
25-34	22 cases
35-44	28 cases
45-55	22 cases
Over 55	8 cases

The principal causes of death were:

	Males.	Females.	Total.	Per cent.
Pulmonary tuberculosis	31	12	43	48.2
Suicide	5	5	10	11.2
Pneumonia	2	7	9	10.0
Unknown	4	2	6	6.7
Cardiovascular disease	2	3	5	5.6
Miscellaneous conditions	8	8	16	18.0

SUMMARY.

1. Of 641 clinically diagnosed cases of schizophrenia, admitted to the Rhode Island State Hospital for Mental Diseases, between 1929 and 1934, and followed for 5 to 10 years, 27.5 per cent were found to be at present in the community, 53.5 per cent were in mental hosiptals, 13.9 per cent were dead and for 5.1 per cent no adequate follow-up information could be obtained.

2. Adequate follow-up data were available for 608 patients; of these 6.6 per cent were much improved, 15.3 per cent improved, 63.5 per cent unimproved and 14.6 per cent dead.

3. Classification on the basis of diagnostic subtype showed that 35 per cent of the simple, 16.4 per cent of the hebephrenic, 30 per cent of the catatonic, 16 per cent of the paranoid and 36.4 per cent of the "other" group were much improved or improved. Many of the cases in the latter category were of the so-called schizo-affective type.

4. The age of onset of psychotic symptoms had little bearing on the outcome, except that those cases having an onset at the age of 45 or later tended to do less well than those having the onset at a younger age.

5. Of those cases with symptoms of less than 6 months duration prior to hospitalization 34.5 per cent were much improved or improved. For those with symptoms of 6 to 11 months, 29.4 were benefited; for those with symptoms of 12 to 23 months 27.8 per cent benefited; while for those with symptoms of 2 years or longer only 16 per cent were benefited.

6. 75.3 per cent of the patients requiring a hospital stay of 6 months or less were improved while only 4.2 per cent of those remaining in the hospital for 5 years or longer improved. Patients requiring hospital care for more than 2 years showed little tendency to improve.

7. Disregarding the dead and untraced cases, 259 or approximately 50 per cent of 519 patients left the hospital at some time or other for more than one month; of these 129 have continued to remain outside the hospital while 47 returned temporarily and 83 permanently. The much improved and improved groups show a higher percentage of cases remaining out of the hospital continuously and a smaller percentage of returns than the unimproved group.

8. Pulmonary tuberculosis was the most common cause of death, being responsible for 43 or 48.2 per cent of 89 deaths. 10 (11.2 per cent) cases committed suicide.

NOTE.

The appended table compiled from the recent literature shows the comparative results obtained in various psychiatric centers. In comparing the results, the type of the hospital, whether private, state or psychopathic, the selection of patients for admissions, and the duration of the follow-up study must be considered.

APPENDIX.

Results obtained in other hospitals:

Place.	Much improved, per cent.	Improved, per cent.	Unimproved, per cent.	Dead, per cent.	No. of cases.	Duration of follow-up, years.
R. I. State Hospital.....	6.6	15.3	63.5	14.6	608	4½-10
Colorado Psychopathic (2).	7.5	15.6	76.0	...	345	1-3
Iowa State Psychopathic (5)	22.0	10.0	58.0	10.0	177	5-9½
Rochester, N. Y. State (6).	18.4	16.7	64.9	...	641	3½
Penna. Hospital, Phila., Pa. (4)	10.3	21.5	56.8	8.5	116	5
Bloomingdale, (1) White Plains, N. Y.	26.0	16.0	59.0	...	500	2-12
Maudsley, Eng. (3).....	21.5	21.3	50.9	4.1	188	2-4

BIBLIOGRAPHY

1. Cheney, C. O., and Drewry, P. H., Jr.: Am. J. Psychiat., 95: 203, 1938.
2. Romano, J., and Ebaugh, F. G.: Am. J. Psychiat., 95: 583, 1938.
3. Guttman, E., Mayer Gross W., and Slater, E. T. O.: J. Neurol. and Psych., 2: 25, 1939.
4. Bond, E. D., and Braceland, F. J.: Am. J. Psychiat., 94: 263, 1937.
5. Malamud, W., and Render, N.: Am. J. Psychiat., 95: 1039, 1939.
6. Hunt, R. C., Feldman, H., Fiero, R. P.: Psychiatric Quart., 12: 414, 1938.
7. Statistical Manual—National Committee for Mental Hygiene—Sixth Edition, 1934.
8. Hunt, R. C., and Appel, K. E.: Am. J. Psychiat., 93: 313, 1936.

THE RELATIONSHIP BETWEEN EARLY SCHIZOPHRENIA AND THE NEUROSES.*

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Although the term "schizophrenia" has generally replaced the older term "dementia præcox," it has chiefly rung the changes on emphasizing the rôle of affective, associational and attention defects, rather than stressing the terminal aspects of the disorder. Studies from both physiological and psychological approaches have not as yet helped us to formulate, in terms of a working hypothesis, the basis for the deficiency in affectivity or the other primary changes. More recently, the rapidly expanding series of patients treated with insulin and metrazol have shown us that, whatever may be the factor in bringing about remissions in certain individuals, it does not achieve it in the majority, no matter how certain we are of the diagnosis. Whether this group is further refined by selecting only early cases, or those of a similar reaction picture, we continue to find that remissions cannot be predicted. This immediately raises the old question—is schizophrenia a disease entity or is it a number of disease entities, or what is even more likely, a variety of reaction types which have certain points in common. Kraeplin emphasized the essentially deteriorating aspects of the reaction. Bleuler discussed the fundamental associative disorder, even emphasizing the possibility of its having a metabolic basis. Adolf Meyer,[1] as early as 1906, in discussing the then new concept of "dementia præcox" as outlined earlier by Kraeplin, decried the implication present in Kraeplin's concept. He preferred particularly to emphasize the development of the reaction in the setting of a certain personality background. More important was his pointing out that milder forms of reaction types occurring in simple maladjustments, psychasthenias and hysterias also turn up in this more inadequate setting, with "destructive rather than helpful results." The principle contained in this still

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From the Iowa State Psychopathic Hospital.

challenging article, which is of importance to us today, is that Meyer stressed the necessity of starting with the "premises rather than the results." This, in the face of what then was Kraeplin's monumental contribution to psychiatry has been affirmed over and again by the modern dynamic approaches.

The great impetus given to dynamic psychiatry by Meyer's development of psychobiology and by the Freudian psychoanalytic investigations, has served to focus our attention on those "premises." The result has been that if we can secure enough facts from the life history and an understanding of what those experiences mean in terms of the inner life of the individual, we soon appreciate that the development of a mental illness follows an orderly understandable progression or evolution. To use Meyer's term, this "experiment of nature," like any other experiment, adds to our knowledge, if the conditions of the experiment are sufficiently known.

When human behavior has deviated from the average to the point which can be called "schizophrenic," it has gone through a series of less malignant reactions, which show themselves as maladjustments or more clear-cut neurotic reactions. It is true that part of the process may have been more hidden or less overt than at another stage. But so is much of normal human behavior. The whole mystery of "mind" or "functioning at the psychobiological level" is contained in its unique characteristics of being largely subjective and "personal." The psychoanalytic school has made its great contributions in the exploration of behavior from this aspect. The subjective behavior is then to be considered the forerunner of what sooner or later will be overt performance, whether a neurosis, a psychosis or useful accomplishment.

For the purpose of this paper, we have selected a series of early schizophrenic reaction types, which have shown during their development sufficient overt evidence of the underlying process, to demonstrate to the observer something of the nature of the illness before its final picture appears. That these earlier reactions should resemble familiar syndromes of neuroses is not unusual and they are found in many cases of schizophrenia in which a careful history is obtained. If we conceive of the neuroses as either part disorders of the personality or as solutions to varying degrees of emotional conflicts which could not be solved on a more realistic basis,

they are forms of complex behavior, neither as severe nor as deeply disintegrating as the schizophrenic reaction. But they are clearly steps on the way to more severe disorders, if some sort of equilibrium is not reached before then. The reasons why some reactions stop at the level of a neurosis, and others go into a psychosis, are complex ones. In some it seems to be largely a matter of underlying personality factors, in others it seems to depend more on the defect in the method of handling disturbing experiences related to the early instinctual life. That the two factors interplay constantly in determining what crystallizes as the personality pattern for the individual is well known.

That some individuals are born with traits, characteristics and constitutional determinants of such a type that leads directly to the schizophrenic reaction sooner or later, seems to be true. That others, because of early experiences and conditioning factors, have these same modes of reaction well established and greatly increase their chances of falling back upon these methods of behavior under later stresses, also seems to be true. As to whether or not the third group, with relatively more stable personality structures, but who also under repeated assaults upon their attempts at meeting life, retreat step by step, first by maladjustments, then by neurotic compromises and finally by a full surrender into schizophrenic, is the concern of this paper. Zilboorg [2] in his analysis of cases by the analytic method has already stated that he believes this often to be the case. Myerson [3] also has more recently, from the non-analytic viewpoint proposed the same concept. Both of these authors stress the reversibility of the reaction, and that having reached the stage of schizophrenic behavior it is not necessarily a fixed or static state, nor does it always lead to ultimate deterioration. In many individuals quite the reverse is true. It is a reversible process, particularly in the group we are concerned with, who throughout their lives have demonstrated resources in personality reaction.

The first case is that of a 22-year-old college girl who was brought to the hospital in January, 1938, because she had developed acutely disturbed periods in which she had assaulted her mother. The onset of the illness was dated from June, 1937, when she began to complain that God was displeased with her. She complained of being unable to concentrate, talked vaguely of having slapped the Holy Ghost in the face, thought her head was wooden and her limbs were leprous. Following a vacation with much outdoor activity there was some improvement, but on returning to college in the fall there was a

return of symptoms. She began to speak of hearing God's voice and the Devil's voice. One would reprimand her, the other encourage her to go on and carry out her impulses. It was in this setting that the violent attitude toward the mother began. Because of this she was taken to a local hospital and then admitted from there to the Psychopathic Hospital.

The patient, however, dated her illness back to a much earlier period. To use her own description—"As a child I began to call God and Jesus names and wondered why. I asked my aunt how to stop thoughts one shouldn't think, for it bothered me, for I kept calling them things and thought some day I'd land in an institution. Imagine a 12-year-old child thinking that. I attached sex to God and secretly hated God and Jesus, because as I realize now, they were of masculine gender. Then God was so wrathful. I knew if I died I would never go to heaven. When I was 14 I called God awful things again thinking I had to do it because there was something wrong with my mind. I felt so mean and wicked that I began to search the Bible to see if there was any sign of forgiveness. I came across the part telling of the unpardonable sin (Matthew, Chapter 12, Verse 31) which scared me almost to death when I saw it. I cried and cried, for I hadn't already done it, but I was sure to some day."

These obsessive thoughts and compulsive urges were associated with many elaborations and rituals, until the patient's life became reduced to a rigid pattern. At the time of the flaming up of this psychasthenic picture, the patient had begun to menstruate, and she was in the throes of masturbation with intense feelings of guilt. During her later years of high school and early years of college, it gradually died out. In its place appeared a period of marked crushes on her teachers and older girls, accompanied by a tremendous push of work and scholastic accomplishment. The important point for us is that the onset of the present illness was preceded by a return of these same psychasthenic symptoms. The onset of the psychosis proper, however, was so different and so clear-cut that the patient herself dates it definitely to June 6, 1937. She says, "I felt something coming I couldn't escape." Later she says, "Absolutely I felt as if my doom had been pronounced." Still later she described it as, "It felt as if mountains and rock and bricks were crumbling." There were many somatic delusions and the later stages were striking because of the affective flatness.

Under psychotherapy, as previously described by Malamud and the writer,[4] a good recovery has since been achieved, but with some affective flatness.

The next case came to us not in the psychotic reaction stage, but during the neurotic reaction, characterized chiefly by hysterical manifestations—pain, convulsive attacks and amnesic periods. She was a high school girl of 14 years, from a family of inferior status. After the death of the father, when the patient was ten, a change in the disposition was noticed by other members of the family. It was characterized by irritability, moodiness and occasional crying spells. She always attributed these to her feeling of un-

happiness over the loss of her father. In the spring of 1937, six weeks before the first admission to our hospital, she was seized with nausea, vomiting and pain in the right lower quadrant. This was diagnosed appendicitis and the appendix removed. After returning home, she began to develop confused attacks, when she would throw herself about as though in a struggle. When clear she would have no memory for these occurrences. She was then admitted to the Psychopathic Hospital. A rather quick recovery was made after she talked about her antagonism toward her mother for going about with a man whom she regarded as an intruder in the household. Shortly after discharge, however, she began to complain of physical distress again, which continued throughout the winter, but not enough to prevent her continuing school. These finally terminated in a return of her amnesic and convulsive attacks, necessitating a return to the hospital. On this admission she formed a strong rapport with another psychiatrist, and brought out material which revealed the sexual fantasy behind her attacks and illness. They were based on incestuous relations begun by the father when she was seven and continued by her brothers after his death. With the appearance of puberty and the gaining of knowledge from outside sources concerning pregnancy, her abdominal symptoms began. Following the bringing out of this material, the patient developed a psychotic reaction characterized by apathy, incontinence, auditory and visual hallucinations. She heard the voice of her mother urging her to break windows, and felt compelled to carry out the orders, injuring herself badly in doing it. She hallucinated her father, and said he had returned from the grave to care for her. She developed the delusions that her family had substituted someone else for him when he was buried. There were bizarre postures maintained for days, with queer manneristic patterns. She explained these as having a special significance. After a period of several weeks, having continued psychotherapy she made a recovery, going out to a boarding home, and finally to her own home.

This patient was of unusual interest, because under observation she gradually changed from classical hysteria to a reaction with all the outward manifestations of schizophrenia. Not only was the behavior recorded and observed, but the dynamic factors were also known. It is of interest to note here that the recovery was not made in the reverse order, but through the psychosis and with some sacrifice again of affectivity.

The next illustrative case was that of a 24-year-old single man, in whom the first manifestations of difficulty appeared when he was about eight years of age. He was the oldest of three siblings, brought up in a fairly strict Roman Catholic family. The mother was the dominating figure and was the main factor in determining the patient's attitudes toward life. Previous to the age of eight, there had been no evidence of any difficulty in adjustment. About this time there was some mutual sex play with a boy playmate. The

mother found out about it, and the issue was made an extremely serious one in the life of the patient. He describes the reaction to this as follows: "I guess I built up a sensitiveness in my childhood. You might call it a girlish attitude. I was so bashful I'd go to confession. I probably had the wrong idea about all that (referring to sex matters). I do remember I was scared to death to touch my sex organs. I'd built up that attitude from my contact with that little boy. I think that influenced my whole life—the negativeness I put in my idea. All I knew was to pray and disregard that part of my life. When I was in the sixth grade, I just went wild. I went around with a wild kid named Johnney. He got into trouble with girls, but I just raised hell in class. A teacher suspected me of every deed on a sexual basis. I got punished for things I didn't do. The trouble was I had to think so much of those dirty things that when she accused me of it, I just couldn't talk." Patient goes on to describe how he learned about his parents having sex relations and couldn't understand how the Church would permit it. The description of his outward personality at this time shows him to have developed an over-conscientiousness and over-meticulousness regarding dress and a beginning of a tremendous drive for work and study. Life became an increasingly rigidly organized and stereotyped routine. Only in this way, with every waking moment occupied did this boy seem able to manage his underlying feelings of guilt and remorse. He became a leader in his classes and went to college on a scholarship. Puberty had occurred at 14, but he had shown no interest in girls until the summer before going to college. At this time he became increasingly aware of his sexual urges, first through masturbation and petting parties after hearing boys at camp describe their experiences, and then the following year after his first serious amatory interest, with considerable worry relative to sex drive and nocturnal emissions. The following summer he went to camp, and he describes the sensations accompanying the development of his psychosis as follows: "The summer I went up there (to camp) I went all to pieces. I just seemed to lose myself completely while I was there. I just couldn't seem to find myself or get control of myself. I slept in a room alone and every night, or rather every morning, there were so many and so much wet dreams. I was worried to death all the time. Instead of it being a happy vacation, it was a hideous nightmare." From then on, the patient became convinced he was disintegrating physically, and began to seek relief in two ways. First by trying to develop himself physically and secondly by consulting physicians. Unfortunately, he was given medication, of a type which emphasized the physical symptoms. He finally became more and more frantic and began to express himself in terms of change in bodily configuration—"runting" was his word. X-rays were taken of his skull and he tried to convince everyone that these changes were taking place. Because he could get no help, he gave up college and took to his bed, giving himself up to unrestrained masturbation, explaining he had to "drain his system." In this hospital he was outstanding because of his florid somatic delusions and the pronounced associational disorder in his productions. Under metrazol therapy given at another hospital he has had a good remission.

In this patient, one could hardly describe the change that took place following eight years of age as a clear-cut neurosis. It was more of a character change, the dynamic factors of which, however, were neurotic in nature. The result was a rigid psychasthenic, perfectionistic, over-conscientious, guilt-ridden boy. When the added physiological burden of his maturing sex demands were added to this top-heavy personality structure, it was no longer adequate to protect him from his early childhood feelings of guilt. Instead his personality began to disintegrate along the lines of bizarre somatic symptomatology, with the added development of a pseudo-philosophic system to account for his failure. That the process was a reversible one seems to have been shown by his reaction to metrazol. Again the recovery is marred by the scar of a poor affect and loss of drive.

In general, what may we say as to the relationship of the neuroses to early schizophrenia? From this group of cases, selected from a large number of our early schizophrenias, we should agree with Meyer's statement that "schizophrenia is the outgrowth of a mass of dynamic factors." In every case it is not easy to secure evidence as to the interplay of these factors, unless, in the development of the personality, they appear in behavior as symptoms of early neuroses or traits of character. If we were fortunate enough to be treating a neurosis, which progressed to a reaction characteristic of schizophrenia, then we have an ideally experimental situation, many variables of which we are made aware (Case II). If we are treating an early schizophrenic reaction, in which affectivity is sufficiently well preserved to insure a fair degree of rapport, we can often secure a detailed reconstruction of the life situation, tracing the dynamic development of reactions culminating in the final illness (Case III). The third possibility lies in the study of the personality of a recovered patient who has maintained insight into his reaction, and with whom a detailed analysis of the entire development and recession can then be subjected to study (Case I).

That there are schizophrenic reaction types, in whom constitutional factors play a leading rôle, there seems to be little doubt, although with these we are not specifically concerned in this paper. In such cases the final illness would appear to be an exaggeration of already strongly accentuated personality traits. However, in

the group of patients with whom we are concerned the schizophrenic reaction can be considered as another step in the effort of the personality to find a balance between the inner driving forces of life and the demands of reality, along the paths dictated by personal trial and failure.

In none of these patients have underlying personality characteristics of the "schizoid" or "shut in" type been outstanding. To reach the final regressive behavior which corresponds to the clinical picture of schizophrenia, there has been a gradual retreat, with every evidence of a struggle to maintain contact with reality. Even when reality was finally surrendered, under the final stress, there remained in these individuals the potentialities for recovery.

It would seem to be beside the point to say that the diagnosis was incorrect, and that we are probably not dealing with schizophrenia. At the best, schizophrenia still is a descriptive term, and will remain so until definite etiological factors are found. Even if one follows the more definite formulations of Bleuler and insists that the primary disorder is in affectivity, we are still dealing descriptively with a more subtle form of behavior; subject to a marked variation in evaluation by different observers.

Let us say then that the final psychotic reaction labeled schizophrenia may be reached in a variety of ways. One of those ways we have found appearing repeatedly in certain patients, not exhibiting the schizoid or shut-in type of personality, and this is through the route of the neuroses.

BIBLIOGRAPHY.

1. Meyer, A.: Fundamental conceptions of dementia præcox. Brit. M. J. London, 2: 757-760, Sept., 1906.
2. Zilboorg, G.: Deeper layers of schizophrenic psychosis. Am. J. Psychiat., 88: 493-511, Nov., 1931.
3. Myerson, A.: Neuroses and neuropsychoses, Am. J. Psychiat., 93: 263-297, Sept., 1936.
4. Malamud, W., and Miller, W.: Psychotherapy in the schizophrenias. Am. J. Psychiat., 88: 457-480, Nov., 1931.

PSYCHOTIC MANIFESTATIONS IN THE PRESENCE OF ACUTE CEREBRAL VASCULAR LESIONS.*

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Several years ago the writer undertook a statistical study of correlations existing between acute cerebral vascular lesions, as found at necropsy, with psychotic manifestations, as noted clinically. The material on which this study was based consisted of 1,000 cases of such lesions described in the records of 12,503 consecutive necropsies done at Los Angeles General Hospital between March, 1918, and November, 1934. The object of this communication is to submit, in briefest outline, a report of the resulting findings.

Cases presenting the following types of acute cerebral vascular lesions were included in the material selected for study:

	Cases.
Embolism	133
Thrombosis	351
Sinus and venous thrombosis	98
Hemorrhage	418
Total	1,000

The clinical records, routinely made, contain no detailed and complete descriptions of psychotic manifestations, nor any refined psychologic analyses of them. One gains the impression that only the more obvious and spectacular psychotic manifestations have been noted in the clinical records, and that the milder ones have been ignored or overlooked.

For the purpose of this study the psychotic manifestations have been classified into four groups: 1. Confusion with irritability.

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The material for this communication was obtained from an extensive monograph on Vascular Encephalopathy, which included an intimate survey of 1000 necropsied cases, presented to the Graduate Medical School of the University of Pennsylvania as the partial requirement for the doctorate degree in neurology.

if severe enough to be considered of psychiatric character. 2. Delirious states, noisy, manic-like or lethargic. 3. Delusional or hallucinatory trends with or without belligerence or other abnormal behavior. 4. Established defects of judgment and memory, indicating mental deterioration, probably, in contrast with the first three groups, not reversible.

Psychotic manifestations of one or another of these types were recorded in 170 of the total of 1,000 cases studied.

It goes without saying that among the cerebral manifestations of these vascular lesions there were also such symptoms as coma, aphasia, convulsions, twitchings, paralyses and the like, in combination with the psychotic manifestations or independently. However, it is rather significant that in 78 of the total of 170 cases with psychotic manifestations, *i. e.*, 45.9 per cent, no neurologic signs were recorded.

No doubt, in this connection, too, the statistical figure understates the case. The mere fact of the presence of such psychotic manifestations as confusion, excitement or deterioration results often in such incooperation on the part of the patient as to preclude the possibility of eliciting neurologic signs which may be present.

Perhaps it should be noted here that the material which forms the basis of this study is hardly comparable to the chronic psychoses which are associated with cerebral arteriosclerosis, seen in state hospitals and described in psychiatric textbooks. We are dealing here mainly with acute vascular lesions manifested by acute clinical phenomena. Moreover, by selection, we are dealing with the most severe, regularly fatal cases. The average survival period for all patients in the entire survey, with emboli, was 8.3 days, with thrombi 14.5 days, and with hemorrhages 6.8 days. In some of the cases the psychotic manifestations were attributable, at least in part, to factors other than the vascular lesion, especially those of infectious or toxic nature.

The following brief case summaries will give a general idea of the material on which this study is based.

CASE 1.—A male, 73, had been much disturbed mentally for ten days. His blood pressure was 215/80, pulse 112, and a systolic mitral murmur was present. He became unmanageable and was transferred to the psychopathic department, dying the third day afterward. Extreme nephrosclerosis and

sclerosis of the cerebral vessels, with a 2 cm. softening in the right temporal cortex, were found at necropsy.

CASE 2.—A male, 74, was admitted to the hospital in noisy, delirious semi-stupor. He had been irrational for several days and mentally disturbed on a number of occasions the previous six months. The blood pressure was 180/90. He was almost moribund when admitted, but lived four days afterwards. In the brain a hemorrhagic area of softening was present just beneath the right lateral ventricle at the border of the corpus striatum and extending into it. A partially organized thrombus was contained in the small vessel of this area.

CASE 3.—The occipital lobe was found entirely softened following complete thrombosis of the right posterior cerebral artery in a man 58, hospitalized for mental observation. He was confused, irrational, incoherent and noisy when admitted. He had attempted suicide, expressed delusions of persecution and was violent at times, frequently hammering his head against the wall.

CASE 4.—A female, 37, was admitted in a confused mental state, resistive and maniacal, with hallucinations of sight and hearing. The attack had begun the day before with a very acute manic-depressive syndrome. Following her death, on the fourth day after the onset of her illness, the basilar artery was found completely thrombosed for 1 cm. from its distal end, with extension of the process into the right posterior cerebellar artery for 1 cm. No hemorrhage or softening was present.

CASE 5.—A sudden unconsciousness lasting 24 hours occurred in a female, 50, about two weeks after satisfactory convalescence from a mild upper respiratory tract infection, with apparent complete recovery. Three weeks later she suddenly became apathetic, showed no interest whatever, became resistive, would not talk, and refused food. This condition had existed for a week when she was admitted to the hospital mentally confused, disoriented and resistive. The reflexes were equal, tone normal, and no evidence of motor or sensory impairment was found. The blood Wassermann reaction was negative and the spinal fluid normal. She died on the eighth day of her hospital residence. Coronal cerebral sections disclosed 5 cm. areas of degeneration of the globus pallidus of both lenticular nuclei adjacent to the anterior limbs of the internal capsules.

CASE 6—A male, 49, had a history of four years' illness of insidious onset with progressive mental impairment. Two years after the onset of this condition he developed sudden leg weakness and dropped to the floor. This was repeated several times and periods of general weakness with mental depression and frequent emotional episodes ensued. On one occasion, when psychotic manifestations were marked and the patient was difficult to care for, he was admitted to the psychopathic department. He was confused and disoriented, had frequent crying spells and was extremely unstable emotionally. The blood pressure was 218/160. The pupillary reactions were

normal. The patellar reflexes were exaggerated and a suggestive Babinski reaction was elicited on the left side. Brain examination disclosed marked arteriosclerosis but no occlusions. Numerous areas of softening were present throughout the brain substance, the two largest, 3 x 1 cm., in the caudate nuclei, external to the lateral ventricles, on both sides. A small softened area was noted in the right putamen, and one, still smaller, in the white substance above this. Another small softening in the left claustrum involved the left external capsule. There were numerous 1-3 mm. areas scattered throughout, chiefly in the anterior lobes.

To some extent the development of psychotic symptoms seems to be dependent on the type of the acute vascular lesion, as may be judged from the following figures:

	Per cent.
Embolism (133 cases), psychotic symptoms in	22.6
Thrombosis (351 cases), psychotic symptoms in	20.8
Sinus and venous thrombosis (98 cases), psychotic symptoms in	8.1
Hemorrhage (418 cases), psychotic symptoms in	14.1
All cases (1000), psychotic symptoms in	17.0

As to localization of the cerebral pathology in the patients with psychotic symptoms, the figures show that cortical destruction in the area of supply of each of the three major vessels—anterior, middle and posterior cerebral arteries—gave an approximately equal frequency of mental symptoms. Such symptoms were definitely much less frequent where there was involvement of the basal ganglia or implication of any of the perforating vessels.

The incidence of pathologic involvement of the two cerebral hemispheres in the entire series was approximately equal for all types of lesions, but there was a somewhat greater incidence of right cerebral involvement, particularly thrombotic, among the patients displaying mental symptoms. The significance of this finding is more than doubtful, not only because the contrast is so slight as to be well within the statistical limits of probable error, but also because there is every reason to believe that the psychotic manifestations in these cases are to be attributed, for the most part, not to any special localization or laterality of the lesions, but rather to the diffuse effects of the general intracranial turmoil occasioned by the acute lesions, which were so severe and so extensive as to lead to a rapid fatal termination.

In the appended table the data are presented in detail.

VASCULAR DISTRIBUTION OF LESIONS DIVIDED AS TO TYPE AND INCIDENCE OF PSYCHOTIC MANIFESTATIONS.

(In 1,000 cases found at 12,503 consecutive necropsies.)

	Embolism (133), No.	Psychotic (30), No.	Thrombosis (449), No.	Psychotic (81), No.	Hemorrhage (418), No.	Psychotic (59), No.	Total (1,000), No.	Psychotic (170), No.	Per cent.
Left anterior cerebral	4	1	4	..	12	2	20	3	15
Right anterior cerebral	2	1	10	2	12	3	24	6	25
Anterior communicating	0	..	0	..	2	1	2	1	50
Left middle cerebral	8	3	35	4	2	..	45	7	15.5
Left mid-cortical	10	1	26	5	10	1	46	7	15.2
Left mid-perforating	11	2	35	4	70	3	116	9	8.0
Right middle cerebral	21	4	43	4	2	..	66	8	12.1
Right mid-cortical	8	1	13	3	11	2	32	6	18.7
Right mid-perforating	5	1	30	8	65	6	100	15	15.0
Left posterior cerebral	3	..	7	3	4	1	14	4	29.0
Right posterior cerebral	4	1	7	4	9	3	20	8	40.0
Basilar and pontile	(2)	..	31	5	24	1	55	6	10.9
Vertebral	(1)	..	4	1	0	..	4	1	25.0
Left cerebellar	3	1	4	..	15	2	22	3	13.6
Right cerebellar	0	..	4	1	13	1	17	2	12.1
Carotid arteries	8	1	14	6	1	1	23	8	34.8
Bilateral	14	7	34	9	14	3	62	19	30.6
Anterior cerebral	3	3	1	0	..	..	4	3	75.0
Middle cerebral	2	1	0	..	0	..	2	1	50.0
Mid-cortical	4	1	14	4	1	..	19	5	26.3
Mid-perforating	4	1	18	5	11	2	33	8	24.2
Posterior cerebral	1	1	0	..	0	..	1	1	100.0
Cerebellar	0	..	0	..	2	1	2	1	50.0
Internal carotid	0	..	1	..	0	..	1	..	0.0
Multiple lesions	19	6	50	14	58	9	127	29	22.8
Petechial involvement	12	..	..	..	24	4	36	4	11.1
Sinus thrombosis	..	..	88	8	..	..	88	8	9.0
Longitudinal	..	..	16	3	..	..	16	3	18.7
Lateral	..	..	38	2	..	..	38	2	5.2
Cavernous et al	..	..	34	2	..	..	34	2	5.9
Venous thrombosis	1	..	10	1	0	..	11	1	9.0
Subarachnoid hemorrhage	..	..	..	..	30	7	30	7	23.3
Subdural hemorrhage	..	..	..	..	37	9	37	9	24.3
Intraventricular hemorrhage	..	..	..	..	3	..	3	0	0.0
	133	30	449	81	418	59	1,000	170	17.0

DISCUSSION.

DR. WILDER PENFIELD (Montreal, Canada).—It requires a good deal of courage for one who deals only with cases in which there is an organic lesion of the brain to discuss and follow Dr. Irish who handles primarily patients with psychotic manifestations, but without organic evidence of trouble.

Nevertheless, there is common ground, and that common ground may be in the field of attempting to find evidence for localization in psychotic patients.

I was very much interested in Dr. Irish's statistics. The psychotic man, regardless of localization, will frequently show a thrombotic process where there was a hemorrhagic vascular change. In the last two cases, there is an area of the brain that is knocked out suddenly and completely. It undergoes absorption rapidly, with cleancut demarcation of what is dead brain from what is living.

It produces quite a different effect from the result of thromboses which occur often gradually and diffusely. Thromboses of that sort produce a different effect as judged histologically in the brain. There is greater evidence of perhaps a toxic factor. At least, the neuroglia is more apt to show evidence of chronic change, such change as occurs in the neuroglia as the result of toxic influence.

So far as that goes, therefore, there may be in the thrombotic process an element of stimulation as well as destruction of areas of brain, small areas, but there is also evidence for a period of time of a toxic element which perhaps accounts for the larger incidence of psychotic change in those individuals.

I was hoping that Dr. Irish would point to a certain area of the brain in which the involvement was most frequently found. My feeling is that there must be such an area.

In turning to cases of brain tumor, for instance, there are certain areas of localization where mental disturbance is much more frequent. These do not include the frontal lobes, in my experience. The frontal lobe tumor gives a slowing, a lack of initiative, and that sort of thing, but does not give evidence of mental disturbance in the sense in which I take it that Dr. Irish uses the word "psychotic."

On the other hand, tumors of the temporal lobe in our experience are much the most frequent causes of mental disturbance, and I suspect it is not because the tumor is nearest the surface in the temporal lobe but because that tumor is nearest certain deep structures beneath the temporal lobe that is the explanation for this localization.

Still more frequently the localization of organic production of mental change, in my opinion, is the region of the third ventricle. Supra-pituitary tumors which are of long standing have sent a great many patients to asylums. A number of the cases that have been recorded as frontal in asylums are in fact, I think, disturbances due to the fact that the tumor extends back into the supra-pituitary region.

We have had a number of examples of tumor of the wall of the third ventricle producing psychotic change. I have a girl in my care at the

moment who has a neoplasm in one wall of the third ventricle whose disturbance began with a seizure which has been followed continuously by psychotic alterations. She has been negativistic and sometimes she becomes violent.

Her behavior would place her, I suppose, in the group of dementia præcox, although I fear to use difficult terms which I do not understand. But it is in those regions of the brain which, if destroyed, unconsciousness is produced. It is in those regions that disturbance produces psychotic manifestations.

That is a hypothetical suggestion, but it is borne out so far as we can see by the localization of organic lesions which are associated with psychotic manifestations.

DR. CULLEN WARD IRISH (closing).—With regard to localization of these lesions, as Dr. Penfield suggested, in vascular lesions the cerebral insult is almost always 100 per cent more acute and more devastating with the accompanying edema, irritation, and other acute symptoms that arise from any condition affecting the brain in a rapid or sudden manner.

We could not, for instance, classify the lesions as to location of pathology in quite the same manner as you could a more slow, progressive process like a tumor. Hence they could not be well correlated. I attempted to do that.

THE EFFECT OF TREATMENT OF DEPRESSION IN THE MENOPAUSE WITH ESTROGENIC HORMONE.*

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AND

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The relationship between glandular function and psychic disturbances has recently been the subject of considerable clinical investigation. The availability of potent estrogenic hormones and improved methods of evaluating their action have stimulated interest in possible therapeutic applications in both mild and severe reactions occurring in association with the menopause.

Results obtained by different investigators in the treatment of menopausal syndromes with estrogenic hormone have varied. It is generally agreed that the common menopausal syndrome with vasomotor symptoms and mild depression is relieved. Among those who have noted favorable results are Papanicolaou and Shorr,[1] Sevringhaus,[2] Frank, Goldberger and Salmon,[3] Wiesbader and Kurzrok,[4] and Hawkinson.[5] In reporting results in severe depressive illnesses there has not been unanimity of opinion. Werner [6] concluded that "for all practical purposes" this therapy "is specific for involutional melancholia." Improvement in patients with involutional and other severe depressive illnesses has also been reported by Jones, MacGregor and Tod,[7] Suckle,[8] and Little and Cameron.[9] Bowman and Bender [10] after studying seven cases of involutional melancholia reported that patients who show depression, indecision, delusions and hypochondriacal trends will probably not be benefited. Schube, McManamy, Trapp and Hauser [11] who gave protocols of 10 patients with involutional melancholia treated

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with estrone found that in none was there evidence of improvement. Hutton and Schiller [12] stated that in cases of involutional psychoses treated with estrone the percentage of those who improved was not more than that usually reported for spontaneous remissions.

Not only is there disagreement concerning the results of therapy but frequently there is failure to differentiate clearly between the clinical syndromes under treatment. For example, in the literature some use the term involutional melancholia as the diagnosis of any depression in the menopause rather than confine it to the syndrome described by Hoch, characterized by depression, anxiety, feelings of unreality and hypochondriacal and nihilistic delusions. This psychopathological picture suggests that involutional melancholia is a different entity rather than an accentuation of the vasomotor changes such as hot flushes, chills, sweats and palpitation that are seen in the mild reactions and that are usually lacking in the severe psychotic cases.

The psychological and physiological manifestations in depressive illnesses of the post-menopausal age period are of great complexity and not clearly understood. In view of the obscurity of the etiological factors it is important to isolate and study the effect of one such possible factor at a time. This investigation deals with a study of the diminution of ovarian activity and the effect of replacement with a single ovarian hormone.

In the group we are reporting adequate amounts of hormone were given to produce a gradual transformation from the menopausal type of vaginal smear to the type found during the follicular phase of the normal menstrual cycle just prior to ovulation. Frequent examination of vaginal smears was made by the method described by Papanicolaou and Shorr.[1] The use of the vaginal smear as a guide to therapy is of particular importance in mental illnesses because of the difficulty in using the subjective indices by which the effect of this hormone is commonly evaluated.

A group of 20 patients suffering from depression in the menopausal and post-menopausal age group have been studied in the Payne Whitney Clinic of the New York Hospital and the effects of treatment with estrogenic hormone evaluated. Detailed consideration was given to each individual case with attention to the broader psychobiological aspects such as hereditary, constitutional,

physical, social and economic elements. In addition to treatment with estrogenic hormone the therapeutic measures of psychotherapy, regulated exercise, hydrotherapy, selected recreation and occupational therapy were used. All the patients were sufficiently ill to require treatment in the psychiatric in-patient department. There was however considerable variation in the character and severity of the symptoms.

The patients in this investigation fell into three diagnostic groups: (1) seven cases of involutional depression; (2) six cases of depression of the manic-depressive type; and (3) seven cases of depressive illnesses of a more reactive nature. In some patients of this last group inherited tendencies to depression were present as well as psychoneurotic factors. The ages ranged from 39 to 58 years.

All patients had a menopausal type of smear before treatment was given. Two estrogenic hormone preparations were used: estradiol benzoate (progynon-B) and estrone (amniotin).* In each course of treatment, from 10 to 70 days in length, dosages varied between 500 and 6000 R. U. of estradiol benzoate and between 1250 and 3750 R. U. of estrone daily. A variable number of courses were given to different patients. Total dosages to individual patients were from 48,000 R. U. to 851,000 R. U.

Since time does not permit a detailed presentation of all the cases, three have been selected for discussion as representative types.

CASE 1.—The patient was a 59-year-old, divorced woman admitted to the hospital February 5, 1938, suffering from an involutional depression. In October, 1937, after the death of a brother who was much attached to the patient, she commenced to worry excessively, was afraid to remain alone and spoke in a self-depreciatory manner. Her weight gradually dropped from 134 to 104 pounds. Three weeks before admission she began to sleep poorly and was tense and agitated. She expressed the feeling that she was a sinner and had to punish herself. Menses had ceased when she was 43. The gynecological examination showed no pathological changes.

Estradiol benzoate was given in doses of 1000 R. U. daily from March 6 to 18, and 2000 R. U. daily from March 19 to 22. The original smear was of the menopausal type. As the smear response was not satisfactory, from March 24 to April 1 2500 R. U. of estrone was given. Since there was no further improvement in the smear picture, the dosage was increased to

* We are indebted to Dr. E. Schwenk and Dr. G. Stragnell of the Schering Corporation for the supply of estradiol benzoate (progynon-B) and to E. R. Squibb and Sons for the supply of estrone (amniotin).

3750 R. U. of estrone from April 2 to 16. There was a moderate improvement in the smears. Throughout this period she continued to be restless, agitated, uncooperative, indecisive and despondent. She muttered to herself, screamed, slept poorly and soiled herself with urine. She was very self-accusatory, saw unusual meanings in happenings on the floor, talked much about her sins, and frequently ate so poorly that tube feeding was necessary. She had moderate vaginal bleeding from April 18 to 26. After discontinuation of treatment the smears regressed to the menopsausal type. There was no change in her symptomatology. On May 17 she was given 1875 R. U. of estrone and from May 18 to June 13 3750 R. U. During the first week in June there was a moderately good smear response to the treatment without change in symptomatology. During the second week in June regressive changes in the smears occurred while treatment was being given. From June 17 to 21 she had slight vaginal bleeding. The total amount of estradiol benzoate administered was 21,000 R. U. and of estrone 181,925 R. U. The total amount of estrogenic hormone was 202,925 R. U. She was discharged to another hospital, in an unimproved condition, after five months, having received hormone treatment for three and one-half months.

CASE 2.—The patient was a 45-year-old, married woman admitted to the hospital June 29, 1937, suffering from a depression of the manic-depressive type. In the spring of 1935 her husband had suffered economic reverses and plans were being made for them to move to another city. At this same time the patient's menstrual flow had become scant and she developed a depressive illness with complaints of slowness of thinking, difficulty in concentration and in carrying on her usual activities, tenseness, early morning awakening, constipation, and hot flushes. In October, 1936, she became overtalkative, overactive, and entered into many impractical activities. In March, 1937, she again became depressed and noted symptoms similar to those she had had in 1935, which continued up to the time of admission. There was no family history of mental disorders. Gynecological examination showed a mild senile vaginitis.

Estradiol benzoate was given in doses of 2000 R. U. from August 14 to 20 and 3000 R. U. from the 21 to 25. Under treatment vaginal smears changed from the menopausal to the follicular type. Hot flushes disappeared and she took slightly more interest in her surroundings. After treatment was stopped smears regressed to the menopausal type. From September 14 to 23 she was given 3000 R. U. daily of estradiol benzoate. Smear changes were similar to those previously noted. There was no apparent change in her mental condition. On October 14 she was given 4000 R. U. of estradiol benzoate, from the 15 to the 22 2000, from October 23 to November 5 3000, and from November 6 to 11 4000. She again showed a follicular type of vaginal smear and had no improvement in her mental status. She had slight vaginal bleeding from November 16 to 18. A total of 145,000 R. U. of estradiol benzoate was given. She continued depressed until the spring of 1938 when she again became elated. In this case hormone treatment was administered for a period of three months.

CASE 3.—The patient was a 52-year-old, married woman admitted to the hospital January 9, 1938, suffering from a depression of a primarily reactive type. Following a hysterectomy and oophorectomy in August, 1937, for uterine fibroids, she began to have waves of nausea followed by hot flushes four or five times a day. She gradually became more tense, depressed and worried over trifles, noted increased thirst and had great difficulty in sleeping. She was preoccupied about growing old and dyed her hair. Through a personality study it became apparent that concern about aging was the primary factor in her depression. A brother of the patient had a schizophrenic illness and did not recover. The gynecological examination showed no pathological changes.

Estradiol benzoate was given in doses of 1000 R. U. daily from January 18 to 27 and 2000 R. U. daily from January 28 to February 1. Five days after administration was begun she commenced to sleep better, became less tense and was more cheerful and at ease. Ten days after treatment was started hot flushes disappeared. Vaginal smears changed from the menopausal to the follicular type. Four days after treatment was discontinued she felt more depressed, tense and discontented. The next day there was a return of hot flushes. From February 8 to 12 sterile saline injections were given. Sleep became poor, she worried more and noted increased muscular tension. She described her feelings as "more jittery." From February 13 to March 19 she was given estradiol benzoate in doses of 2000 R. U. daily. Five days after treatment was started she began to feel more cheerful and less tense and anxious. She had a good vaginal smear response following the treatment. During this course of therapy she continued to feel well except for occasional periods of restlessness, slight discouragement and "heavy feeling in the abdomen." Nine days after treatment was discontinued she had a slight flushed feeling and felt that the helpful effects of the therapy were wearing off. From March 29 to April 15 she was given 2000 R. U. of estradiol benzoate daily with improvement similar to that noted previously. The total dosage was 234,000 R. U. On home visits during her hospital residence her response to sexual intercourse which had never been satisfactory improved. After three months of hormone treatment the symptoms that she presented on admission had disappeared and she was discharged from the hospital to return home in a greatly improved condition.

The results following hormone treatment may be tabulated as follows:

	Involutional depression.	Manic-depressive depression.	Depressive reactions.	Total.	Percentage.
No improvement.....	4	1	0	5	25
Slight improvement...	3	3	2	8	40
Moderate improvement.	0	2	4	6	30
Marked improvement..	0	0	1	1	5

DISCUSSION.

The results of this investigation suggest that the milder depressive reactions are more favorably influenced by treatment with estrogenic hormone than the well defined depressive illnesses. The depressions with agitation, feelings of unreality, suspiciousness, hypochondriacal and nihilistic delusions, negativism, abusiveness and combativeness showed little if any symptomatic response to the treatment given. In the less severe depressions of the manic-depressive group and of the reactive type there is partial or complete relief of such symptoms as hot flushes, chills, sweats, palpitation, anxiety, abnormal muscular tension, headache, dizziness, pressure sensations, sleeplessness, weakness, nausea and poor appetite, where such symptoms exist. Such relief resulted in an increase in a general feeling of well being which was helpful in adding to the comfort of the patient so that she was able to deal more satisfactorily with her illness. The data obtained did not support the view that replacement of the deficiency of estrogenic hormone offers a specific therapeutic agent in the types of menopausal and post-menopausal depressions studied.

Vaginal smears offered a valuable objective gauge of the physiological effect of the treatment. It was found that there was considerable variation in the amount of hormone necessary to produce complete replacement as indicated by the development of the follicular type of vaginal smear. If this standard is used, objection to the conclusions of others could be made on the basis of inadequate dosage.

CONCLUSIONS.

In the group of menopausal and post-menopausal depressions studied the beneficial effect of estrogenic hormone was confined to the relief of vasomotor symptoms with associated improvement in feelings of well-being. There was no evidence that the depressive illness as such was influenced specifically or its course shortened.

BIBLIOGRAPHY.

1. Papanicolaou, G. N., and Shorr, E.: The action of ovarian follicular hormone in the menopause, as indicated by vaginal smears. Am. J. Obs. and Gynec., 31: 806 (May) 1936.

2. Sevringhaus, E. L.: Relief of menopause symptoms by estrogenic preparations. J. A. M. A., 104: 624 (Feb. 23) 1935.
3. Frank, R. T., Goldberger, M. A., and Salmon, U. J.: The menopause: symptoms, hormonal status, and treatment. N. Y. State J. Med., 36: 1363 (Oct. 1) 1936.
4. Wiesbader, H., and Kurzrok, R.: The menopause; consideration of symptoms, etiology, and treatment by means of estrogens. Endocrinology, 23: 32 (July) 1938.
5. Hawkinson, L. F.: The menopausal syndrome; 1000 consecutive patients treated with estrogen. J. A. M. A., 111: 390 (July 30) 1938.
6. Ault, C. C., Hoctor, E. F., and Werner, A. A.: Theelin therapy in the psychoses. J. A. M. A., 109: 1786 (Nov. 27) 1937.
7. Jones, M. S., MacGregor, T. N., and Tod, H.: Estradiol benzoate therapy in the depressions at the menopause. Lancet, 1: 320 (Feb. 6) 1937.
8. Suckle, J. E.: Treatment of involutional melancholia by estrogen. J. A. M. A., 109: 203 (July 17) 1937.
9. Little, G. A., and Cameron, D. E.: Effects of theelin on anxiety. Canad. M. A. J., 37: 144 (Aug.) 1937.
10. Bowman, K. M., and Bender, L.: Treatment of involutional melancholia with ovarian hormone. Am. J. Psychiat., 11: 867 (Mar.) 1932.
11. Schube, P. G., McManamy, M. C., Trapp, C. E., and Houser, G. F.: Involutional melancholia; treatment with theelin. Arch. Neur. and Psychiat., 38: 505 (Sept.) 1937.
12. Hutton, J. H., and Schiller, M. A.: Endocrine therapy in the psychoses, with special reference to substitutive treatment of the involutional psychoses. Elgin Papers, 3: 183 (Dec.) 1938.

DISCUSSION.

DR. J. Y. NOTKIN (Poughkeepsie, N. Y.).—Having been impressed with Werner's reports we have decided to try out the estrogenic hormone in 15 involutional melancholia patients. The duration of the psychosis in this group ranged from two years to nine years and the average age was about 45 years. We gave our patients rather large doses; they received 10,000 international units intramuscularly twice a week and 5000 units orally five times a week, or a total of 25,000 units a week. The treatment lasted three months.

Nine of these patients showed slight improvement during the course of treatment. They became more quiet and more amenable to the hospital routine. At the end of the course of treatment, that is, three months later, six patients definitely relapsed, but two other patients who failed to respond began to show some signs of improvement about this time. At a re-check three months later, there were only three patients who maintained some sort of improvement and one patient began to improve about that time.

I must say that the improvement was of institutional character only; they became a little more quiet and not so restless; none was in condition to leave

the hospital to resume life in the community. At a re-check three years later every patient who showed some improvement at one time or other was definitely worse.

For control studies we observed at the same time a group of ten patients of the same clinical type, of about the same age and with about the same duration of psychosis. We gave them physiologic salt solution intramuscularly at the time when the treated patients were receiving the follicular hormone. Of these 10 patients only one showed some evidence of improvement during the first observation period of three months but she too relapsed and none of the other patients showed any improvement during the three years observation period.

I may add that three treated patients had transitory uterine bleedings during and after the course of treatment.

DR. TITUS H. HARRIS (Galveston, Texas).—Dr. Ripley has made a very worthwhile contribution to what has been a highly controversial subject, at least as far as reports in the literature would indicate and it would appear that his report should answer the question as to the value of the estrogenic hormones in the treatment of involutional melancholia.

Since the estrogenic hormones were first used in the treatment of the so-called menopausal symptoms, I have been interested in the subject and it has been my opinion that a deficiency of these substances does not play a part in the nervous or mental symptoms occurring in women at this period of life. This view is supported by the fact that a number of years ago many favorable reports were given following the use of ovarian hormones, and yet today we know that the preparations used then were without definite pharmacological value and could not be given in doses sufficient to bring about physiological changes in the body. And again, the clinical pictures other than involutional melancholia which occur around the menopausal period are also seen at other ages and in both sexes. Although some of the psychoneurotic patients that Dr. Ripley reported showed some improvement, I would feel that there are other factors in the treatment procedure which must be evaluated.

A year ago at the Galveston State Psychopathic Hospital we undertook a study similar to that which Dr. Ripley has discussed. Progynon B was given in massive doses and the total number of units usually exceeded the recommended dosage of most investigators who have reported favorable results with this form of treatment. A vaginal smear was used as an index for determining whether the dosage was sufficient to bring about physiological changes in the body. This study was conducted for the purpose of determining whether estrogenic substances are of value in the treatment of the true involutional melancholia. The results of this study are shown on the chart on the opposite page.

In conclusion, I would again express the opinion that the estrogenic hormones have no place in the management of psychiatric patients.

Name.	Symptoms.	A. Family. B. Previous personality.	Dosage hormones.	Smear: A. Before. B. After.	Results.
Mrs. S. L. L. Age, 56. Duration of illness 8 months.	Dejected, hopeless, bodily complaints, agitated, self-accusatory.	A. Neg. B. Sensitive, worrying type.	4 × 10,000 13 × 50,000 1 × 60,000 Tot. 750,000 bi-wkly.	A. Menopausal. B. Good response.	No improvement.
Mrs. A. B. Age, 52. Duration of illness eleven months.	Restless, agitated, self-accusatory, apprehensive, guilt, religious conflict.	A. Sister: Schizoph. Mother: Religious fanatic. B. Stubborn.	4 × 10,000 13 × 50,000 1 × 60,000 Tot. 750,000 bi-wkly.	A. Menopausal. B. Good response.	No improvement.
Mrs. G. L. G. Age, 60. Duration of illness two years.	Anxious, suspicious, self-condemn., seclusive, unworthiness with projections.	A. Neg. B. Selfish, headstrong, strict, precise.	7 × 10,000 14 × 50,000 Tot. 770,000 bi-wkly.	A. Advanced menopausal. B. Very good response, uterine bleeding.	No improvement.
Miss N. C. Age, 66. Duration of illness one year.	Agitation, depression, self-accusatory, somatic complaints.	A. Not known. B. Well adjusted.	7 × 10,000 5 × 50,000 Tot. 570,000 bi-wkly.	A. Menopausal. B. Good response.	No improvement.

DR. HENRY G. SMITH (Cedar Grove, N. J.).—Dr. Ripley states that his dosage has been larger than commonly used. The last speaker showed a much larger dose than Dr. Ripley has given. We have been using 10,000 units on alternate days for a total of 200,000 units.

On this treatment two of twelve cases have evidenced marked physical and mental improvement; three showed only physical improvement. One patient, a 40-year-old manic, following insulin and metrazol, had not menstruated in three and a half years, and had involutional symptoms, with hot flushes, chills, etc. She was given 110,000 units, following which she had normal menstrual periods beginning in July of 1938, and she has been menstruating regularly every month since that time. Her gynecological examination was negative.

Sixty other cases have received from 200,000 to 320,000 units without change.

DR. RIPLEY.—We did consider that other factors that were used in the treatment of these patients might be having a very definite effect, and I think the most important point in our evaluation was that we could see the influence in certain symptoms. Although the patient might even in some respects be somewhat better, there was a return of hot flushes, for example, and some of the vasomotor symptoms, and when the treatment was withdrawn, we cannot say it had any definite effect on the illness, as I mentioned before. Some of our patients did receive up to 850,000 R. U., and we have one who is being followed now who has had over 1,000,000 R. U., and it does not alter our conclusions.

REPORT OF RESEARCH AND EXPERIMENTATION IN EXERCISE AND RECREATIONAL THERAPY.*

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Introduction.

This paper reports the operations of the highly diversified program of physical education carried on over a period of 17 years at the Veterans' Administration Facility, Perry Point, Maryland. From such it was thought possible to make a long-range study of some of the results, failures and general significance and scope in its more modern application to the problems of rehabilitation. The rationale of play as a general balancing and reparative agency has been studied and well presented and the writers have no intention to discuss its purely psychological setting, but rather to indicate what appears to be certain distinctive applications of physical educational methods and form to the increasing armamentarium of therapy.

Historical Résumé.

Seventeen years ago physical education was utilized as a rehabilitative adjuvant at the United States Veterans Rehabilitation Center, which aimed to restore nervous types to economic usefulness through a vocational program. Numerous psychoneurotic trainees evinced an avidity for many types of both formal and informal physical exercise. Volley ball and baseball teams were formed and calisthenic classes organized. These activities served

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to stabilize many trainees and were of acknowledged therapeutic benefit in their re-orientation to more responsible work procedure. The clinical director of the hospital for mental illness was much interested in the results of such a program for the psychoneurotic types and after the closing of the vocational activities asked that the physical director be transferred to the hospital for the purpose of organizing, with the assistance of the staff, a program of physical therapy for the psychotic patients. Before entering actively upon this program, the physical director visited a number of institutions where physical programs were being carried out and held a number of consultations with leaders in psychiatry who were interested in this aspect of rehabilitation.

From the very beginning, he was impressed with what appeared to be a reawakening of more spontaneous activities as patients participated in pleasurable forms of physical exercise. He was impressed also with the fact that many psychotic patients would act in a relatively normal manner while playing and that in many cases they appeared able to sublimate unwholesome conduct as well as to develop more aggressive behavior of a constructive type. Play appeared to represent a most natural activity which would enlist the interest of a large group of patients. It soon became evident, however, that the play mechanism contained potentialities which could be directed into either a wholesome or unwholesome channel and that it was necessary to devise more studious methods in physical education in order to develop its greatest possibilities as therapy.

With this end in view, the physical director from the beginning attempted to make careful observations of the patients' reactions to various types of exercise and to organize a program upon the fundamental basis of the patients' interests. It soon became evident that these agencies, to be effective, must fit into a therapeutic process which advances from the point of interest to effort. Discovery and appropriating play interests of the psychotic patients was a difficult and arduous task. As the wards came out for exercise, they were all lined up for formal calisthenics which was administered by the aid of records with musical accompaniment. It was soon discovered many patients would take part in such formal type of exercise and would not participate in the more socialized games. After the calisthenic exercises, patients would then sit in the grandstand while the physical director and atten-

dants made efforts to enlist them into various types of games. Many patients could not be enlisted by oral methods but if escorted to the field and the ball thrown to them would catch it and from that preliminary stage could be inducted into the game. Other types would respond only to particular attendants or patients for whom they had some close attachment. Others would respond only for the physical director and not the attendants. Day after day, the procedure was followed and attempt was made to place various patients into positions in games in which they could show their best ability and gain the most wholesome recognition. After this procedure was carried on from day to day, it was noted that the chief difficulty in inducting the functional patients into various types of games and various exercises was not the lack of muscular skill and dexterity but that the main difficulty was the social strain which the patients felt in accommodating themselves to types of activity in which they must sublimate anti-social traits and cooperate as part of the social group. It was this natural evolution which resulted in an understanding of this therapeutic experiment as basically a social experience. We were simply groping about in an attempt to utilize this activity which appeared to have such potentialities in mental rehabilitation.

The first three years developed many difficulties in the organization of such a program. There were failures because of lack of appreciation of the functional and organic factors involved; for example, the organic cases were inducted into complex games such as baseball and volley ball and pitted against the functional types. The fundamental difference in the capacity of these two types for coordinated activity becomes increasingly evident. Then there was difficulty in establishing a faith in the fairness of the competitive types of exercise. Patients were frequently critical of the umpires and distrustful of the scores and other athletic records. Many would not play if certain people umpired. There was also considerable difficulty in fitting the physical program into the general routine of the hospital. In those early days, physical education was considered by many as a usurper of the regular routine of the hospital and something that should be done after everything else. It was necessary to educate the personnel as well as the patients and it was a number of years before it was accepted as a therapeutic adjuvant and given respectability, and the impor-

tance of other therapeutic agencies. The next difficulty arose from the failure to project physical education as a psychotherapeutic procedure. There was the early tendency to emphasize skills and motor accomplishment rather than the social readjustment of the patient.

As time went on, however, many of these mistakes became rectified through gradual re-orientation of both the patient and the hospital personnel. It soon became evident that physical education to be effective as a treatment of the mentally ill should be presented as physical therapy and that it could only justify itself as a psychotherapeutic measure. In 1923, to get back to the beginning of the physical program at Perry Point, there was one patient team of which the medical officer in charge was justly proud. The genial and understanding personality of the reconstruction officer and his insight into the psychotherapeutic possibilities of such team organization was of primary value. It was considered to be a great honor to be selected as a member of this team and it included only the most alert patients. Games were played between this team and outside teams with honors of winning about equally divided. At this time there were few patients who played tennis. A small calisthenic class was organized as was also a volley ball team which played outdoors. In 1925, the recreation building was completed and the medical officer in charge turned this building over to the physical department to be used for indoor exercises and games as well as entertainments. This marked an epoch in physical education at Perry Point, as opportunity was provided in this building for indoor volley ball, indoor calisthenics and gymnastics, bowling and swimming. An athletic program was then organized to include volley ball, bowling, calisthenics, indoor athletic meets and pool and billiards for the less active patients. Two attendants were assigned to the physical director and with their help a double tennis court was constructed and a baseball field lined up. With these increased facilities there remained the important problem of enlistment of patients into these activities. Experience had taught the vital necessity of projecting these activities as media for social readjustment and the social concept became increasingly stressed. Attempts were made to provide friendly wholesome contacts with large groups of patients through smokers, athletic discussions, athletic parties, etc. At this stage, it also

became evident that the physical director could not successfully contact large groups of patients in a close personal relationship. It appeared advisable to organize patients in units with responsible leaders and project the program from the clinical director to the physical director and hence to the unit leaders. It was found most practical to appoint attendants as these unit leaders. The utilization of attendants in this way resulted in larger groups taking part and it had many advantages and some disadvantages. In cases in which attendants could be secured with therapeutic insight the plan worked favorably. In other cases in which the attendant viewed the game as simply a medium of winning or losing, results were unsatisfactory. To correct this, attempts were made to teach the attendants the fundamental underlying principles of utilizing play experience as means of social re-orientation in which emphasis was given for leadership and effort and sublimation of unwholesome qualities in a game rather than the exclusive objective of winning or development of dexterity and skill.

As the program developed, it appeared that a wide range of physical activities might provide a distinctive motivation which would enlist many resistive types. The situation was studied from the standpoint of providing such motivation and in carrying out this objective, an intensive program of publicity which would give recognition for effort, leadership, sublimation of unwholesome behavior and the gradual approach to higher levels of social striving was attempted. Patients were encouraged to submit articles stating their ideas about various games, including suggestions as to techniques, interpretation of rules, spirit of the game, sportsmanship, etc. The write-ups were included in the Perry Point Bulletin and a special issue of the Bulletin in August, 1929, was devoted to physical education. This included 49 pictures of patients and personnel in various types of physical education and articles by both patients and personnel. At that time the activities consisted of calisthenics, gymnastics, volley ball, baseball, tennis, bowling, swimming and free-play activities.

Such publicity created considerable interest among the patients and it was a common occurrence for a patient to bring records of his athletic performance, athletic notes, and suggestions for publication. The surprising interest resulting from such forms of recognition led to the consideration of some system whereby public

recognition could be given to the patients who did the best in the physical activities and the outgrowth of this idea resulted in an annual award day at which special recognition was given in the form of diplomas to members of championship teams and medals to the patients who made the highest individual records. The first annual award day exercise was held in 1931. Noted speakers were in attendance and the hospital, with the assistance of ex-service organizations, arranged a program consisting of a detailed account of every patient's record, a report of the progress of the various teams and particularly the many elements of social readjustment which were produced through these activities. These annual award days have become a most important part of the hospital routine and have been carried out regularly from that time, making a vital contribution to a better morale. Each year additional activities have been added and the system of compiling averages and evaluating results has been revised. In 1929, the physical director in order to afford every patient an opportunity to participate in interesting and wholesome activity, interviewed all newly admitted patients relative to their early play experience, capacity, preferences and interest and over a period of seven years a thousand records were secured and utilized as most valuable material for determining the program content and methods. At this time also the behavior reaction of hundreds of patients was noted and individual records of their activity over a period of months was kept. These records proved of considerable assistance in establishing better rapport with the patient. From the results of our experience up to this time it was becoming increasingly evident that physical education to reach its greatest area of usefulness must emphasize mass therapy while also utilizing the endeavor approach. Whole wards were inducted into calisthenics and a group of 360 patients were given mass calisthenics and pictures taken demonstrating the remarkable fact that such a large group of psychotic patients could be inducted into simultaneous exercise. The emphasis of the program from this time on was placed on the large group rather than the individual, although it was recognized that the individualization is most important and has its place in the initial approach and therapeutic study of the patient.

In connection with the utilization of publicity as motivation for the patients, it became apparent that the attendant who occupies

such a close relationship to the patient should have a working knowledge of the fundamental psychotherapeutic principles involved in the utilization of physical education as therapy. Lectures were given for this purpose. These lectures included a statement of the fundamental importance of utilizing physical education as a resocializing agency rather than a means of physical readjustment, suggestions as to methods, approaches, handling, types of calisthenics for three grades of patients, the alert, moderately active, and inactive. A much better therapeutic insight was demonstrated by the attendants in their contacts with the patients as the result of these lectures.

At this stage of our experience another most significant condition became apparent. It was noted that whereas many patients would make the initial participation in some particular form of game and would find at the outset an interest sufficiently strong to inhibit distractions in the form of delusions and hallucinations that as time went on and they became more accustomed to the game they might play it in a listless manner and their delusions and hallucinations might return to upset the normal functioning of their play. This situation led to a study of the various types of activities from the standpoint of substituting extraverting elements and grading them for progressive order. A baseball league was formed and patients were graduated from the intra-ward contests to the first ward team and hence to the all-star team, which competed with strong outside organizations. As soon as this progressive system was provided for baseball players, the effectiveness was apparent and efforts were made to provide such stimuli for other activities. A most significant result accrued from this. As soon as patients became inducted into the form of exercise in which they could realize improvement and graduation to higher levels, a strong interest seemed to develop and whereas they might be vacillating in other respects and other relationships, they would find a strong and relatively permanent interest in these activities. It was most unusual for a patient who was enlisted in this manner in the league to lose his interest and discontinue the game.

Another important situation developed at this time. Noting the unusual interest which large groups of patients took in physical exercise, there developed a tendency on the part of a few to consider this as a sort of end in itself and make excuses to avoid

regular routine in the hospital for the purpose of engaging in athletics. The physical director made a special effort to overcome this and to organize a program so as to merge into the regular routine of the hospital without disrupting or disorganizing other essential activities. At the same time an effort was made to project the physical program as a regular part of the hospital organization and not as something to be done after everything else was completed and the results showed that it was gradually finding its place as an integral part of the hospital administration. The next difficult problem arose in connection with the proper relationship of physical education to other forms of therapy, particularly occupational therapy. Physical education was gradually being received as a responsible part of the therapeutic Trinity of work, rest and recreation, although there were many problems in the actual application of this principle which remained unsolved.

During the last two years a special effort has been made to more effectively coordinate the program of recreational therapy with that of the occupational activities. It had been found advantageous to utilize the various occupational units as a basis for team and league organizations. This extension of the recreational program has been aimed to create a feeling of team work and cooperation among the patient members of the various details and the results along this line were very gratifying. Details have organized their patients into bowling, soft ball and shuffleboard leagues. This participation has been organized upon the basis of league set-ups, each detail having a definite time to report for their regular recreational activities. The activities for the patients have been modified so as to meet their distinctive levels of interest and capacity, the more alert playing on the soft ball team and the others taking part in the less complex activities, such as bowling and shuffleboard. These detail groups look forward with considerable pleasure to their regular participation in these activities and a spirit of social understanding and increased cooperation has characterized the project.

The Acute Service.

Every hospital for the treatment of mental and nervous disease is faced by a difficult problem of the most effective care and treatment for hyperactive cases of combative and assaultive tendencies.

These potentially dangerous types are segregated in the acute service of the Veterans Administration Facility at Perry Point. The conventional treatment of hydrotherapy has been used for many years and while its distinct efficacy as a sedative adjuvant was recognized, it was deemed advisable to attempt a program of a more socializing character to stimulate these patients to enlarge their interests by doing something themselves rather than subject them exclusively to physiotherapy measures. To this end in addition to other occupational activities, an intensive program of physical therapy was organized and carried out.

Résumé of Results of Physical Therapy for the Acute Service.

A wide category of both formal and informal activities has been attempted for the acute service, including calisthenics, swimming, gymnastics, soft ball, volley ball, impromptu games, such as medicine ball circle passing. Of the 40 patients on the ward, ranging in chronological age from 35 to 40, 38 participate in some form of physical therapy, 20 take part in forms of social play while 18 confine their activities to the more mechanical and individual types of exercise, such as swimming and calisthenics, 18 play in the soft ball league. A very definite line of social demarcation is observed between the groups who are able to find elements of extraversion in their play experiences and those who retain their anti-social predominating traits. Eleven patients appear to show some improvement in their social readjustment as evidenced by some of the following: (a) Greater modifiability to social practices; (b) increased range of interest; (c) greater control of emotional field; (d) more cooperativeness; (e) more insight; (f) brighter attitude; (g) increased range of physical activity; (h) stronger volition. The primary motif for these acute cases was to provide sedative and relaxing modifications through exercise.

In observing these patients as they take part in a league soft ball game, for example, one is impressed with the high degree of concentration and the surprisingly intense effort attained. Many apathetic patients will make an unusual effort while playing these games. Very regressed patients are frequently seen backing up plays, demonstrating an acute though spasmodic orientation in the

game. Others are observed to show evidence of appreciation for the good plays of their comrades as well as to criticize faulty plays. The sublimation of antisocial behavior is most evident. In a recent game, a player of the acute ward collided violently with another player, a combative type. It was expected that he would become emotionally upset and assaultive. To the surprise of all, he arose, walked over to the other player, said he was sorry and asked if he was hurt.

The head ward attendant, who is manager of the team, makes these comments: "When the soft ball league started, a number of patients did not want to play. Now they ask to play, are more contented on the ward, discuss games at length on the ward; they work better, there have been no fights or arguments while these men have been playing in the league. Patients look forward to the regular weekly write-ups of their league games in the station periodical."

A natural result of this interest in the soft ball league led to casual gains in hard ball. These impromptu games brought out a number of patients from the acute service who showed not only skill but seemed able to sublimate their anti-social and aggressive tendencies in this game. A team from this group was selected to enter the patients' senior league. It was a most significant fact that these patients, who were difficult to handle on the ward, presented no serious behavior problems whatever when playing. The nurses' reports indicated that they were much quieter and more cooperative on the ward.

Résumé of Results of Physical Therapy for Suicidal Group.

An intensive program of physical therapy was attempted for the suicidal type of patients. There were 35 in this group, ranging in chronological age from 35 to 40 years. Sixteen took part in formal types of exercise, such as swimming, calisthenics and gymnastics and seventeen took part in informal types of exercise, including soft ball, baseball, tennis, bowling, volley ball, code ball. Two did not take part in physical therapy, because of confused, stuporous condition resulting in a disorientation towards these types of activities. During the past six months, eight have been transferred to other wards as a result of their improvement. The objective for these

suicidal cases was to re-establish spirit and morale and this type of therapy seemed admirably suited for the purpose when considered as a long-range activity. At the beginning many were antagonistic toward efforts to enlist them into any form of exercise. They were resentful because of their restriction and did not participate. As leagues became organized and many types of activity included in the general program some of these patients would request to take part and at present writing all but two of these types will take part in calisthenic exercises, many of them showing real interest and spirit, a number have been transferred to another ward and now making a satisfactory adjustment to the more responsible procedures of work.

In observing the conduct of many of these patients over a period of two years it appears that the confidence and satisfaction developed through their play activity has become enlarged until it touches and colors many other relationships. Whereas it was necessary formerly to keep these patients locked up and under the closest supervision, they now enjoy considerable freedom, they come to the recreation building daily for exercise, are most cooperative and amenable and appear to have improved in desirable social striving and morale.

The following cases of individual patients and unit organizations are offered since they are illustrative of general reactions to these forms of therapy:

Patient X.

Psychosis, Manic Depressive, Depressed Type.

When this patient first came to the hospital a number of years ago, he had little interest in athletics. Coming to the Recreation Building regularly with the ward, he engaged in calisthenic exercises in a spiritless manner. One day he was prevailed upon to go on the handball court. The Physical Director played ball back and forth from the court, the patient from time to time hit the ball weakly as it came near him. From this beginning he gradually developed a slight interest in the game and would play, extending but little effort and complaining of his physical weakness, asserting that he was inferior, bewailing the fact that he did not kill himself when he attempted suicide, giving expression to feelings of inadequacy and asserting that he was sexually impotent. He would play with flashes of effort and relapse into what appeared to be apathy, often adopting mannerisms and stereotyped positions, especially folding his hands and dropping his head, assuming a

posture of inferiority. The first year he played handball in a listless, apathetic manner but there was noted cycles of greater persistence. He would often finish a game instead of stopping as he had in the beginning. The second year, the Physical Director made it a point to play with him regularly. Patient showed marked improvement in his motor responses, playing a more accurate and much faster game, and showed at times a concentration and some evidence of planning his play. He insisted all the while, however, that he could never hold his own against even a poor handball player and he complained of a feeling of inferiority, lack of physical stamina and asserted that he had been found wanting and would not be accepted in society any more. The third year showed a most remarkable change in this patient's personality; he improved in skill until he could hold his own and often times defeated the Physical Director. This capacity which he was able to assert seemed to stabilize his conduct, to give him a feeling of reassurance and confidence and to open a more optimistic perspective. Volitionally, he appeared strengthened and emotionally better oriented. He spoke of the fact that he was being received again in social groups, that his ward surgeon had taken him with him on trips to Baltimore and that he had made arrangements to go to Philadelphia to visit his sister, with the permission of the ward surgeon. He then became interested in baseball; went to Philadelphia to see the Philadelphia Athletics play. He also became interested in playing on his ward team and became one of the best players in the league. While at times his old ideas and emotions of incapacity came to the surface, on the whole he was much better balanced.

His mannerisms were not evident upon the athletic field; his hesitancy and indecision were less apparent. He enlarged his activities to include tennis and bowling, began to mix with other patients and members of the community, became a most likeable personality and was able to make acceptable social contacts. He continued to play handball and became very much interested in the weekly handball tournaments, in which he defeated strong patient and personnel players. This record made him eligible for the finals in which he eliminated all other contestants and became the undisputable champion of Perry Point. Upon Annual Awards Day, when he was awarded the medal for championship, he was perceptibly pleased and said that he felt that he was on his feet once again. Upon his discharge from this hospital, one of the first things he did was to joint a Y. M. C. A. and now engages regularly in handball and holds his own against some of the strongest outside players. He frequently writes to the Physical Director telling him of his scores in the Y. M. C. A. tournaments and seems to be getting along well and making a living at the present time.

Patient Y.

Dementia Præcox, Paranoid type.

This patient was a dilapidated and apparently far regressed præcox. Slovenly in dress and careless in other habits, he could be seen frequently

barefooted walking with restricted hysteriform movement and pushing himself around with a long stick. Watching others play croquet one day, he asserted that he could play this game himself and commenced playing. From the beginning it was evident he had much natural aptitude. He advanced from playing with his ward companions to the weekly tournaments which he won frequently. He continued to improve in this game until he won the hospital championship and the medal for croquet champion on Annual Award Day. Believing that he had more than ordinary ability in this game, he was entered into the eliminations for the championship of the State of Maryland. In competition with the best players in the state, he won the state championship and the cup donated by the Baltimore News-Post. He retained this in the following year and at present is acknowledged to be the best player in the state. His improvement in social adjustment is apparent. He is much more careful in his dress, talks in more rational channels, carries out a detail on the ward, and teaches many patients and encourages them to take up the game. His enhanced sense of self respect is commented upon by many.

Ward 4 Baseball Team.

A most interesting example of the tendency of league organization to bring patients together in the establishment of a higher morale and a better social adjustment is to be found in the experience of Ward 4 baseball team. These boys had only a mediocre team and were at the tail end of the league for the first half of 1933. During the second half, however, their head attendant, who has a good grounding in baseball, assumed charge of the team, gave the team systematic practice, brought out their strong points and made the boys feel that they really had a team which could compete successfully in the league. The attendant established a warm contact with the patients because of his self-sacrificing attitude, often times playing with the boys on the day he was off duty. After being at the tail end for the first half of the league, these boys came back and won the second half by the close margin of one game. It then became necessary for them to play the Convalescent team, winners of the first half. Ward 4 lost the first game 6 to 1; they won the second game, 16 to 6 and playing one of the best games of the season won the third and deciding game, 3 to 2, thereby annexing the championship for 1933. The feeling of comradeship which developed among the members of the team was most noticeable. There was a spirit of helpfulness, which one does not find often in the intramural relationships of psychotic patients. Those better acquainted with the game became stimulated to make suggestions, this was particularly noticeable, as a patient would make constructive suggestions, as to the arrangements of players, various plays, etc. Some of the introverted patients could be seen discussing plays and encouraging others to do their best. The team became alert as to the chances of winning and losing and would evaluate these situations with shrewd discernment. One most actively hallucinated patient, would rarely react to hallucinations while playing. Visitors, when observing the team in action,

would frequently comment upon the lack of abnormal behavior. Encouragement and discerning comments of the nurse and ward surgeon contributed much to create a sense of capacity as well as realization of accomplishment as this team worked its way to the championship.

PRESENT SET-UP OF PHYSICAL THERAPY IN VETERANS' ADMINISTRATION FACILITY, PERRY POINT, MD.

INFORMAL ACTIVITIES.

Description.	No. of Participants.	Form of Organization. League Organizations.
Baseball	112	6 team league
Volley ball	92	8 team league
Playground baseball	60	4 team league
Bowling ten pins	150	2-8 team leagues
Bowling duckpins	168	2 leagues
Shuffleboard	180	17-teams—1 league
		Inter- and Intra-Ward Organizations.
Code ball	48	Daily games and weekly tournament
Bowling duckpins	24	Daily games and weekly tournament
Golf	32	Daily games and weekly tournament
Croquet	49	Daily games and weekly tournament
Handball	16	Daily games and weekly tournament
Tennis	20	Daily games and weekly tournament
Pool	78	Daily events
Field day events	200	Activities held for special occasions, holidays, etc.

FORMAL ACTIVITIES.

Calisthenics	500	Three types as to complexity for (1) active; (2) moderately active; (3) inactive type
Gymnastics	112	Stunts in individual exercise

NOTE.—Many patients participate in more than one activity.

PRESENT SET-UP.

The present set-up at the Veterans' Administration Facility, Perry Point, includes a wide category of formal and informal activities. Under informal activities are provided regulation baseball, regulation volley ball, playground baseball, duck pin and ten pin bowling, code ball, tether ball, golf, croquet, hand ball, tennis, shuffleboard, pocket billiards and impromptu games. Under formal

activities are included calisthenics, gymnastics and swimming. Attempt is made to provide a suitable type and grade of activity for the patient's mental, physical and social tolerance. The idea has been to progress the program from the fundamental therapeutic point of interest to effort and in carrying out this plan the aim has been to make the patient feel that the activity is self-imposed and purely voluntary, play is not considered as an end in itself but as an important part of the therapeutic régime of work, rest and recreation. Many patients who will not work, will play and the aim has been to progress these patients from the playful aspects of play to the more responsible forms of work. The attitudinal value of play has been stressed and it has been found effective in getting the patient into more cooperative and responsible adjustment to other hospital procedures. Attempts have been made to differentiate between the many types of physical activity for the purpose of prescribing activities to the more specific needs of the individual. The many distinctive and favorable psychotherapeutic situations which these activities so naturally develop have been observed and attempts have been made to utilize them as media of resocialization. To this end the relational components of games, such as records, athletic discussions and write-ups have been emphasized and efforts have been made along these lines to assist the patient in the relationship of these play activities as to enlargement of his interests and activities into other channels. In order to enlist a large number of patients, it is realized that the program should project from the medical officer in charge to the clinical director to the physical director, hence to the attendants who should be taught to administer the routine exercises. This has been done with the result that the attendants, as well as the patients, have become interested in the program. They have been made managers of teams and have taken considerable interest and pride in their ability to coordinate and unify the patients' interests. There is no doubt but that many therapeutic adjuvants fail to function effectively for the reason that no worthwhile incentives are offered the patient and he is unable to perceive steps of improvement. The physical therapeutic program has been organized so as to afford discernible and worthwhile steps of progression. Patients are encouraged to advance from individual play to ward teams and hence to unit and all-star teams; for example, a patient may first

play baseball in a game between two teams of the same ward, he may then advance to his ward team which plays in the league, and from that team to the first team which plays outside teams. These elements of potential progression serve to vitalize the program. A special attempt has been to assist the chronic cases who will probably spend most of their life in an institution to find some stabilizing interest in some play activity and many patients of this type are thus enabled to make a much more satisfactory adjustment to the hospital environment. For the most hopeful cases, the objective has been to relate their play to the more responsible procedures and activities so as to assist them to leave the hospital. Many patients who have been discharged have written to the hospital commenting upon the value of the games learned during their hospitalization, enabling them to make more agreeable social contacts outside.

While it is felt that a highly diversified program of physical activities contains many possibilities for more effective therapy, the actual carrying out of such a program demonstrates the necessity for a more studious application. Physical therapy can only justify itself as a psychotherapeutic procedure and a trend of all physical programs in mental hospitals is toward a realization of that objective. This involves a better liason between the psychiatrist and the physical director, as well as an intensive course of study for the attendants who will administer the routine exercises.

A more specific differentiation should be made between the automatic and the voluntary responses in physical therapy, a more careful and studied provision for progressive order in therapy, a careful study of types for whom motivation should be emphasized and those for whom organic factors circumscribe their sphere of therapeutic activities. Long and short-range activities should be more specifically provided for various types. A more careful study should be made of the possibility of physical therapy in enlisting resistive and antagonistic types into purposive activity, as well as its effectiveness in providing therapeutic motivation for disinterested types and relaxing and sedative benefits for hyperactive patients and inspiriting morale for suicidal cases.

The results of years of experience appear to indicate the importance of adjusting the activities and benefits more specifically to the different disease entities. The mistake is often made in

inducting patients into activities simply because they are willing, or because they have some special preference for a particular type of activity. For example, the dementia paralytica cases are willing to engage in almost any activity of a play nature and require little motivation. It became evident from our experience at the Veterans' Administration Facility, Perry Point, Maryland, that the problem in these cases was not the establishment of strong motivation, but to assist the patient to enter into forms of activity best suited to his capacity for coordinated action. While these patients will engage gladly in volley ball and baseball, records and personal observation seem to confirm the conclusion that such complex activities are not suited for these organic types. In competition with the functional group, they show most faulty discoordination followed by unfavorable psychic reactions. In activities of a simpler, one-response activity, they are able to show satisfactory coordination and to gain wholesome psychic satisfaction. The functional cases, especially the præcox groups, appear to present a problem of motivation and the therapeutic aim appeared to center about the weaning of the patient from his field of fantasy into constructive forms of behavior. These cases appear able to participate in many complex forms of games, once their interest is aroused and their attention sustained.

The following table of suggested activity for more specific application of disease entities may be found of value. This table is but tentative and subject to a widely elastic range of application. Types of activities suitable for the distinctive needs of the organic cases, for example, may also be utilized for the more alert functional individuals. It appears advisable, however, to utilize the more complex forms for the latter so as to stimulate them by providing requirements more in harmony with their higher capacity level.

TABLE OF SUGGESTIVE ACTIVITIES FOR MORE SPECIFIC APPLICATION TO DISEASE ENTITIES.

INFORMAL ACTIVITIES.

Description.	Disease Entities.
Baseball	For functional types particularly dementia præcox cases and manic depressive cases.
Volley ball	For functional types particularly dementia præcox and manic depressive cases.

Description.	Disease Entities.
Playground baseball	For both functional and organic cases, particularly regressed types of dementia præcox cases and the more alert organic types such as epileptics, encephalitis lethargica cases and dementia paralytica group who have retained control of fundamental movements.
Bowling ten pins.....	For both functional and organic cases but particularly the organic types sufficiently strong to engage in this activity, especially indicated for organic cases who are able to do best in a one-response type of activity, such as the encephalitis lethargica cases, dementia paralytica and epileptic cases.
Code ball	For organic cases requiring a one-response activity, particularly the epileptic and encephalitis lethargica cases of the more alert categories.
Bowling duck pins.....	For organic cases who require a non-strenuous type of one-response activity, such as deteriorated epileptic, dementia paralytica and encephalitis lethargica cases.
Golf	For both organic and inorganic cases. An important type of activity serving as transitional activity to higher levels. One response activity, for those organic cases who can realize steps of motor improvement.
Croquet	For both functional and organic cases requiring a convalescent type of exercise.
Hand ball	A one response type of activity for both functional and organic cases who are able to participate in a strenuous form of exercise.
Tennis	A combination of one and many response activity suitable for all types of the more alert categories; a valuable transitional form of exercise for the patient who is able to advance from one response to many response play.
Pocket billiards	A one response type of activity for both the organic and functional types who require a non-strenuous form of exercise.
Swimming	For both functional and organic types, but particularly negativistic and resistive types who require a strong psychic motivation.
Field day events.....	Organized and graduated to meet specific requirements for all types.

FORMAL ACTIVITIES.

Description.	Disease Entities.
Calisthenics	Mechanical forms of activity, especially adapted for those who cannot enter into more socialized game projects.
Gymnastics	(a) Exercise apparatus for postural, structural or functional readjustment. (b) Mechanical forms of exercise for those who cannot enter into more socializing experiences.
Swimming	(a) For both fundamental and organic types as a general conditioning exercise. (b) As an aid in differential diagnosis, of distortion or restriction of movements due to hysteria or organic causes. (c) As a therapeutic aid in overcoming negativism.

While the simple types of activities such as code ball may be used for both functional and organic cases, it will be found more distinctly effective for the types who cannot engage in complex forms.

Conclusion.

In conclusion, the writers do not wish to create the impression that physical therapy has been viewed as a panacea. Its many limitations, both as to content and methods have been apparent in attempts to utilize many and diversified activities nor has physical therapy been employed at this Facility as a purely independent adjuvant. The ideal therapeutic program advocated and carried out has represented a balanced regimen of work, rest and recreation. Attempts are constantly being made to impress patients with the importance of progressing from play activity to the more responsible rôle typified by work so that they may be assisted in their return to normal responsibility. The importance of utilizing play mechanisms as a distinctive means of elevating the patient's social respect in the direction of more easeful and confident social relationships, which will assist the individual to resume his place in normal society, is being constantly stressed. The physical program has been organized so as to fit most effectively and cooperatively into the regular hospital routine. It is hoped that these observations may lead to further study of the possibilities of this adjuvant in the promotion of more effective rehabilitative methods.

TOTAL PUSH METHOD.

III. Schema for the Recording of Certain Important Attitudes in Chronic Schizophrenia.*

By ABRAHAM MYERSON, M. D., Boston.

In selecting cases of chronic schizophrenia for treatment by what I have called the "total push method," certain objective and apparently fundamental characteristics of these patients appear so conspicuously as to warrant the establishment of definite tests for their elicitation and recording. The purpose of these tests is, first, to enrich the present mental examination of patients with objective phenomena of a behavioristic type and by tracing these backward and forward into the total life history of the individuals to differentiate, if possible, between groups of schizophrenics in a more satisfactory manner than that represented by the present day classification. The second purpose is to establish, if possible, prognostic signs which may be used in relationship to the success or failure of any system of therapeutics, and especially of the total push method. These symptoms are as old as psychiatric observation. Their systematic organization, however, and interpretation gives, I believe, a new emphasis and a reorientation of our efforts to understand and to treat the chronic schizophrenic.

The most prominent attitude of the chronic schizophrenic is his retreat from social relationship, in more general terms, his retreat from organized activity and environmental interrelationship. In most disease there is retreat; the man with the broken leg has perforce retreated from activity, but the social sense and attitudes of the individual remain undisturbed. The individual with a severe neurosis retreats from activity and social contact, but this retreat is mainly on the basis of incapacity and a feeling of distress in the presence of others. There is a painful subjectivism, accounted for on one basis or another, which presents itself in increased heartbeat, flushing, gastrointestinal disturbance, and in what I have called

* From the division of psychiatric research, Boston State Hospital, Boston, Mass., aided by grants from the Commonwealth of Massachusetts and the Rockefeller Foundation.

increased vaso-visceral reverberation. The deeply depressed individual, of whatever type, retreats because he has no interest in his fellows and his mood finds nothing pleasurable. There is, therefore, nothing peculiar in the mere existence of the social retreat of the schizophrenic. It is its quality and the general symptoms which accompany this retreat which are of interest and of specific importance.

The three leading sets of symptoms which I wish to discuss relate (1) to those signs which indicate social retreat, (2) those relating to the polarity of passivity and resistance, and (3) those which betoken an endogenous repetitive excitement in stereotypies of flow of words and/or of movements. These signs and symptoms may be so recorded as to give a profile chart of the status of the individual, and the reaction to treatment can be more definitely established than by most of the other signs and symptoms of schizophrenia.

Signs of Social Retreat.

(1) The aversion complex: the eyes form the main basis of contact of individuals with one another and, in fact, with living things to their environment, whatever form that environment may take. Hearing is episodic; touch can only be carried out under relatively few circumstances and acts as a sort of clincher of contact. Smell and the other modalities of sensation have not as great value to man as vision. The face-to-face association is the essential of social contact, and in this the eyes play the orienting and leading rôle. The chronic schizophrenic may and often does present the following signs: he stands in a sort of flexor attitude with head bowed and with eyes turned away from the examiner or from his fellows. He rarely looks directly at them. Often if one steps into the direct range of vision of such an individual, he turns his eyes to one side or the other, according into which field of vision the examiner intrudes. The direction of the gaze and the capacity to meet the eyes of the examiner form, therefore, an important objective sign. Does the patient look at his vis-a-vis or at others? If his gaze is arrested, does he or does he not turn his eyes away deliberately or reflexly, whatever the mechanism may be? In association with this, an interesting eye sign may be recorded. If the brow or the bridge of the nose is tapped

lightly with the finger, in many cases of chronic schizophrenia there is a repetitive eye closing. If the fingers of the examiner are thrust as if towards the eyes of the patient, there will take place a spasmodic closure or a rapid fluttering of lids. These eye signs are common in what is called the catatonic præcox.

(2) Hand shake: a reflex social contact, firmly established by usage, is the shaking of hands. Ordinarily, if one thrusts one hand towards a person, he clasps it firmly and shakes the hand up and down for a short period of time. This is markedly deficient in chronic schizophrenics and may show the following defects: first, no response whatever is made; second, after a pause the hand of the patient comes up and touches the hand of the examiner, then withdraws; third, a very light clasp may be given; fourth, a few up and down movements may be made. This hand shake thus can be recorded on a definite scale.

(3) Communication: verbal communication is, next to face to face association, the most important social human contact. A few questions form the basis of the greeting complex. Ordinarily, if one asks any individual what his name is, he responds and may in turn ask the name of the questioner. The reaction to this simple question, "What is your name?" may be graded from zero up. If no response is given, it is zero; if a low-pitched hesitant reply is evoked, it may be called one; if the reply is firmly given with some evidence of responsiveness, it may be graded as 2, and so forth, until we reach the point where the reply is adequate with a responsive look and very possibly some added phrase to indicate the social relationship is established. Similarly with the question, "How do you feel?" and the question "How do you like it here?" An adequate reply to this question is "It's all right but I prefer to be home" or some other response of developed type. The mere statement "All right" can be classed rather low down on the scale.

In the chronic schizophrenic the answers to these questions are, so to speak, extorted by the questioner. The answers, if given, are terse and without amplification. The reply to the question, "How do you feel?" may be vividly contrasted with the reply given by the neurotic. A chronic schizophrenic usually says "All right," if he replies at all. The neurotic issues his appeal for help in the form of a long answer.

(4) Smile: an important and primitive social response is the smile. It may be merely the stereotyped smile of recognition of introduction or of meeting. There is an uneasiness in all human contact which becomes bridged over by greetings and smiles. In the case of the chronic schizophrenic, there is rarely a smile of greeting. When there is one, it may be of brief duration and more like a grimace than a smile. In some individuals, the smile becomes excessive and is really a symbol of what will be later discussed under the heading of passivity. It is more like a propitiatory gesture than a manifestation of any pleasure or recognition.

(5) Handkerchief or ball test: one of the forms of social contact is "playing the game" with others, and of the instruments used for "the game" the most common one is the ball. Tennis, baseball, cricket, hockey, pool, billiards and almost every other organized game involves the use of the ball. Just as the wheel is the symbol of transportation, so the ball is the symbol of the game. In testing this out, I use a rolled up handkerchief and without saying anything toss it at the patient. Normally, the quick reflex response is to catch it; the individual usually smiles and perhaps after a preliminary look at the handkerchief throws it back. The range of response in the schizophrenic is as follows: the resistant patient receives the impact and does nothing. He will not pick it up, will not throw it back, does not cooperate or play the game at all. Some patients, after receiving the impact once or twice catch the handkerchief and then gradually learn to throw it back. In some cases the response is rather startling in its evocation of normality. The patient may seem very much interested, smile and throw the ball back with animation and pleasure. I have found this a very valuable index of sociality and also, I believe, it gives a hint as to the type of cooperation the patient will give in his retraining.

While these tests might be elaborated indefinitely, I have seen fit for purposes of terseness and quick orientation to limit social contact to these phases of human interrelationship, to wit, the meeting of eyes and the aversion complex generally, the hand shake complex, the reply to the elementary questions of social relationship, the presence or absence of various types of smiles, and the reaction to the handkerchief or ball test.

Polarity of Passivity and Resistance.

Social psychologists recognize as polarities the rapid shifting of attitudes from one extreme to the other. Thus, there is the polarity of dominance and submission which has been greatly stressed in the social psychological literature. In the case of the chronic schizophrenic, signs of the polarity of passivity and resistance have been given formidable names which, however, are merely descriptive and have not been closely linked up with the underlying attitudes of passivity and resistance. Thus, cerea flexibilitas is described as a symptom; it is really the extreme of passivity. Negativism is mainly resistance, usually of a limited and immediate nature in that it does not express itself by a coordinated and purposeful series of acts but represents rather a resentment to intrusion upon the retreat. Echopraxia and echolalia have also been adequately described from the beginning of psychiatry. They are, on the whole, expressions of passivity and represent the willing, unthinking, uncritical assumption of alien attitudes and words. Echopraxia and echolalia are really imitativeness of the non-purposive type unless the purpose be inferred as submission.

A. The tests for passivity are as follows:

(1) Posture passivity: does the patient assume and maintain whatever posture he is passively put into? The degree of passivity may be measured by the duration of the maintenance of the attitude into which the patient is put and also by the grotesqueness and irrelevance of the attitude. Thus, if the head is twisted to one side and maintained in this position, and if one leg is lifted, and the patient maintains this position, he expresses an extreme passivity. If he merely holds a hand up and then quickly or gradually puts it down, this is passivity also but of much inferior degree.

(2) Imitation: does the patient imitate spontaneously the attitudes of the examiner as he stands before him? Thus, if the examiner spreads apart his arms, does the patient after a brief pause or immediately imitate this motion? If the examiner assumes a grotesque motion deliberately, does the patient follow this suggestion into realization?

(3) Commands: a third phase of passivity is the obedience to commands sharply given as if drilling a soldier. This obedience is

part of the passivity of the individual. It may be extreme; it may not exist at all.

It will be seen that many other tests could be elaborated for passivity, but I believe that posture passivity, the imitativeness of postures and words, and obedience to commands may be placed on a scale which will afford sufficient objective criteria to establish the degree of passivity of the individual.

B. *Resistance.*—Resistance is, practically speaking, measured by the tests for passivity but yields opposite results. The individual is usually averted. In the extreme case he resists everything that is done by an opposing motion. In the minor forms, he resists but does not put into play the opposing mechanisms. The foregoing is true of postural resistance, communication, hand-shaking, smiling and handkerchief throwing. Occasionally a positive hostility is evoked by repeated intrusion upon the patient's resistance. Rarely does this, in the cases here discussed, take the form of a planful organized hostility. It is more like the fighting back of the cornered animal in whom hostility subsides as soon as aggression on the part of others ceases.

Endogenous Automatic Excitement.

The third group of symptoms is here called endogenous automatic excitement, as manifested by the familiar repetitive stereotypies of motion and speech. This group of symptoms seems to bear no relationship to either passivity or resistance, being present in patients who are very passive and obedient and also in patients who are very resistive and hostile. The verbal range may be from an occasional interjected muttering to a continuous repetitive stream of stereotyped phrases and sentences. The motor automatisms range from a grimace of the face, one of the familiar ones being the so-called *schnaut krampf,* to tics of all parts of the body, carried out with great rapidity and power and forming an almost continuous activity of the patient.

These, it seems to me, are recordable accurately and may be placed under two general headings, (1) those of speech, and (2) those of motion. In respect to speech, only a roughly approximate record may be made. In respect to motion, the parts of the body involved, the rapidity, the force and the chronicity can be more

accurately stated. A motion picture might definitely give us an adequate measure of this phenomenon.

Another approach to the measurement of the endogenous automatic activity is its resistance against distraction. In some patients, this activity almost ceases when the patient is talked to and examined, and especially if he is engaged in physical activity, games, etc. On the other hand, there are patients who continue to manifest their endogenous automatic excitement even while under examination and, to a certain extent, when participating in games. It has seemed to me, although this is without direct corroboration, that in the extreme form, this type of schizophrenic resists therapy of the total push type very markedly.

RECORD OF TESTS.

I. *Social Contact:*

A. Aversion complex:

1. Does the patient look at the examiner?
2. Does he hang down his head in the flexor attitude?
3. Does he avert his eyes and face if looked at?

B. Hand shake:

1. Does the patient lift his hand at all?
2. Does he touch the hand and then withdraw?
3. Does he give a feeble clasp and hold the hand stationary?
4. Does he give the examiner's hand a clasp and shake it?
5. Does he smile and greet the vis-a-vis?

C. Communication:

1. Is there spontaneous greeting and talk?
2. Does the patient respond to "What is your name?" tersely, fully and adequately, or partially and inadequately?
3. Does he respond to the question, "How do you feel?" (Other questions may be built up in a definite order of merit. This gives us some idea of the communicativeness of the patient.)

D. Smile:

1. Is there any smiling beyond stereotypy during the examination, that is, is there friendly or relevant smiling? The reaction of the patient to a humorous question may be tested. The stock one to give is, "When did you last kiss your best girl?", which evokes a smile very readily.

E. Handkerchief or ball test for game cooperation:

1. Does the patient catch the ball?
2. What does he do with the ball? If he returns it immediately and smiles, he rates a good score. If he does

not return it but has to be urged to do so, he rates a lesser score. The ball throwing may be recorded as (1) catching; (2) spontaneity of returning; (3) vigor and pleasure manifested in game.

II. *Passivity:*

A. Postural passivity: the type of posture that will be maintained should be recorded, as the extreme form of turning the head to one side and standing on one leg with one arm raised; if maintained, this is the +++ of postural passivity. A short elevation and then a quick return to normal posture is +.

B. Imitation: if the patient does not spontaneously imitate, the score is zero; if he spontaneously imitates the examiner, no matter what the examiner does, this is the complete degree of passive imitation.

C. Commands: the response to commands can be listed in similar fashion. A few tests for this, such as stretching arms, bending body, are usually carried out fairly well. The schizophrenic in the main resists running and jumping commands, certainly at first. He feels either grotesque or clumsy, or dislikes these activities. The abolition of spontaneous jumping and running is a striking feature of the chronic schizophrenic.

III. *Resistance:*

This can be recorded by the same tests as outlined for passivity.

IV. *Endogenous automatic excitement:*

A. Automatic speech activity: loudness of utterance, rapidity of utterance, extent of variety of phrases used, distractability of utterances by questions, throwing the ball, etc.

B. Motor movements: vigor, frequency, number of body parts involved, distractability by questions and by throwing the ball to the patient.

TABLE SHOWING RECORD OF THREE DIVERSE TYPES OF SCHIZOPHRENICS.

Patient designated as I shows low social contact; on the other hand is extremely passive, shows no resistance, and little or no endogenous excitement.

Patient designated as II shows moderate social contact, is moderately passive, imitative and obeys commands only moderately well, but shows a high degree of endogenous automatic excitement, both of speech and motion.

Patient designated as III is described as negativistic, shows a low or absent social contact by all the tests, has no passivity, but is completely resistive and shows some endogenous speech excitement, some but less endogenous motor excitement.

SOCIAL CONTACT.

Score.	Aversion complex.	Greeting complex and communication.	Handshake.	Smile.	Ball or handkerchief test.
4	I, III	....	....	....	....
3	....	....	II	II	....
2	II	II	....	....	....
1	....	I	I	I	I
0	....	III	III	III	III, II

	PASSIVITY.			RESISTANCE.		
Score.	to posture.	to imitation.	to command.	to posture.	to imitation.	to command.
4	I	....	I	III	III	III
3	....	I	....	....	....	....
2	II	II	II	II	II	II
1	....	....	....	....	I	I
0	III	III	III	I	....	....

ENDOGENOUS AUTOMATIC EXCITEMENT.

Score.	Speech.	Motor.
4	....	II
3	II	....
2	....	I
1	I, III	....
0	....	III

BIBLIOGRAPHY.

Myerson, A.: Theory and principles of the "total push" method in the treatment of chronic schizophrenia. Am. J. Psychiat., 95: 1197-1204, 1939.

Tillotson, K. J.: The practice of the total push method in the treatment of chronic schizophrenia. Am. J. Psychiat., 95: 1205-1213, 1939.

THE JUDGMENT OF INSIGHT.

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The evaluation of insight and judgment by average psychiatric criteria is most ambiguous and confused. Insight and judgment are so very intangible that they cannot be precisely defined nor logically explained. The measure of each depends largely on subjective interpretation and can never be otherwise.

Judgment has been defined as (a) "the ascription of meaning to the given," (b) "the reference of a particular to a general concept," or (c) "the arousal of associated ideas." The verity or falsity of judgment is determined by the adequacy of the ideas aroused by a given situation or stimulus. Belief is a by-product of judgment. It is the result of only one group of associations aroused by the given situation. If more than one group of associations is aroused incompatibility enters. Doubt or indecision may thus arise and depends then upon the habits, temperament and character, or volitional attributes of the experiencing person. These latter faculties are therefore secondary determinants of judgment. The proofs and reasons for judgment are only mere rationalizations, or at best pure psychological camouflage hiding innate motives, beliefs, feelings or sentiments.

We speak of poor judgment if accepted mores and ethics are insulted and of good judgment if our own preconceived concepts and ideas are paralleled. The integrity of judgment is estimated by consideration of the subject's ability to make reasonable plans and to give due value to exigency. Defective judgment is present if inappropriate associations arise in response to a situation or belief. Such associations may nevertheless harmonize with the subject's affect and consequently meet with no opposing associations. Such false judgment occurs frequently in normal life; many so-called beliefs differ only in degree from the delusions of the insane therefore, and exist only because they are accepted by the majority of people as doctrine or dogma. Such beliefs usually imply complex affect and are often challenged by the minority, but with belated effect.

Insight and judgment involve similar mental functions, and either alone is an exact index of the other. In Gestalt terms insight is "an act or an ability to effect a direct configuration, a connected whole which is evident in the meaningful reaction to a stimulus-structure." Spearman states that insight is "the quality in consciousness that attaches to belief based on adequate (self-experienced) events." The popular concept of insight is the quality of penetration of the essence of, or the meaning of, things and happenings. The psychiatric concept of insight is none of the above but has been stated as (a) "the amount of realization a patient has of his own condition"—a quantitative estimate made by the psychiatrist—something a neurotic or a recovered psychotic is assumed to have and a not recovered psychotic is tacitly assumed not to have; and (b) "appreciation by a patient of the motives and genesis of his symptoms."

Etymologically the original meaning of insight referred to "internal insight, *i. e.*, insight is the act of the mind having inner vision and discernment." This is grossly akin to the earlier meaning of consciousness as the mind's mediate cognition of its own experiences. A later definition of insight is, "the power of penetrating with the eyes of understanding into the character of . . . . things." Still later perversion of the original meaning, is the following definition: "A more or less sudden realization of the meaning, significance, or use of a situation which is not based on previous experience in a like situation," and again: "Apprehension of truth ordinarily hid from man, in a direct unmediated fashion without reasoning or sense." This is more of a mystical experience; thus those with an imperative idea may or may not fully recognize its rationality or irrationality. Psychiatrically, insight implies "understanding of one's true condition when mentally ill," or in the words of Aubrey Lewis, "a correct attitude to a morbid change in oneself."

Expanding this definition: To have a "correct attitude" involves integrity of the perceptive mechanisms and an accessibility of perceptions to consciousness. Every mental act or activity is attended by association of the apprehended quality and this quality is apart from the actual object of apperception. Mentation as a process is a dynamic act whereby an idea or a concept is accepted into consciousness either by bringing to light (a) what was implied

in the idea or (b) adding to it. The process is therefore continuous. Ideas and associations are cumulative; each engram is subject to secondary isolation or compartmentalization as a temporally limited experiential phenomena. One not only perceives an object or idea but is aware of the act of perception normally. In mental disorder, for example in the phenomena of derealization, the patient may be aware of the unfamiliarity of the perceived environment, and also of the change in quality of the function of subjective awareness.

For the apprehension of judgment the immediate experience is subjective. Only with an attempt to describe is there any objectivation. This is not to say that experience need to be actively objectivated to be cognized. Experiential judgment may be quantitative or qualitative, gradual or abrupt, superficial or profound, and awareness or cognizance of it varies proportionately. The apprehension of change is the important point, and insight depends directly on the degree of exact judgment of continuous changes.

Insight, then, is concerned primarily with the awareness—or capacity for awareness—of change. Secondarily, with the judging of this change as to whether the alteration in the sphere of apperception is recognized as morbid; or in a sick mind the change may be judged to be the result of demoniacal possession, religious influence or some other preternatural intervention. Additional evidence of morbid change may be available to a person in the form of an appreciation of lessened intellectual capacity. This would be indicated by mistakes, or rebukes, or restrictions or other forms of social discipline. Communication of this knowledge of change to the psychiatrist is essential if insight is to be assumed. Such judgment usually finds expression in a declarative sentence in negative or positive terms. Every such sentence is necessarily the verbal expression of the change, the changing process, or result of change.

Normal insight is either a statistical or an ideal conception. The statistical norm psychologically, is idealistic in itself, because of the insurmountable difficulties encountered in the measurement of mental attributes. The ideal norm philosophically, sets up a canon of harmony dependent upon fluctuating, non-experiential values, both ethical and æsthetic, serving as a standard for comparison only, and not as an empirical criterion.

If purely biological and social averages are used, it is possible to establish more nearly a practical norm. Those qualities of reaction which best serve the ends of acceptable adaptation may then be termed normal. What is normal thus must be considered as both locally and temporally variable.

Another view is that that which follows natural laws is normal. But the abnormal follows its own laws; and therefore one cannot say that a thing is abnormal simply because it fails to conform to laws established for the majority of phenomena, or the laws found to have the widest validity. In ordinary usage all three views, statistical, teleological and biological, enter into the meaning of normal and abnormal, and all three roughly interlock.

It is important to stress that abnormality is not a disease. A genius is abnormal but not ill. A dwarf may be quite healthy but is nanoid, and is either normal or abnormal therefore, according to viewpoint. A pygmy is not abnormal in the confines of his habitat in relation to his fellows.

What then is morbid? As a physical condition, morbidity means pathological cellular or humoral alteration accompanied or followed by discomfort, disfunction, disuse and decease. As a mental condition, morbidity immediately involves polemical theorizing leading readily into abstraction.

For some psychiatrists the existence of morbidity is a matter of quantity; a mental anomaly becomes morbid only when it transcends a certain degree (Wilmanns). For others it is a matter of value: to be mentally ill is to be potentially harmful, undesirable or inferior (Jaspers). Kraepelin's view combines these quantity and value concepts. Bleuler emphasizes the social criteria. At best psychiatrists are catholically vague in their conception of morbidity.

The discrepancies and confusions in psychiatric thinking arise either from practical every day needs, or what are naively set up to be scientific criteria. The working method is a biological, pathological and metaphysical mumbo-jumbo, derived from textbooks, clinics, individual theories and biased statistics. The layman's viewpoint is more logical: He feels ill only if his life is disrupted, either by discomfort or forced inactivity.

The mentally sick person and layman generally, consider mental anomalies as obscure visitations, often with implicit moral or social obloquy. A psychotic's fellows may shun, stigmatize, ridicule or

sympathize with him, depending upon the individual intelligence. The ill person and his fellows rarely think that the situation constitutes an illness in any physical sense. The manifestations are referred to empirical, social and moral standards and compared to individual credos or motivations. If a psychopath's behavior or speech are not obtrusive or obnoxious to the crowd, he is not stigmatized as insane. A mystic is vulgarly called mad only because his concepts are either not conventional or not comprehensible. The alcoholic or drug addict is not regarded as ill if his conduct is confined to what is socially acceptable. A criminal is only "temporarily insane" if his conduct is manifestly anti-social. Even very apparent insanity as a concept may be routed by personal or ethical considerations. Insight must thus be considered to have a general connotation as well as an individual meaning.

General and individual concepts are further confused by the distinction between what constitutes mental morbidity and mere nervousness. "Nervousness" is assumed to have no basis in structural changes in the nervous system. The nervous person is considered one of the odd varieties of personality. The nervous person's symptoms or state may be similar to those consequent upon organic changes and may be completely judged to be organic but without any proven organic foundation. Or the nervous person may be considered to harbor a mild or incipient mental disorder, but one which is easily comprehensible in form and nature, and usually attributed to the effect of the environment. And this may be true.

There is no theory of psychopathology which can displace the preponderant rôle of one's bodily feelings in giving substance to consciousness and relevancy to reality. The patient's deference to the opinions of others, especially doctors, causes him concern as to the effects of an avowal of his subjective feelings which he recognizes reflect an attitude out of harmony with prevailing or professional opinion. At once the patient may thus give a false impression of insight. Also inadequacy or economy of language may mislead the judgment of the doctor as to the patient's insight. In mental illness, it is not from any external noxa as in physical disorder, but rather from the unfathomable depths of one's own personality that symptoms ultimately arise. The subjective attitude is said to be informed by the unconscious of genesis of symptoms. Lewis believes this is untenable; rather, insofar as immediate vague

awareness of the mental change occurs, it refers to a contrasted change in qualitative experience; secondary data such as akinesias, amnesias and perverse acts and imperceptions, are also in some measure available to the patient consciously.

In any mental disorder whether severe or mild, brief or persistent, obscure or comprehensible, it is with the whole disordered personality that the patient contemplates his state or his special symptoms. Insight is therefore bound to be distorted in one contributing function or another. A mere statement such as, "I must be mad," or "I ought to be in an asylum," is not necessarily evidence of insight. It may be a characteristic disturbance of affect, such as severe anxiety, self-reproach or "hysteria." It is not far from being similar to delusions of sin or possession. Sometimes convoluted statements of insight, partial interpretations, or even correct and painful ones, struggle within the illness and undergo unpredictable fluctuations from time to time.

The question of insight is of great clinical importance as far as prognosis is concerned, and the purpose of this paper has been to suggest some of the conditions and difficulties of its evaluation. In summary: insight and judgment both are purely relative concepts and dependent upon analysis and opinion which are variable locally temporally and individually. The presence or quality of insight or judgment should never be stated categorically but only with appropriate explanatory terms.

DETOXICATION OF SODIUM BENZOATE IN NEUROPSYCHIATRIC DISORDERS.

The Excretion of Hippuric Acid After the Ingestion of Sodium Benzoate.*

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The relation of disease of the liver to disorders of the central nervous system has been, for a long time, a problem in neuropsychiatry. In 1912, Wilson [1] described the simultaneous occurrence of hepatic cirrhosis and lenticular degeneration as a familial disease. This renewed interest in the relation of liver disease to disease of the central nervous system, particularly of the basal ganglia.

The importance of the detoxicating function of the liver is suggested by this relationship since Weil and Crandall [2] have reported toxins in the blood of dogs about the fourth day after liver damage was produced by ligating the common or pancreatic ducts. These toxins caused destructive changes in rat spinal cord in test-tube experiments.

Man eliminates benzoic acid almost completely as hippuric acid and in man the liver is the organ concerned in detoxicating benzoic acid which is conjugated with glycine to form hippuric acid. The body does not have a store of preformed glycine. The rate of formation of hippuric acid depends on the rate of synthesis of glycine by the body. The greater the amount of benzoic acid administered, the greater the amount of glycine is produced until the maximum capacity for this synthesis is reached.[3] Quick [4] introduced a liver function test based on the excretion of hippuric acid in the urine after the ingestion of sodium benzoate. The

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hippuric acid test estimates a well defined function of the liver—its ability to conjugate benzoic acid with glycine to form hippuric acid. The hippuric acid test for hepatic function has been found reliable among others, by Snell and Plunkett.[5] Quick's method consisted in giving a patient 5 grams of benzoic acid (as the sodium salt), and measuring the amount of hippuric acid excreted within four hours of the time of administration. Expressed in terms of benzoic acid, typical values for the excretion in normal individuals reported by Quick were 2.99g., 3.55g., 3.20g. Vaccaro[6] reported the following normal values in 10 cases free from hepatic and renal disease: 3.16, 3.34, 3.06, 3.46, 3.54, 3.28, 2.98, 3.03, 3.19, 3.13.

Recently, Quastel and Wales[7] using the hippuric acid test in schizophrenia reported that all of 18 patients with catatonic dementia præcox who were tested gave values of hippuric acid excretion, as expressed in equivalent benzoic acid, lower than normal. They inferred that a metabolic disturbance in the liver affecting benzoic acid detoxication may be a characteristic feature of catatonic patients. We attempted to verify Quastel and Wales' results in catatonia and also applied the test to other neuropsychiatric disorders.

We followed the technic used by Quastel and Wales in which the correction value of hippuric acid still remaining dissolved in the urine is given as .45 gram per 100 cc. This is more accurate than Quick's factor of .33 gram per 100 cc. which is the saturation value for water. Urine is a better solvent for hippuric acid than water. Six grams of sodium benzoate dissolved in water was given in the morning and the amount of hippuric acid was determined in the urine, hourly, for 4 hours. We shall not repeat the method here as it is given in detail in Quastel and Wales' paper.

We performed the test on 88 subjects in whom renal disease was ruled out by clinical and blood and urine examinations and in whom there was no clinical evidence of hepatic disease. In several patients the test was performed twice or three times. Among the subjects tested were 17 patients with catatonia, 9 with hebephrenic dementia præcox, 20 with chronic encephalitis, 26 with epilepsy of whom 15 were extramural and 11 were institutionalized, 8 with multiple sclerosis, 3 with paraplegic state, 1 with progressive muscular dystrophy and 4 normal subjects.

RESULTS.

Of 17 patients with catatonia, 8 showed hippuric acid excretion, expressed as benzoic acid, less than 2.9 grams. The average for the entire group was slightly less than 2.9 grams (2.87). The mean deviation was ±0.5. The excretion of hippuric acid, expressed as benzoic acid as low or less than 2.9 grams indicates a deficiency in detoxication of the 6 grams of sodium benzoate ingested. The patients with hebephrenic dementia præcox had a normal average excretion of hippuric acid which was 3.71, only 1 having an excretion below normal. The average excretion of benzoic acid of the patients with residuals of encephalitis lethargica was normal (3.16) although 7 of the 20 patients showed an excretion below normal. The mean deviation in this group was ±0.8, and there was a great variability in the group as shown by the coefficient of variability involving the mean deviation which was 25.6. The tests showed very interesting results in the epilepsy group. The extramural patients with epilepsy had a normal average value of hippuric acid excretion, which was 3.97, only 1 of 15 showing a value slightly below normal. In this group the mean deviation was ±0.4 and there was the least variability of all groups studied, the coefficient of variability being 10.0. The intramural patients with epilepsy, however, had an average value less than normal (2.67) and 6 or 54.5 per cent of the patients showed defective benzoic acid excretion. The mean deviation of the institutionalized patients with epilepsy was ±0.9 and this group showed the greatest variability of all the groups studied, the coefficient of variability being 34.8. The patients with multiple sclerosis showed a low normal average value for the excretion of benzoic acid (2.99) 2 of the patients having values below normal. Of 3 patients with paraplegic state due to compression of the cord, 1 had a value below normal. One patient with progressive muscular dsytrophy showed a normal excretion of benzoic acid. The 4 normal subjects showed normal values. The numerical values are given in Table 1.

COMMENT.

Although the average excretion of benzoic acid in the catatonic group was less than normal (2.87), 9 of the patients had values above normal, 8 or 47 per cent having below normal values. These results are different from those reported by Quastel and Wales

who found all of their eighteen catatonic patients to have values below normal. Ström-Olsen, Greville, and Lennon [8] have also recently reported on hippuric acid synthesis in schizophrenia and have failed to confirm Quastel and Wales' results. Of 28 catatonic patients investigated by Ström-Olsen *et al.*, only 5 showed hippuric acid excretion, expressed as benzoic acid, as low as or less than

TABLE 1.

AVERAGE HIPPURIC AND EQUIVALENT BENZOIC ACID EXCRETION AFTER INGESTION OF SODIUM BENZOATE.

Diagnosis.	No. of patients.	Hippuric acid excreted. Average in gms.	Hippuric acid excreted. Range.	Equivalent benzoic acid excreted. Average in gms.	Equivalent benzoic acid excreted. Range.	Number and per cent of patients with values below normal (2.9 gms. or less).
Chronic encephalitis..	20	4.66	2.63-6.82	3.16	1.79-4.64	7—35.0%
Epilepsy (extramural)	15	5.85	4.15-6.87	3.97	2.82-4.49	1— 6.6%
Epilepsy (intramural).	11	3.88	1.22-5.52	2.67	0.83-3.72	6—54.5%
Catatonia	17	4.24	2.09-5.87	2.87	1.42-3.99	8—47.0%
Hebephrenic dementia præcox	9	5.46	3.98-6.37	3.71	2.71-4.33	1—11.1%
Multiple sclerosis	8	4.40	2.47-6.88	2.99	1.68-4.68	2—25.0%
Paraplegic state	3	4.16	2.39-5.14	2.83	1.62-3.49	1—33.3%
Progressive muscular dystrophy	1	6.20	.....	4.22	.....	0—0
Normal subjects	4	6.03	5.78-6.28	4.08	3.86-4.27	0—0

2.9 grams. Moreover, we cannot infer from the results of hippuric acid synthesis that there is a metabolic disturbance in the liver of patients suffering from catatonic dementia præcox, because the low hippuric acid excretion in some of our catatonic patients and in all of those reported by Quastel and Wales may have been due to the rigidity and immobility of the muscles characteristic of the disease. That this may be a possible explanation for failure in the synthesis of glycine was shown in one of our hebephrenic patients who was immobilized in a body cast. While he was in the cast his equivalent benzoic acid excretion was 1.88 grams. When the cast was removed and he resumed normal activity, several determinations showed an equivalent benzoic acid excretion above 4 grams.

The results in chronic encephalitis are of interest since disease of the basal ganglia is associated with cirrhosis of the liver in progressive lenticular degeneration and in pseudosclerosis of Westphal. The average hippuric acid excretion in 20 patients with chronic encephalitis was normal. There is no defect in the function of the liver in this disease, as based on the average of the group but we are unable to explain the values below normal in several of the patients and the great variability from the mean.

The tests for hippuric acid synthesis in epilepsy show very interesting results. In contrast to the extramural patients with epilepsy, the institutionalized, deteriorated patients had an average, equivalent benzoic acid excretion below normal. Paskind and Brown [9] have reported constitutional differences between deteriorated and non-deteriorated patients with epilepsy. Whether deteriorated patients with epilepsy have a defect in the synthesis of glycine and whether the defect is a constitutional error in metabolism cannot be inferred from the hippuric acid tests.

Multiple sclerosis, as a group, did not show any evidence of liver dysfunction although 2 of the 8 patients showed benzoic acid excretion below normal. Other factors in addition to hepatic function should be studied in such a disabling disease as multiple sclerosis as for example diminished muscle metabolism to account for the low values of hippuric acid excretion in some of the patients.

Since glycine is used in the therapy of progressive muscular dystrophy it was of interest to determine the ability of a patient with this disease to synthesize glycine used in conjugation with the ingested benzoic acid to form hippuric acid. The patient we tested showed a normal function of glycine synthesis and excreted a normal amount of equivalent benzoic acid (4.22). The ability of patients with muscular dystrophy to synthesize glycine at the same rate as normal controls, after feeding of sodium benzoate, has been reported by Friedberg and West, Linneweh and Linneweh and Thomsen.[10] Whatever may be the therapeutic value of glycine in the dystrophies, it is not due to an inability of the organism to synthesize it.

After our paper was completed an article appeared [11] in which it was pointed out that the solubility of hippuric acid in urine is considerably greater than 0.33 per cent and moreover that it shows

substantial variation with different urine specimens. In such cases as for example in catatonia the apparent low hippuric acid output may be due to a correspondingly greater solubility of the urine for this substance. The solubility for hippuric acid of urine from various diseases, will be determined and reported in a subsequent note.

SUMMARY AND CONCLUSIONS.

The rate of detoxication of benzoic acid was determined in patients with various neuropsychiatric diseases. About one half of the patients with catatonia showed a defect in hippuric acid synthesis. This may be due to the state of muscular rigidity and immobility of these patients. Deteriorated patients with epilepsy showed a defect in the synthesis of hippuric acid, whereas extramural patients with epilepsy showed a normal excretion of the equivalent benzoic acid. This may indicate a defect in glycine synthesis in deteriorated patients with epilepsy. Normal average values were found in post-encephalitic Parkinsonism and multiple sclerosis. A patient with progressive muscular dystrophy was able to synthesize glycine at a normal rate.

BIBLIOGRAPHY.

1. Wilson, S. A. K.: Progressive lenticular degeneration, a familial nervous disease associated with cirrhosis of the liver. Brain, 34: 295, 1912.
2. Weil, A., and Crandall, L. A.: Die Beziehungen zwischen dem Lipasegehalt und der neurotoxischen Wirkung des Serums nach experimenteller Leberschädigung. Zeitschrift für die gesamte Neurologie und Psychiatrie, 140: 577, 1932; Crandall, L. A., and Weil, A.: Pathology of Central Nervous System in Diseases of the Liver. Arch. Neurol. and Psychiat., 29: 1066 (May), 1933.
3. Quick, A. J.: The conjugation of benzoic acid in man. J. Biol. Chem., 92: 65, 1931.
4. Quick, A. J.: The synthesis of hippuric acid. A new test of liver function. Am. J. of Med. Sciences, 185: 630, 1933; Clinical value of test for hippuric acid in cases of diseases of liver. Arch. Int. Med., 57: 544, 1936.
5. Snell, A. M., and Plunkett, J. E.: Hippuric acid test for hepatic function; its relation to other tests in general use. Am. J. Digest. Dis. and Nutrit., 2: 716: 721 (Feb.), 1936.
6. Vaccaro, P. F.: The synthesis of hippuric acid. Its value in detecting hepatic damage secondary to diseases of the extrahepatic biliary system. Surgery, Gynecology and Obstetrics, 61: 36, 1935.

7. Quastel, J. H., and Wales, W. T.: Faulty detoxication in schizophrenia: Abnormal excretion of hippuric acid after administration of benzoate. Lancet, 2: 301-305 (Aug.), 1938.
8. Ström-Olsen, R., Greville, G. D., and Lennon, R. W.: Hippuric acid synthesis in schizophrenia. Lancet, 2: 995 (Oct.), 1938.
9. Paskind, H. A., and Brown, M.: Constitutional differences between deteriorated and non-deteriorated patients with epilepsy. Am. J. Psychiat., 95: 901 (Jan.), 1939.
10. Friedberg, I. H., and West, E. S.: Glycine synthesis in pseudohypertrophic muscular dystrophy. J. Biol. Chem., 101: 499, 1933; Linneweh, W., and Linneweh, F., Zur Frage des Glykokollmangels und der Glykokolltherapie bei progressiven Muskeldystrophie. Deutsches Arch. f. klin. med., 176: 526, 1934; Thomsen, A.: The glycine synthesis in patients with progressive muscular dystrophy. J. Clin. inv., 16: 231, 1937.
11. Weichselbaum, T. E., and Probstein, J. G.: Determination of hippuric acid in urine. J. Lab. and Clin. Med., 24: 636 (Mar.), 1939.

DISCUSSION.

Dr. Edwin F. Gildea (New Haven, Conn.).—This paper of Dr. Finkelman opens up many different problems, as he has already indicated. When we heard of Quastel's work, it occurred to us that one of the reasons he had these low excretions was that there was a failure of absorption in some of these very sick patients, especially catatonics. As we had some experience with the differences between sugar curves by mouth and sugar curves given intravenously, we went at the problem of using the intravenous test instead of the one by mouth. We have not done very many—about eight—and we have only had two patients who had extreme degrees of catatonia, and to date our recoveries have been well over 100 per cent of hippuric acid. We did one of the patients by mouth and had a borderline recovery, so we have a suspicion that the gut may be responsible for some of these low curves.

I would also like to suggest that the work of Dr. Rose be considered. He has done a great many hippuric acid tests for liver function, and he has found a very low hippuric acid output, as Quick did, in a great many kinds of liver disease, and also in patients who were suffering from the "B" complex deficiency, and their hippuric acid output recovered on adequate vitamin therapy.

Consequently, in dealing with these very sick patients, one has to consider the gut, the kind of food they are getting, whether it is deficient in vitamins, as well as the problem of muscular activity. We happen to have two patients who have been in casts for three months and pretty completely immobilized, and by the intravenous route these patients were able to transform benzoic acid into hippuric acid in the normal fashion. So it may be that hippuric acid involves the gut as well as the muscles.

DR. ARTHUR WEIL (Chicago, Ill.).—Dr. Finkelman and his associates should be commended for their attempt to replace the vague conception of "endogenous intoxication following liver disease" by more definite statements about the disturbance of partial functions of the liver.

While the synthesis of hippuric acid is an important indicator of a normal liver metabolism, its disturbance indicates an already advanced impairment of the liver cells. This test should be followed by more sensitive ones like the elimination of bilirubin or by other experiments testing the function of the reticulo-endothelial system.

It now seems to be generally accepted that liver disease frequently leads to disease of the brain with a predilection of the neo-striatum. The question arises why the neo-striatum should be a place of predilection in this relationship. One of the answers possible may be found in the "Kernicterus" of newborn children. Here the bile-pigments diffuse through the wall of the blood-vessels in the region of the basal ganglia, while later in life these vessels become impermeable for the same pigments. One then might assume that in certain individuals this change in permeability does not occur at all or only incompletely or that the permeability is restored following damage of the endothelium by certain toxic products circulating in the blood. In this case toxic metabolic waste-products circulating in the blood might easily diffuse through the vessel wall into the brain itself.

DR. ROY G. HOSKINS (Boston, Mass.).—Buscaino a few years ago succeeded in convincing himself that liver disfunction played an important rôle in schizophrenia, but that the patient after a time became adjusted to the disorder and that the liver function tests in the chronic cases were essentially normal, but he reported that he got marked evidence of the deficiency in the early phases of the disorder.

I might say that we were quite interested in this same problem in Worcester. A few years ago Dr. Sleeper, especially, used various liver function tests, tests for the bile, pigments, and so on. We were not able to convince ourselves in the group that we worked with—chronic patients—that there was evidence of liver disfunction, but we always reserved judgment because of Buscaino's claim that it is the early cases that show.

DR. M. K. HORWITT (Elgin, Ill.)—I should like to express my appreciation to Dr. Gildea for giving us a current explanation as to why we are getting differences in patients who are active and those who are inactive. I should like to ask him if he can tell us how much sodium benzoate he injected and how much he felt was safe, so that we could check back on the absorption of the material.

DR. EDWIN F. GILDEA.—Dr. Quick has developed a technique of giving sodium benzoate intravenously and it has so far, with us, produced no untoward effects in the patients. Dr. Quick wrote to us that he had less trouble with gastro-intestinal disturbances, and that the toxic results and complexes were also less frequent when they gave it intravenously. You only give

1.7 grams, which is a good deal less than you give by mouth, but you can recover a gram on the controls without any difficulty. As a matter of fact, the difficulty we have had is that the factor of correction often gives us recoveries of 115 and 120 per cent, and that is puzzling us more than getting incomplete recoveries of the hippuric acid. That is another problem that we haven't made much progress on. But I think that doing both the tests, by mouth and intravenously, may help to clear up the differences in the results.

CHAIRMAN NEYMANN.—I would like to ask Dr. Finkelman a question.

You discussed, I believe, the difference in values found between institutionalized epileptics and non-institutionalized. Was the factor of their diet, which Dr. Gildea has already suggested, taken cognizance of, and was the fact that in institutions people notably have less exercise than when they are out at large, considered in your conclusions?

DR. ISIDORE FINKELMAN.—I am sincerely grateful to Dr. Gildea for bringing up the question of absorption. As Dr. Horwitt mentioned, we were discussing that the first time we started this work, and Quastel mentioned that before he reached his far-reaching conclusion that there is a metabolic disturbance in catatonia, he had a question-mark in the back of his mind as to whether it isn't a defect in absorbability; but nevertheless he reached that conclusion that there is a defect in liver function.

What we want to point out is that we have reached conclusions opposite to those of Quastel, that there is no liver damage in catatonia, and the chances are, as Dr. Gildea has pointed out, that it is due to a failure in absorption. We merely pointed out that it may be also related to immobility.

This failure of absorption in catatonic dementia præcox is to us of greater importance than eliciting any liver damage, because it fits in with all the recent work on disturbances in the autonomic nervous system. For instance, in Elgin we tried injecting pitressin in a few patients with dementia præcox, and we noted the startling result that, whereas, if we injected the pitressin to internes, who are normal subjects, they immediately had to obey nature's call and complained of marked abdominal cramps with peristalsis. The same dose, or even greater doses, produced no results in demential præcox cases. They did have a blanching of the face, and so on, but as far as gastro-intestinal activity is concerned, nothing happened no matter how long we watched them. So there is something in the movements of the gastro-intestinal tract which we believe is more of a factor in the hippuric acid excretion than any liver damage.

We are also very grateful to Dr. Weil, who has done very important neuropathologic work in the relation of liver disease to disease of the central nervous system, and his remarks are very important on the subject.

We are, of course, in entire agreement with Dr. Hoskins.

Now, in answer to Dr. Neymann's question as to the diet, we of course found patients with hebephrenic dementia præcox who had normal values though they had the same diet as those who were suffering from epilepsy, and the same activity. We did find normal values in institutional patients despite

the control of exercise and the diet. We merely called attention to the fact that in extramural epileptics there was a normal output of hippuric acid, whereas in the intramural epileptics there was a low value. But the hebephrenics who were also in the institution had a high value, so that the factor of exercise and diet does not enter. The factor of absorption even in the intramural epileptics may enter, and may not be due to a defect in glycine synthesis, and may again be due to a defect in absorbability from the gut.

AN OBSERVATION ON THE TREATMENT OF MENTAL CASES WITH SUB-SHOCK DOSES OF INSULIN.

By C. R. BENNETT, M. D., and T. K. MILLER, M. D.,
Camarillo State Hospital, Camarillo, Calif.

Since the introduction by Sakel of insulin as a therapeutic agent in the treatment of mental disease, its value has been quite definitely established. Practically all who have extensive experience with this form of therapy, recognize it as an advance over any single treatment method previously employed on a large scale. At the same time all judicious observers are aware of its limitations and are cognizant of the fact that much experimental work must still be done to make clear the rationale of the treatment, and hence its better understanding and more effective use.

Sakel in his original papers stated that patients of an irritable and generally disagreeable nature presented, after treatment with insulin, a much more congenial and cooperative attitude. This observation, made first on drug addicts, was repeated over and over again in his later experiments with psychotic patients.

This observation led us to wonder whether insulin in smaller doses than required for shock, might not influence favorably the various types of reactions among mental patients which create problems in their care and management and which have led in the past to the use of restraint and sedation which are often undesirable.

Such problems are presented by the manic patient. Large doses of sedatives and their continued use are often dangerous because of the depressing effect on respiration and circulation and particularly because of the risk of toxic manifestations in the form of excitement and hallucinations. Physical restraint is always undesirable, and for hydrotherapy, which requires a trained staff in constant attendance, adequate facilities are not always available.

With resistive catatonic patients the necessity of tube feeding possibly over a period of weeks or months, is not without dangers, and trauma of the mucous membranes of the nasal and throat passages is often the cause of ulcerative lesions. The operation is time consuming and objectionable in many ways.

In every hospital there are patients who are always in trouble, annoying other patients, consuming the doctor's time to no advantage, etc. If we can alter that disposition favorably, we feel highly compensated for our effort.

In short, we all appreciate the atmosphere of a quiet, well-behaved ward in contrast to the quarrelsome, loudtalking, fighting type of conduct on the "disturbed ward" so well known to every hospital man. In the following report we hope to demonstrate that this transformation can be accomplished simply and without danger.

Type of Cases Treated.

Any case which presents a behavior problem is a candidate for sub-shock insulin therapy. Our series to date would indicate only two contraindications; namely, old arteriosclerotic individuals where there is evidence of coronary sclerosis, and secondly, patients who are already in extremis or so badly reduced physically that the slightest disturbance might prove disastrous. Suspected or known cases of active pulmonary tuberculosis and all other acute infectious diseases would of course be contraindications during the period of activity.

Technique Employed.

It will be useful first to explain the physical plant in which this work has been carried on. Camarillo State Hospital is still in the process of construction. When completed it will house approximately 7000 patients. At present we care for 2400. The buildings now being used were built primarily for male patients only, anticipating a second unit for female patients. Approximately half the space now available has been adapted to care for female patients. As yet, we have no hospital or receiving unit, and pending the construction of such a unit, it becomes necessary to utilize custodial wards for hospital and receiving services. Under present conditions we are using one 60-bed ward for the combined purpose of caring for newly admitted patients, acutely ill and acutely disturbed cases. A reasonable degree of isolation of the three groups is possible, but there are obvious handicaps. This circumstance is mentioned only to emphasize the results obtained even under very unfavorable conditions. This three-in-one com-

bination serves a male population of approximately 1200 patients and a similar arrangement cares for the same number of female patients.

Because of lack of space every effort is made to adjust a patient on the custodial ward. Only when it appears impossible to care for the patient properly on that ward, when he is either combative, continuously manic to the point that the entire ward is apt to be kept in turmoil, or when the patient refuses to go to the main dining room and requires individual feeding available only on the hospital ward, is he admitted to that ward. Therefore the group which constitutes the material with which we have worked, is composed of the extreme cases of the entire hospital population.

The prescription is written for insulin to be given from one to two hours before breakfast and lunch. The doses range from 15 to 50 units, with an average of about 25. An evening dose is not given because of the danger that unfavorable reactions might occur during the night when the patient was presumably asleep and therefore go unnoticed. The dose is arrived at arbitrarily, taking into consideration the size of the patient, his physical condition and the desired effect. The intent is to administer sufficient to produce the prodromal symptoms of shock without actually producing coma. If the patient, after insulin has been given, refuses to eat, he is observed for untoward reactions and tube fed if they occur. If no unusual symptoms are manifest, the patient is allowed to go without food until after his evening meal has been served. If, at that time he still has not eaten, a gavage feeding of 1000 cc. egg-nog, with 50 gms. of dextrose added, is administered. No instance of nocturnal hypoglycemia has occurred where this routine has been followed. If gavage is not retained, 50 gms. of 30 per cent glucose must be given intravenously. The insulin is always given by a nurse thoroughly familiar with the technique.

If the desired therapeutic effect is not obtained within 48 hours and the dose administered has produced no systemic effects, the dose is increased five units per day, per dose, until the patient either shows clinical improvement, or unfavorable reactions become manifest. The maximum dose in any case in this series was 50 units twice a day. Small doses of five or ten units seldom give evidence of producing the desired effect. In certain instances in which it is inconvenient or undesirable to give the morning dose,

one dose is given before the noon meal. This dose is not increased in lieu of the omitted morning dose.

Results Obtained.

The study here reported covers a 15-month period commencing May, 1938, and including August, 1939. Approximately 125 cases have been treated by this method, including 85 male and 40 female patients.

It is difficult to tabulate results statistically as the therapy was instituted purely on an empirical basis and detailed records were not kept. Conclusions, therefore, were drawn from objective analysis of our experience. The general effect of the treatment is, of course, most apparent to the ward personnel. In this hospital the doctors' offices are located on the ward and from eight to ten hours of the physicians' time is spent on the ward in immediate contact with the patients. Our observations were, therefore, not the result of routine ward rounds, but of close personal contact during most of the patients' waking hours.

Our observations were as follows:

1. Nearly every case of acute manic excitement has been controlled without the use of sedatives or hydrotherapy within an average period of 36 hours after adequate insulin dosage. This has been accomplished without any serious side effects from the insulin and without any of the dangers of heavy sedation usually necessary for such cases.

2. The use of sedatives has been reduced approximately 60 per cent in all cases, and almost 100 per cent in cases of extreme psychomotor activity. The use of sedatives is limited now almost entirely to epileptics and to standing orders for short periods of nocturnal disturbances where it is impractical to give insulin.

3. Tube feedings have been eliminated 90 per cent. Within the past ten months it has been necessary to tube feed only six patients, with an average of three feedings for each patient before the insulin has become effective and eating voluntary. Only one case, a catatonic schizophrenia 17 years of age, has required feedings over a period of three weeks because in this case we felt there was sufficient evidence of status lymphaticus to contraindicate doses of insulin sufficient to give the desired response. Before

institution of this therapy it was not uncommon to have three or four patients being tube fed daily over a period of several weeks.

4. Clinical improvement in newly admitted cases of early dementia præcox and manic-depressive psychosis, as well as other depression states such as involution melancholia, is in many instances almost miraculous because of the rapidity with which the changes occur. It has been our experience, however, that this improvement does not continue if the insulin is discontinued as soon as improvement is noticeable. In most instances from three to eight weeks of continued therapy is required to produce satisfactory periods of remission.

5. All patients improve markedly in their physical status, gaining weight on an average of 15 pounds within six weeks.

6. Patients suffering from caruncles, carbuncles and acne simplex show rapid disappearance of these conditions. It is largely routine now, to give insulin to any patient threatened with "boils" as an abortive measure. Acne does not always respond so favorably.

7. The necessity of a so-called "disturbed ward" is practically eliminated.

Comment.

It has been our observation that this therapy is not as effective with female patients as with male patients although we are not prepared at this time to say that such is the case without reservations. The therapy was not instituted on the female wards until about six months after it had been used on the male patients, and was started largely as the result of the obvious benefit resulting from its use on the males. Also, in starting this therapy on the females, smaller doses were given on the assumption that females do not require as large doses as males to produce the same effect. Disregarding this assumption and having made the dosage more nearly equal, the results with female patients have been more uniformly satisfactory.

These observations have been for us a most unusual experience, and are presented in this preliminary report with full realization that the conclusions will require further corroboration. Plans are being made to continue the study with proper controls and it is hoped that similar studies may be undertaken by others in order that the question of the value of the therapy herein outlined may be definitely answered.

Conclusions.

1. We have observed, in the past year and a half, among a mixed population of over 2400 patients, that a course of sub-shock doses of insulin is effective in controlling most of the problems of management of uncooperative mental patients.

2. The necessity of sedation by drugs can be limited to epileptics and emergency nocturnal disturbances.

3. Difficult feeding cases can be largely corrected by the use of sub-shock doses of insulin.

4. Sub-shock doses of insulin are effective in all types of cases where there are no physical contraindications.

5. No special equipment or personnel is necessary to carry out this form of therapy.

6. Sub-shock insulin therapy is effective in aborting caruncles, carbuncles and acne simplex, as well as in producing more rapid healing in the well-developed lesion.

7. Sub-shock insulin therapy is in no sense considered a curative therapy although many favorable remissions occur.

8. There is need for further controlled study of this type of therapy.

THERAPEUTIC QUARTAN MALARIA IN THE TREATMENT OF NEUROSYPHILIS AMONG NEGROES.

A Progress Report.*

By GEORGE C. BRANCHE, M. D.,
Veterans' Administration Facility, Tuskegee, Ala.

An initial report, based on the study of 36 Negro neuro-syphilitic patients treated with quartan malaria from November, 1932, to September, 1933, was made before this Association in June, 1934.[1] This paper deals with 346 patients, the total number treated from November, 1932, to November, 1938.

It may be well to state at the outset that the same satisfactory results as shown in the previous report still obtain. The original strain as secured from the United States Public Health Service Laboratory, Columbia, S. C., on November 24, 1932, is still in use and there is no appreciable evidence of decrease in virulence although 46 actual transfers have been made over the six-year period. Shipments of blood have been made to some of the Veterans' Administration facilities, the United States Public Health Laboratory at Columbia, S. C., State Hospitals, State Department of Public Health, private institutions and private physicians. Reports from many of these sources indicate that the same therapeutic benefits experienced by us are being observed by them.

The strain has been free of gametocytes since 1935. Although it is rather generally accepted that clinical manifestations are caused by the non-sexual parasites, yet it has been uniformly true that during this apparent asexual period the clinical course of the disease has been milder with an even lower incidence of the few complications previously experienced during our early observations. On the whole the patients are permitted to run a longer course without any signs of complications, especially our most

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dreaded one, nephritis. This has been a serious complicating factor in eight of the 346 patients treated, only three of these occurring since 1935 although more than half of the total number have been treated since that year. During the last two years, three of our patients experienced 30, 41 and 44 paroxysms respectively with an average rise in temperature to 104.2°F., 103.5°F. and 103°F. Termination was spontaneous in each of these cases and none exhibited signs of severe anemia, nephritis or other untoward effects. A marked improvement both physically and mentally has been shown in each case. None of the patients treated to date has experienced jaundice or convulsive seizures, two conditions rather frequently noted by other observers during treatment by inoculation malaria. Six patients developed herpes labialis which cleared up after the third or fourth paroxysm. One case of splenic enlargement necessitating therapeutic termination was reported in our first series of 36 cases. However, subsequently this condition has not occurred in a single case. Our most serious complication experienced in this present series of cases which responded to treatment was that of Case No. 16,968 which suddenly developed acute nephritis after the sixth paroxysm resulting in complete blindness consequent to albuminuric retinitis. Vision was finally restored following termination and symptomatic treatment. A pronounced improvement was shown in his mental and physical status.

Perhaps our most interesting observation since our previous report has been the use of tryparsamide therapy throughout the incubation period. In 23 cases tryparsamide was administered beginning shortly before inoculation and continued until the first paroxysm without appreciable effect upon the incubation period or percentage of "takes." The drug is especially indicated in the under-nourished but otherwise good risk for its tonic action and resistance building power plus the known therapeutic benefit in neurosyphilitic patients. The efficacy of its use in the types of cases mentioned is readily appreciated when it is considered that the incubation period of quartan malaria is from 30 to 45 days. The drug is rapidly excreted through the kidneys and the entire intestinal tract without any appreciable damage to the kidneys even when given over a prolonged period. Hence, the vulnerability of this organ is apparently not materially increased with the onset of malaria—the drug being discontinued as soon as the patient re-

sponds with chills and fever. The incubation period was essentially the same as in those cases in which tryparsamide was not used.

In 12 cases selected at random among 23 patients treated by this method since 1934, 11 responded to the first inoculation with an incubation period ranging from 29 to 41 days or an average of 35 days. The twelfth Case No. 11,713 (Table 1) responded

TABLE I.

TRYPARSAMIDE CONTINUED THROUGHOUT INCUBATION PERIOD—QUARTAN MALARIA.

Case no. and color of skin.	Date tryparsamide began.	Date of inoculation.		Date of first paroxysm.	Date tryparsamide suspended.	Av. temp. height.	No. of paroxysms.	Mode of termination.
No. 8526								
Dark brown..	8/6/36	8/27/36	.....	10/5/36	10/1/36	104° F.	10	Atabrine
No. 11,485								
Black	2/8/34	3/15/34	.....	4/26/34	4/26/34	103.5° F.	11	Quinine
No. 11,537								
Dark brown..	3/8/34	3/15/34	.....	4/20/34	4/26/34	104.4° F.	18	Quinine
No. 11,713								
Brown	3/29/34	4/2/34	5/21/34	5/23/34	5/17/34	104° F.	12	Quinine
No. 11,986								
Brown	6/7/34	8/20/34	.....	9/24/34	9/24/34	103.8° F.	14	Quinine
No. 12,167								
Mulatto	7/16/34	7/30/34	.....	8/31/34	8/23/34	105.4° F.	10	Quinine
No. 12,168								
Light brown..	7/12/34	7/30/34	.....	8/28/34	8/23/34	104° F.	12	Atabrine
No. 12,211								
Brown	7/19/34	7/30/34	.....	9/3/34	9/6/34	105° F.	14	Quinine
No. 15,538								
Dark brown..	8/27/36	8/27/36	.....	9/28/36	9/24/36	102° F.	10	Spontaneously
No. 15,674								
Brown	8/20/36	8/27/36	.....	10/6/36	10/1/36	104° F.	10	Atabrine
No. 15,712								
Brown	8/27/36	8/27/36	.....	10/2/36	10/1/36	104° F.	10	Atabrine
No. 15,892								
Brown	8/27/36	8/27/36	.....	10/6/36	10/1/36	104.5° F.	14	Atabrine

to the second inoculation in a total elapsed time of 51 days. There was no difference in the temperature elevation or type of curve for the strain. Two patients, one brownskin in color and the other a mulatto, maintained an average height of temperature per paroxysm of 105°F. and 105.4°F., respectively. The general average for the 12 cases was 104°F. The patients experienced from 10 to 18 paroxysms. Termination was spontaneous in one case after 10 paroxysms, quinine was employed in six and atabrine in five. Each patient received from 6 to 12 injections of tryparsamide before

the onset of first paroxysm. Thus it is clear that tryparsamide administered during the incubation period has no appreciable effect upon the course of the malaria nor does it tend to cause spontaneous termination.

It is probably not amiss to state at this point that each case showed a good improvement both clinically and serologically within a relatively short period following fever therapy. Despite this fact, post-malarial chemo-therapy in the form of bismuth, tryparsamide or both was given in each case. Eight of the 12 patients have been discharged. Two of the eight were recently readmitted for treatment for other than mental disorder. Clinical and serological checkup in these revealed no evidence of relapse. Three of the six remaining discharged patients have been out for more than three years and three from one to more than two years.

Solomon and Epstein [2] in making a study of results obtained in their clinic in the treatment of general paresis by various methods found that a group of patients who had received a fairly long course of tryparsamide without satisfactory result, ample time for improvement having elapsed in each case, responded well both clinically and serologically when given fever therapy. These observers noted that in almost each instance the cerebrospinal fluid became negative, and in the majority of cases the spinal fluid response following malaria was much quicker than is to be expected in cases treated from the outset by fever.

It would therefore appear, from our experience and these observations as recorded by Solomon and Epstein that the combination method, namely, tryparsamide administered for a reasonable period prior to the onset of fever, suggests the possibility of having a distinct advantage, certainly in selected cases, over the usual procedure of initial treatment by fever therapy.

In three of the cases reported in this series, bismuth in the form of bismuth salicylate in oil or iodo-bismitol was given bi-weekly in conjunction with tryparsamide throughout the incubation period without any noticeable effect upon the period of incubation or course of the fever. However, because of its greater degree of kidney irritation and lesser tonic or resistance building qualities as compared with tryparsamide, it is our feeling that bismuth is better indicated as a post-malarial form of therapy alone or combined with tryparsamide.

Syphilitic primary optic atrophy has been a complicating factor in 14 of our total number of cases. An interesting feature in this connection is that this condition is definitely arrested and improved by continued inoculation in the few cases that fail to respond with actual chills and fever in the same manner as those who exhibit normal paroxysms. Two of the fourteen cases failed to respond to inoculation malaria but showed plasmodia in the blood stream. Visual disturbance was arrested and improvement maintained through continued inoculation supplemented by bismuth therapy. One had eight inoculations and the other 10 at intervals varying from two to four months. Both have useful vision in that they are able to go about without assistance but lack occupational vision.

Clinical and serological improvement from continued inoculation in all cases failing to respond with chills and fever as previously reported has been definitely borne out in these further observations. A marked improvement with pronounced gain in weight, remission of mental symptoms and favorable modification of the spinal fluid findings is the rule. It has been noted, however, that these cases tend to relapse clinically and serologically unless inoculations are continued or chemotherapy supplemented. Likewise, it is true that patients who respond to the first or second inoculation and terminate spontaneously after four or five paroxysms, relapse unless treatment is augmented by chemotherapy. On the basis of this observation, we have arrived at the conclusion that the greater the number of actual paroxysms, or the longer the plasmodia are active in the individual the sooner permanent clinical and serological results will obtain.

We are convinced from our experience that most patients who receive the benefit of several paroxysms, not less than 10 or as many more as the physical status will permit, are not likely to show clinical and serological relapse. However, as a safety measure and because of the relative resistance difference in the individual cases, it is our feeling that post-malarial chemotherapy in the form of bismuth or trivalent arsenicals or both should be continued routinely in all cases with or without the addition of tryparsamide as indicated until the blood Wassermann particularly has been satisfactorily modified or until adequate treatment for this purpose has been administered. While the spinal fluid re-

sponse following malaria therapy did not parallel the clinical response in most of our cases, yet it has been uniformly true that satisfactory modification has been rather prompt and in some instances the serology of the spinal fluid became entirely negative within a few weeks after malaria even though no change in the blood Wassermann was noted at that time.

Rather frequently we see an irregular temperature curve in our use of the quartan strain. Sometimes it is quotidian or more often a double cycle. The latter seems to occur especially when reinoculation is necessary to obtain response. It is obvious that the patient is better able to withstand a prolonged period of typical quartan malaria with its advantageous rest periods between the paroxysms. It has been our observation that small doses of quinine given over a short period will tend to reestablish the typical quartan curve in many instances. This is especially true in the double cycle type of irregularity in which as a rule one cycle predominates over the other by as many as one to three degrees.

The cardiovascular system seems to share in a definite way in the general improvement noted in our patients after malaria therapy. We believe that it is generally accepted that a higher incidence of cardiovascular diseases exists among Negro veterans admitted to Veterans' Administration Facilities than among the whites. It has been noted particularly with regard to the blood pressure readings that the decrease shown during the course of fever is continued to an appreciable extent following treatment. A study of the cardiovascular changes in general, consequent to therapeutic quartan malaria, is underway at this time.

A clinical analysis of the 346 patients treated from November, 1932, to November, 1938, reveals the following: The diagnosis of general paresis was made in 306 cases, tabo-paresis in six cases, meningo-vascular neurosyphilis in 33 and tabes dorsalis in one.

The average age incidence of the patients receiving this treatment in 1932 was 39 years and 2 months; in 1938, 45 years and 5 months.

Two hundred forty-two patients responded to the first inoculation, 71 to the second, 15 to the third and 18 failed to respond after repeated inoculations. Hence, there were 328 successful inoculations causing chills and elevation of temperature repre-

senting 94.7 per cent "takes" as against 91.6 per cent "takes" reported in the first series.

One patient responded with typical quartan paroxysms 18 days following inoculation, another after 56 days; but the average incubation period was approximately 33 days as previously reported. Malarial relapse occurred in 12 cases after intervals of 21 days to three months following apparent termination. Six of these occurred after quinine therapy; three following atabrine and three following spontaneous termination.

Clinical and serological relapse occurred in 14 patients at intervals of six months to two years following treatment. This group represents, for the most part, individuals in whom the syphilitic process was of long standing and resistive to all forms of treatment. In practically every instance the serological improvement in the spinal fluid or blood Wassermann or both failed to parallel the clinical response despite intensive post-malarial chemotherapy.

The results of therapy in this series of 346 cases are classified as:

GROUP I.

Complete remission with minor or no residual defects, 146 cases or 42 per cent. 108 of this number have been discharged.

GROUP II.

Good remission with mild to moderate residual defects, 103 cases or 30 per cent. These are showing excellent hospital adjustment and requiring limited amount of supervision outside of the institution.

GROUP III.

Unimproved or arrested with pronounced residual defects, 71 cases or 21 per cent.

GROUP IV

(A) Died directly or indirectly from malaria, 6 cases or 2 per cent.
(B) Died from other causes, 20 cases or 5 per cent.

It may be noted in summarizing the foregoing that of the 346 cases treated, 249 or about 70 per cent evidenced definite improvement. The unimproved or arrested cases showed marked mental dilapidation or evidence of gross brain damage prior to inocula-

tion. Cases were selected for inoculation primarily on the basis of the physical status. Hence, every patient regardless of the degree of clinical manifestations of mental deterioration was inoculated if the physical condition warranted with the hope that he might at least attain a more satisfactory hospital adjustment.

Death which occurred during the course of malaria or shortly thereafter was regarded as due directly or indirectly to malaria. Those listed as having died from other causes died from three months to four years following malarial termination and it appeared quite certain in each case that malaria was not a contributory factor among the causes of death.

The observations made in 346 colored patients inoculated with quartan malaria over a six-year period would seem to warrant the following deductions:

1. The conclusions as drawn in our initial study of the use of the quartan strain of malaria have been fully supported by the findings recorded in this progress report, and from this follow-up study the following additional conclusions seem warranted.

2. The clinical course of therapeutic quartan malaria has been milder and has presented a lower incidence of the relatively few complications previously reported during our early observations; although there has been no evidence of decrease in the virulence of the strain over the six-year period. The apparent absence of sexual parasites as observed since 1935 may be a factor in the low incidence of complications.

3. Tryparsamide therapy may be used beginning prior to or at the time of inoculation and continued to the initial paroxysm without appreciable effect upon the incubation period, percentage of "takes," or clinical course of the malaria. This drug administered for a reasonable period prior to the onset of fever apparently increases the therapeutic success of quartan malaria in neurosyphilis; it most certainly does in selected cases. It is believed that this combination method has a distinct clinical and serological advantage over the usual procedure of initial treatment by fever therapy.

4. Syphilitic primary optic atrophy is arrested and improved by continued inoculations in the few cases that fail to respond with actual chills and fever in the same manner as those exhibiting normal paroxysms.

5. Post-malarial chemotherapy in the form of tryparsamide, bismuth or trivalent arsenicals or a combination of these as indicated should be administered routinely as a supportive measure against clinical and serological relapse. It seems especially indicated as supplementary treatment in the cases failing to respond with chills and fever after repeated inoculations or in those terminating spontaneously after a few paroxysms.

6. It seems apparent from the clinical and serological observations noted in this progress report that the quartan strain of malaria is of great value as a therapeutic agent in the treatment of neurosyphilis and particularly in the Negro. It deserves a trial in every case in which the physical condition does not contraindicate its use.

ACKNOWLEDGMENT.

The author wishes to express his grateful appreciation to Dr. Edwin H. Lee for the splendid part he has played in this work and to all other personnel who have given assistance including particularly the laboratory technicians and nurses.

BIBLIOGRAPHY.

1. Branche, George C: Therapeutic quartan malaria in the treatment of neurosyphilis among Negroes. The Journal of Nervous and Mental Disease, Vol. 83, No. 2. February, 1936.
2. Solomon, H. C., and Epstein, S. H.: General paresis: Treatment by tryparsamide—induced fever sequence. Am. J. Syphilis, Gonorrhœa and Veneral Diseases, Vol. 20, No. 3, May, 1936.

DISCUSSION.

DR. WALTER L. BRUETSCH (Indianapolis, Ind.).—The work of Dr. Branche and his associates in Tuskegee represents an admirable contribution and has an important practical application. It also comes at the right moment, that is, just at the time when the enthusiasm for the treatment of general paresis with heat cabinets is, generally speaking, on the decline. It can be stated quite safely that the majority of state hospitals are using malaria treatment at the present time.

One disadvantage of the malaria treatment, using the tertian strain, is that some patients, in particular colored individuals, will not develop fever. Dr. Branche has solved this problem by using quartan malaria obtaining 94 per cent "takes," which is a remarkable accomplishment in an exclusively colored group of patients.

Now as to complications, with the exception of acute nephritis, which I have never seen using the tertian strain, there are apparently no serious complications in quartan malaria. Dr. Branche also stressed the absence of severe anemia following malaria treatment. Much has been said about the development of anemia following or as a result of the malaria treatment. My own experience over 15 years with this treatment is the same as Dr. Branche's, which does not bear out such statements.

The great disadvantage of the quartan strain is the long incubation period, varying from 35 to 45 days. In tertian malaria the incubation period ranges from 8 to 12 days; this is followed by 10 to 14 days with paroxysms, making a total of the entire treatment of about 28 days. This, of course, is a great saving of time for those patients who are not committed, but take the treatment in a general hospital.

The excellent clinical results obtained with the quartan strain are another important feature. It almost seems that these results are better than those obtained with tertian strain. This is apparently due to the fact that the patient inoculated with the quartan strain carries the plasmodia at least twice as long as the one inoculated with tertian strain; or in other words, the more malaria, the better the results.

The results seem to be less due to elevated temperature which, as Dr. Branche told us, average between 103 and 105 F. only. It has been shown by Frazier quite conclusively that temperatures between 103 and 105 are without significant effect upon spirochetes. Now, clinical and serological improvement in cases which failed to respond even to quartan malaria also showed favorable results, and this is further evidence that elevated temperature as the main factor in the treatment of general paresis has been greatly overemphasized.

There are many other good points in this paper, in particular the treatment with tryparsamide during the incubation period, on which one could elaborate. But I want to ask Dr. Branche a question. What is your experience with shipping your strain to distant points? Do you believe the plasmodia will survive a trip from Tuskegee to Indianapolis, for instance?

The reason I am asking this is that I am inclined to introduce in our hospitals a quartan strain, but we don't want to give up the tertian strain because we supply a great many physicians in the Middle West with the tertian strain, and those physicians treat patients who are not committed to state hospitals, and there, of course, the time-saving element is an important factor. But for cases treated in institutions, I would not be surprised if the quartan strain were superior.

I am certainly delighted to discuss this paper, and I believe it is one of the best contributions which has been made in recent years in the treatment of neurosyphilis.

DR. JOHN E. LIND (Washington, D. C.).—Dr. Branche's paper is of especial interest to myself and the other physicians from St. Elizabeth's Hospital, in that it parallels our own experience to a large extent. We gave malaria to our first case of general paresis in December of 1920. At that

time our colored population comprised about one-fourth of our whole census, and that proportion has been maintained until the present time, although now we have fifteen or sixteen hundred patients. Consequently we had some opportunity to observe that in those patients some more effective therapy had to be found than the tertian malaria.

Various theories have been advanced as to why the colored race is more or less immune to malaria, especially the benign forms; the most logical probably being that since malaria is essentially a tropical disease, there is some sort of inherited immunity. At any rate, we have found, as others have found, that the colored patient didn't take tertian.

We have therefore been using quartan malaria for a number of years. In general, we have come to the same conclusion as Dr. Branche, with a few differences. For example, where he reports over 90 per cent successful inoculations, our figure is only 53.2 per cent. Our therapeutic results, moreover, were not quite so favorable as he quotes.

There are, perhaps, several explanations for these differences. In the first place, our subjects included a large percentage of female patients, and in the second place, our colored patients were pretty far advanced cases, much more so than the white cases. We explain this by the assumption that they are drawn on the whole from a social and economic group where the earlier signs of the disease attract less attention. It is not at all uncommon—in fact, it is a weekly occurrence—to have colored patients admitted with almost non-understandable speech, ataxic gait, marked confusion and even incontinence, and to have the relatives date the onset of the illness back only a few months, or even a few weeks, when in reality it must have been a year or two, or even more.

As to Dr. Branche's observation that the administration of tryparsamide up to the time of the first paroxysm increases the therapeutic effect of the malaria, even though he has the endorsement of Dr. Solomon and Dr. Epstein, I can only say that in our cases, which number roughly around five hundred, we have not been able to observe this effect. Nor have we noted that syphilitic primary atrophy was arrested or improved, even with poor paroxysms. Here again, however, there may be enough difference in Dr. Branche's material to account for this.

We are in full agreement with him as to the value of post-malarial tryparsamide therapy, together with bismuth.

We are in complete agreement also with his final conclusion that quartan malaria is of great value in paresis in the Negro; in fact, we would go further and say that it is the best method yet available.

DR. BRANCHE.—In answer to Dr. Bruetsch's question with regard to our experience in shipping our strain to distant points, I should like to state that we have not experienced any difficulty in shipment over long distances. The usual method is to prepare the blood by the addition of 1 cc. of a 2 per cent sodium citrate solution to each 10 cc. of blood and ship in an ordinary thermos jug. We have shipped it as far as Taft, Oklahoma, St. Louis, Mis-

souri, Danville, Illinois, and Kecoughtan, Virginia, requiring perhaps two to four days from the time the blood is drawn, and we haven't had any difficulty thus far.

With reference to Dr. Lind's percentage of only 53.2 successful inoculations, I am wondering what methods of inoculation he used. Ordinarily we use the subcutaneous or intramuscular route. If we fail there we use the intravenous method. Usually with the latter we secure a favorable response, sometimes after having tried the former method three or four times.

I am very grateful to both Dr. Bruetsch and Dr. Lind for their discussion.

Comment.

INDEXING ELECTROENCEPHALOGRAPHIC RECORDS.

Dr. Leon J. Robinson of the Monson State Hospital, Palmer, Mass., has submitted the filing card reproduced below for recording encephalographic data in epileptic patients.

This card measures approximately 5″ x 8″ and contains in the upper portion the pertinent clinical data including type of seizures (grand mal, petit mal or psychomotor attack), medication, etc., and in the lower portion the encephalographic findings. In the latter portion the six horizontal lines correspond to the usual scalp areas, and the three extra spaces provide for other areas as desired. The fourteen columns each in the resting and hyperventilation records are for registering the voltage and then in order the wave formation as spike wave, fast activity, one per second to ten per second waves, and, lastly, for localization out of phase. To indicate the presence of any of these data a plus sign is placed in the appropriate space.

The cards are filed alphabetically.

For the recording of E. E. G. findings the department of medical research, University of Toronto, uses 3″ x 5″ cards in six colors to indicate different clinical conditions. A sample card is reproduced herewith. The clinical conditions presently indexed are: (1) epilepsy, (2) psychosis, (3) tumor, (4) other organic neurological conditions, (5) behavior problems, psychoneurosis, (6) normal.

Dr. G. E. Hall, professor of medical research, University of Toronto, offers the suggestion that other workers in this field be invited to submit the recording devices which they have found most useful for their respective purposes. By comparing the recording methods in use at various centres it is possible that a standard form of indexing might be agreed upon and generally adopted to facilitate the interchange of information.

To this end it is hoped, therefore, that those interested will send to the JOURNAL sample forms or suggestions for indexing electroencephalographic records.

ELECTROENCEPHALOGRAM

Name	SEX	AGE	DEPT.	Date	Fam. Hist.	Clin. Diag.	Date Onset	Duration	Date last Seiz.	Seizures			Med.	I. Q.	Other
										Gm	Pm	PS			

	Resting																Hyperventilation															
	V	S	W	F	A	1	2	3	4	5	6	7	8	9	10	^ V	V	S	W	F	A	1	2	3	4	5	6	7	8	9	10	^ V	Seiz.	Other
RF																																	
RM																																	
RO																																	
LF																																	
LM																																	
LO																																	

INTERPRETATION: NORM. ABN. DOUBT. NORM. ABN. DOUBT.

REMARKS:

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7-1/2 Right Sided	17 M	4-1/2	LM-LP	DOE, JOHN
m per Sec.	25 per Sec	OTHER	FOCUS?	AGE 46 M Jan. 20/39 G-5 A-60085

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 20 30 40 50 60 70

Diag. CARCINOMA SECONDARY - Left Rolandic Area (Died)

E. E. G. Findings

Alphas slow (7-1/2 sec) and coming chiefly from Right Side.
Lack of alpha activity indicates LM-LT Areas.

Slow waves generally over entire Left cortex, with most frequent foci around LM or slightly anterior to LM.

[OVER]

Pathological Findings

(At autopsy) Large tumour approximately 1-3/4" diameter, coming to surface almost exactly under LM electrode. Considerable oedema on Left side.

News and Notes.

MISSISSIPPI VALLEY MEDICAL SOCIETY 1940 ESSAY CONTEST.—The Mississippi Valley Medical Society offers annually a cash prize of $100, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics) and practical value to the general practitioner of medicine. Certificates of merit may also be granted to the physicians whose essays are rated second and third best. Contestants must be members of the American Medical Association who are residents of the United States. The winner will be invited to present his contribution before the next annual meeting of the Mississippi Valley Medical Society at Rock Island, Ill., Sept. 25, 26, 27, 1940, the Society reserving the exclusive right to first publish the essay in its official publication—the *Mississippi Valley Medical Journal* (incorporating the *Radiologic Review*). All contributions shall not exceed 5000 words, be typewritten in English in manuscript form, submitted in five copies and must be received not later than May 1, 1940. The winning essay of the 1939 contest appears in the Jan., 1940, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.). Further details may be secured from

Harold Swanberg, M. D., Secretary,
Mississippi Valley Medical Society,
209-224 W. C. U. Building, Quincy, Ill.

REPORT OF NOMINATING COMMITTEE.—In accordance with the provisions of Article VI of the Constitution of The American Psychiatric Association, the Nominating Committee herewith reports the following nominations for officers of the Association for consideration and action at the 96th annual meeting to be held in Cincinnati, Ohio, on May 21, 1940:

For President: George H. Stevenson, M. D., Ontario.
For President-Elect: H. Douglas Singer, M. D., Illinois.

For Secretary-Treasurer: Arthur H. Ruggles, M. D., Rhode Island.
For Councillors for three years: William C. Sandy, M. D., Pennsylvania; Frederick W. Parsons, M. D., New York; Bernard T. McGhie, M. D., Ontario; LeRoy M. A. Maeder, M. D., Pennsylvania.
For Auditor: Horace G. Ripley, M. D., Vermont.

HENRY I. KLOPP, M. D., *Chairman,*
TITUS H. HARRIS, M. D.,
C. M. HINCKS, M. D.,
GLENN E. MYERS, M. D.,
WILLIAM J. TIFFANY, M. D.

ANNUAL MEETING, SOUTHERN PSYCHIATRIC ASSOCIATION.—The Southern Psychiatric Association held its annual meeting in Louisville, Kentucky, October 10 and 11, 1939, and was entertained by the Kentucky State Psychiatric Association and the Kentucky Mental Hygiene Association.

The meeting was well attended and over a hundred applicants for seats at the banquet, which was held in the large ballroom of the Brown Hotel, had to be turned away because of the lack of seating capacity. Dr. Arthur H. Ruggles, secretary of The American Psychiatric Association and Dr. Irvin Abell, former president of the American Medical Association were guest speakers at the banquet.

The 1940 meeting will be held in Jacksonville, Florida, but because of the climatic conditions will meet in the latter part of November or early part of December.

The officers elected for 1940 are: Dr. C. D. Mitchell, Whitfield, Miss., president; Dr. Arthur J. Schwenkenberg, Dallas, Texas, president-elect; Dr. William E. Gardner, Louisville, Ky., vice-president; Dr. Newdigate M. Owensby, Atlanta, Georgia, reelected secretary-treasurer; Dr. Charles S. Holbrook, New Orleans, La., and Dr. J. K. Hall, Richmond, Va., councillors.

A committee consisting of Dr. Harry Stack Sullivan, Dr. Ross McC. Chapman, and Dr. Newdigate M. Owensby was appointed by the Association to promote national defense and to memorialize the Surgeon General of the Army and Navy and Public Health Service regarding the immediate appointment and instruction of recognized psychiatrists for duty in the event of a military or other

crisis. Dr. W. C. McConnell of St. Petersburg, Florida, was appointed to investigate the possibilities of establishing a Journal for the Association.

ROCKEFELLER GRANT FOR PSYCHIATRIC EDUCATION.—It is announced by Dr. Franklin G. Ebaugh, chairman of the committee on psychiatry in medical education that the Rockefeller Foundation has made a grant to The American Psychiatric Association through its committee on psychiatry in medical education for the organization and support of two teaching institutes for state hospital groups for the years 1940, 1941 and 1942.

The committee is now actively engaged in the organization of these institutes and the development of teaching content centering around psychobiology and psychopathology, clinical psychiatry and clinical neurology, pathology and neuropathology. The geographical areas in which these institutes will be conducted will be selected in the near future.

HOSPITAL LIBRARIANSHIP, UNIVERSITY OF MINNESOTA.—The Division of Library Instruction of the University of Minnesota announces that its course for the training of hospital librarians will be given for the fourth year during the spring quarter beginning April 1, 1940.

The field for this specialized type of librarianship is steadily, even if slowly, opening up as evidenced by inquiries received from hospital administrators. Beginning salaries average from $85 to $100 per month.

The individual subjects of the course remain the same as in other years: Hospital Libraries, Administration 3 credits; Book Selection for Patients 3 credits; Work with Mental Patients 2 credits; Medical Reference 3 credits; Six weeks internship 4 credits.

At present this is the only course in hospital librarianship leading to a degree in an accredited library school. All students who take this course for credit receive a special certificate authorized by the Regents of the University.

Inquiries for further information should be made as soon as possible and should be addressed to Mr. Frank K. Walter, Librarian, University of Minnesota, Minneapolis, Minn.

COLLOQUIUM OF PSYCHIATRISTS AND PSYCHOLOGISTS AT TUFTS COLLEGE.—More than sixty professional psychiatrists and psychologists from eastern Massachusetts were guests on the evening of November 13 of three members of the department of psychiatry of the Tufts College Medical School and the nine members of the American Psychological Association who are on the Tufts faculty at a meeting at the home of Leonard Carmichael, president of Tufts College and president of the American Psychological Association.

Among those who spoke briefly on the relationship between psychiatry and psychology were: Douglas A. Thom, professor of psychiatry, Tufts Medical School; Abraham Myerson, professor of neurology, Tufts Medical School; A. Warren Stearns, professor of psychiatry and dean of the Tufts Medical School; C. Macfie Campbell, professor of psychiatry, Harvard Medical School; Truman Lee Kelley, professor, Graduate School of Education at Harvard; Edwin G. Boring, professor of psychology, Harvard; Gordon Allport, professor of psychology, Harvard; Ross A. McFarland of the Fatigue Laboratory, Harvard Business School; Edna Heidbreder, professor of psychology, Wellesley College; David Shakow, clinical psychologist, Worcester State Hospital; Vernon Jones, head of the psychology department, Clark University; and E. Stanley Abbot, psychiatrist and psychologist of Boston.

President Carmichael's purpose was to make psychologists and psychiatrists better acquainted with each other's work and to explore further possibilities of collaboration. To this end he proposes holding similar meetings from time to time.

DISTRICT REPRESENTATIVES—AMERICAN PSYCHIATRIC ASSOCIATION.—With the approval of the Council, the President has designated twenty-four (24) Fellows of the Association as district representatives in the United States and Canada and they have accepted the assignment.

It is not expected that these representatives will be unduly burdened with responsibilities. They will, however, be furnished with appropriate letterheads and will be available to assist the Association from time to time in various ways such as in stimulating the

return of the completed biographical and military questionnaires to the New York office by psychiatrists who have neglected to send in their replies.

The full cooperation of the members of the Association with these district representatives will be very much appreciated. The following is the list of representatives, with the districts for which they are responsible:

UNITED STATES.

District.	Representative.
Virginia, West Virginia, North Carolina	Dr. R. Finley Gayle, Jr.
South Carolina, Georgia, Alabama, Florida	Dr. Roger W. Swint
Mississippi, Louisiana, Arkansas	Dr. C. S. Holbrook
Tennessee, Kentucky	Dr. Frank H. Luton
Illinois, Indiana	Dr. Charles F. Read
Michigan, Wisconsin, Ohio	Dr. R. Philip Sheets
Kansas, Missouri, Iowa	Dr. Emit F. Hoctor
South Dakota, North Dakota, Minnesota	Dr. William L. Patterson
Nebraska, Colorado, Wyoming	Dr. Howard A. LaMoure
Oklahoma, Texas, New Mexico	Dr. Arthur J. Schwenkenberg
Arizona, Utah, Nevada	Dr. Garland H. Pace
Idaho, Montana, Washington	Dr. William N. Keller
Oregon, California	Dr. Benjamin W. Black
Pennsylvania, Maryland, Delaware	Dr. Harry F. Hoffman
New York (Up State)	Dr. John L. Van De Mark
New York (Metropolitan)	Dr. George W. Mills
New Jersey	Dr. Guy Payne
Massachusetts, Connecticut, Rhode Island	Dr. Ralph M. Chambers
Vermont, New Hampshire, Maine	Dr. Carl J. Hedin

CANADA.

British Columbia	Dr. Arthur L. Crease
Manitoba, Saskatchewan, Alberta	Dr. Alvin T. Mathers
Ontario	Dr. B. T. McGhie
Quebec	Dr. C. A. Porteous
Nova Scotia, New Brunswick, Prince Edw. Isl.	Dr. R. W. M. MacKay

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COMMITTEE ON ARRANGEMENTS, CINCINNATI MEETING.—Attention is called to the fact that the chairman of the Committee on Arrangements for the 1940 meeting is Dr. Thomas A. Ratliff,

assistant professor of psychiatry, Medical College, University of Cincinnati. Correspondence for this Committee may be addressed to Dr. Ratliff at 991 Dana Avenue, Cincinnati, Ohio.

NEW STATE HOSPITAL IN NEW YORK.—Dr. William J. Tiffany, Commissioner of Mental Hygiene for the State of New York, reports that the state is acquiring 875 acres of land in Deer Park, Long Island, as a site for a new state hospital for mental patients. This land is adjacent to the property of the Pilgrim State Hospital, and it is expected that construction will begin the spring of 1940.

Chapter 466, Laws of 1939 appropriated $5,628,000 of bond money for the commencement of construction of a new state hospital to accommodate not less than 8000 patients eventually. This beginning of a new hospital will include a reception service with accommodation for 600 patients and infirmary service for 1500 patients; kitchen and dining facilities; superintendent's residence; medical and non-medical staff building for 50; accommodation for not less than 200 employees; garage facilities; power plant equipped to service not less than 2300 patients; water supply; sewage disposal; tunnels; corridors; heating, sanitary and electric service connections; roads, walks, grading, landscaping and other necessary facilities and appurtenances.

It is anticipated that the next legislature will allocate money to continue construction to include accommodations for continued treatment patients, staff and employees quarters and other necessary utility buildings.

The new institution will receive patients from Greater New York and it is hoped will somewhat relieve the overcrowding of other metropolitan state hospitals. The location is about 35 miles from New York City and is accessible by both railroad and highways.

DR. JELLIFFE HONORED BY THE DUTCH SOCIETY FOR PSYCHIATRY AND NEUROLOGY.—Professor B. Brouwer, president of the Dutch Society for Psychiatry and Neurology has announced that at a recent meeting of the Society Dr. Smith Ely Jelliffe of New York City was elected an honorary member in recognition of his services to psychiatry and neurology.

NEW ADDRESS OF ASSOCIATION HEADQUARTERS.—The business office and permanent headquarters of The American Psychiatric Association have been removed from the previous location in the Academy of Medicine, 2 East 103rd Street, New York City, to the R. C. A. Building, 50 West 50th Street, Room 921. From many points of view the new location is a more advantageous one particularly because of the numerous health and welfare associations having head offices in this building, and with which close association is desirable. The National Committee for Mental Hygiene occupies offices on the floor immediately below the new Association headquarters.

This very central location in New York City will serve the convenience of members wishing to visit the office, and in this way it is hoped will augment the service which the Association may render its members.

MOTION PICTURE EXHIBIT AT THE ANNUAL MEETING.—We wish to have an extensive and interesting showing of motion pictures at the annual meeting in Cincinnati. These will be shown as a separate exhibit, as at previous meetings. Those who have films will kindly communicate with:

Dr. J. D. Reichard,
U. S. Public Health Service Hospital,
Lexington, Kentucky.

Please give (1) title, (2) a brief summary of the subject, (3) sponsor or director of film, (4) footage or running time. Facilities will be available for 16 mm. film, either sound or silent.

A program of approximate times of presentation will be distributed at the time of the meeting.

It is hoped that sponsors of films will be present at showings to discuss the material and to answer questions.

NOTICE TO CONTRIBUTORS TO CINCINNATI PROGRAM.—Readers of papers at the Cincinnati meeting who have slides or motion

pictures to be shown in connection with the presentation should communicate with:

Dr. J. D. Reichard,
U. S. Public Health Service Hospital,
Lexington, Kentucky.

Please give author, title of paper, section, approximate time of presentation (*i. e.*, a. m., or p. m.), and type of material to be projected.

Early notification is requested. Failure to give such notification may result in lack of projection facilities.

ENVIRONMENTAL INFLUENCES.—"The conditions and accompanying strains of present day living have a definite effect on the health of the individual. It is well recognized by those familiar with these problems that serious disability often arises from the impact of environmental factors. The social, economic and emotional influences on the health of the individual have had far too little attention although they represent the essential contributing elements in many illnesses and are important in the diagnosis, prognosis and therapy of a large proportion of medical problems. Insufficient emphasis on these phases constitutes one of the major defects of medical education and medical services, developed as they have been in recent years so largely on the more objective laboratory and technical advances."

The above quotation is from the presidential address, "The Challenge to Medical Education," delivered by Willard C. Rappleye, dean of Columbia University College of Physicians and Surgeons, at the fiftieth annual meeting of the Association of American Medical Colleges, at Cincinnati, Ohio, October 23-25, 1939.

NEW COMMITMENT STATUTE FOR ILLINOIS PREPARED BY THE ASSOCIATION OF FORMER PATIENTS.—The Executive Committee of Recovery, the Association of Former Patients of the Psychiatric Institute of the Illinois Research and Educational Hospitals, announces the completion of the preliminary draft of a new commitment statute. The statute was prepared with the aid of faculty

members of Northwestern University Law School and will be submitted to leading psychiatrists and jurists for criticism and suggestions. After its final revision has been effected, it will be introduced in the next regular session of the Illinois legislature. The main features of the proposed statute are the abolition of court action and the elimination of the "court record." Under the new plan a patient, after proper certification by two physicians, will be admitted to a state hospital without petition, writ or trial. The hospital staff will be required to make an examination within ten days of admission and to send a report to a State Board of Supervisors composed of physicians, lawyers and lay people.

The Recovery Association was founded November, 1937, by 30 patients who were discharged from the Psychiatric Institute as recovered. Today it comprises as dues paying members close to 150 former patients and upward of 500 relatives and friends. The Association publishes the bi-monthly journal, *Lost and Found*, in which the adjustment problems confronting the recovered patient are discussed. Recovery has the endorsement of the University of Illinois, the State Department of Public Welfare, the Illinois Psychiatric Association and the Illinois Society for Mental Hygiene. Inquiries should be addressed to Recovery, 1819 West Polk Street, Chicago, Illinois.

Book Reviews.

THE ORGANISM. A HOLISTIC APPROACH TO BIOLOGY DERIVED FROM PATHOLOGICAL DATA IN MAN. By *Kurt Goldstein, M. D.* (New York: American Book Company, 1939.)

The text contains 12 chapters, an introduction and concluding remarks, totaling 519 pages. The foreword is by K. S. Lashley; there is a creditable bibliography; relatively few of the references are in the English literature; there is a complete index. The book is essentially a translation by the author of the German original, with certain additions and omissions, and the difficulty of converting the spirit of thinking in one language into another is recognized. It is excellently edited, but is difficult to read: "Catastrophic reactions of the organism . . . . to its milieu" is used in the text to mean biological failures.

The author is a physician, a neurologist with unusual background in the biological sciences, who has lost faith in the practical value of scientific theory, lectures the biologist and proposes interesting new approaches to the understanding of the organism as a whole. He sponsors the empiric approach as opposed to the atomistic, the integrated whole in contradistinction to its parts, in a study of the organism and gives repeated examples and hypotheses to substantiate his views. He begins with the study of the complex integrated whole and then tries to understand its parts; he begins with the highest and most complex organism, man, and tries to derive from it guiding principles in studying living organisms at lower levels; he proceeds from pathological phenomena, especially with respect to the nervous system, rather than from the normal, in gathering insight into the processes of the organism; he reasons from effect to cause; he is more concerned with the problem of being sick than that of disease; he reasons always from the "holistic entirety."

The author's wealth of clinical experience in cases of head injury and his antipathy for anthropomorphism, are welcomed heartily by the clinician but this enthusiasm will fade as the reader proceeds into the involved formulations of the subject matter. The author utilizes animal experimentation and laboratory findings in many instances to prove his own theories. However, a large portion of the book is devoted to analytic critique of the usual methods of investigation. He is critical especially of the usual destructive isolating methods of research and compares his holistic approach to biology with Gestalt psychology; the author is a Gestaltist; he refers to "physiological Gestalten" and finally arrives at a biological Gestalten, to which psychological Gestalten is apparently subservient.

The author's wide experience with cortical lesions, his thoroughness in dealing with patients, and his ingenious "method of determining symptoms" are at once obvious. Contemporary thought of recognized neurophysiologists is critically and constructively utilized. The author's critique of the theory of localization, of simple reflex mechanisms and of physico-chemical analyses of physiological processes is interesting and well done.

The author believes that knowledge based upon facts is always of a positive character; reflexes are real performances which are not based upon negative factors, *e. g.*, removal of cortical inhibitions. In normal life, if emergencies arise, reflex reactions may arise as a relative expression of meaningful response to stimuli. They are the most primitive method of supplying protection against destruction. The highest organisms manifest reflex-behavior much more frequently than the lower organisms. He analyzes the functional significance of various reflex phenomena, believing it to be futile to try to understand one pathological phenomenon by attributing it to the loss of function of another. It is stated dogmatically that conditioned reflexes are functions of the cerebral cortex and represent "drill results." Also, man differs from lower animals in that conditioned reflexes are subservient to volitional performance. In general the author believes that the reflex theory is inadequate to explain normal behavior because reflexes, as such, occur only in artificially isolated parts of the nervous system. He feels that errors of reflexology are traceable to faults in the method of procedure. Both reflex action and instinct are considered holistically, in terms of their reference to the whole organism. Instincts, however, belong more essentially to the living organism and are analyzed as processes in the nature of Gestalten. Drives are "impelling forces of self-actualization."

The theme of the entire volume is that each individual performance has a relationship to the whole; that any change in one locality in the organism is accompanied by simultaneous changes in other localities; that group movement is only the outstanding part of the whole event, consequently when the local outstanding movement or function is thwarted, as in paralysis, the excitation in the rest of the organism is disturbed and various associated movements or manifestations occur. The author uses numerous examples, including successful nerve transplantation, to illustrate his point that performance is always mediated from the total situation. This is a timely blow to the diagram makers of brain maps. It is emphasized that the character of symptoms which may result from a local cerebral lesion depends on many factors: the nature of the disease process, the condition of the entire nervous system, the state of the circulation, the constitution of the patient, the reaction of the patient to the difficulty, the difficulty of the performances which elicit the symptoms.

The various sense modalities, as expressed in language, are related to the other bodily processes, such as posture and tonus, and to moods and attitudes; perception is a specific pattern of the whole organism.

The theory of agonist and antagonist, of reciprocal antagonistic innervation of muscles, of reciprocal inhibition, of agitating and inhibiting humoral

mediators are criticized and postulated in terms of "physiological Gestalten." Elements such as reflexes and tonic relationships are explained merely as responses observable in the organism under certain strictly regulated sets of conditions.

There is never complete equilibrium between the organism and the world. This dis-equilibrium, when it reaches the point of failure of the organism in coming to terms with the world, results in serious "catastrophic reactions" which are subjectively experienced as anxiety. The object of this dysharmony is never recognized by the patient. Brain-injured persons surrender to the anxiety situation as long as they are not safeguarded through limitation of their world, in contrast to the normal person whose urge tends toward order, new experiences and expansion of his sphere of activity.

The terms "unconscious" and "repression," like "inhibition," express something negative and do not do justice to the positive aspects of life. "Repressed" material cannot be remembered because it is no longer part of the attitudes of the individual, there having been a continual formation of new patterns. The original material can be revived or recalled only if the individual is brought into a situation which is similar to that under which it originated: "What appears as the unconscious is nothing but the entry of a former reaction pattern of the organism into a present response, when the situation is suitable." The author believes the theory of psychoanalysis precludes the possibility of criticism because the principle itself always supplies auxiliary hypotheses to meet the situation.

Behavior may be described either through the index of the so-called mind or through the index of the body but "we must always bear in mind that . . . . we are dealing with data which have to be evaluated in the light of their functional significance for the whole." Any performance signifies the reaction of the organism to an environmental situation. Successful performance depends first on the starting position and second on the equalization process which tends toward the preferred situation: "The fact that definite kinds of performances occur in the situational preferred behavior becomes instrumental for determining what the essential attributes (constants) of the organism are." Attributes or constants which are achieved in the laboratory are reached because of artificial circumstances at the expense of performances very essential for the organism. "The tendency toward the good Gestalt finds its explanation as an organismic phenomenon. . . . . Only through analysis of the interactional totality of the outer and inner field do the reasons become clear just why a certain pattern, a certain action appears as 'good Gestalten.' . . . . One might merely realize that the best Gestalten means the best for a coming to terms of the organism and the world." The author then emphasizes that in order to understand human behavior one must realize that the organization of the human being consists of the potentiality to behavior partitively as well as holistically. It is admitted that the organism is only a part of the "greater entirety" and "we halt with the individual as a preliminary whole simply because we have arrived factually at a relatively satisfactory result."

"Being sick . . . . is a disturbance in the course of the life process . . . . and requires a conception of the individual nature as a starting point." Health consists of ordered functioning which is normal for the individual and is expressed in definite constants, responsiveness, and in preferred behavior. Rehabilitation or recovery after disease corresponds to adaptation to a new individual norm but entails some loss in "essential nature" if there are residual defects. The organism may become capable of relatively normal performances again, even if they may be somewhat circumscribed, but this is accomplished only by modification of the milieu.

There is a critical discussion of contemporary methods of genetics, eugenics and sterilization.

In chapter xi "On life and mind" it is not surprising that the author concludes that mind is part of the individual whole, as also are feelings and bodily processes. In discussing the "Heirarchic structure of life," also in chapter xi, there is failure to postulate the holistic theory clearly and the Gestalt is not mentioned in this part of the chapter. The psychiatrist who reads this volume is likely to interpret some of the author's formulations as metaphysical reasoning used in "the inquiry into nature."

"The organism . . . . does not commence with procreation . . . . and does not end with death," is one of the author's concluding remarks. The reviewer at times feels that he is vacillating between physical science and philosophical argument. The author finally arrives at the philosophical conclusion that man alone can modify his environment because he has the capacity to bear suffering. I doubt if the author can make such a deduction by following his own line of reasoning in analyzing the organism, nature and the universe as a whole.

The original edition in German, *Der Aufbau des Organismus,* published at The Hague in 1934, was reviewed in *The Journal of Nervous and Mental Diseases,* vol. 83, p. 107, 1936.

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The Endocrine Glands. By *Max A. Goldzieher, M. D.* (New York: London: D. Appleton-Century Co., 1939.)

It is becoming an increasingly dangerous task for one man to attempt to write a comprehensive treatise on even a limited aspect of medicine. The general impression left by Goldzieher's book upon the reader was that it was almost impossible for one person to make a success of the task which the author had set for himself. Sections with which one was less familiar read well and no inaccuracies were noted. On the other hand in other sections, the literature of which is fairly well known to the reviewer, a number of inaccuracies were obvious. It would appear that many of the chemical and physiological aspects of the subjects considered are inadequately treated and are not placed in the proper perspective.

The book is useful as a reference to the endocrinological literature but the general conclusion is that it serves as an argument for the preparation of monographs on very small divisions of the subject by workers who are thoroughly familiar with the field involved.

The English is fairly good although numerous mistakes can be noted. This is not the sort of book which one would recommend to students of the subject but it may well serve a useful purpose as a review for pathologists and clinicians who already have some familiarity with the field involved and who wish to find references to the recent literature.

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Mental Disorders in Urban Areas. An Ecological Study of Schizophrenia and Other Psychoses. By *Robert E. L. Faris and H. Warren Dunham.* (Chicago: The University of Chicago Press, 1939.)

This book represents an excursion of sociology into psychiatry. The result in facts is fruitful and important even though the findings are given an interpretation which is open to criticism; but it is only fair to say that the authors advance their explanations in a purely hypothetical manner.

In his introduction, Professor Burgess, whose sociologic work is the basis of this study, makes some interesting remarks. He states (p. 12), "Mental disorders appear to be more prevalent where the population is mobile and heterogeneous than where it is stable and homogeneous and where life-conditions are complex and precarious rather than simple and secure." Yet, following this there appears to be a contradictory statement, "The financial depression beginning in 1929 was accompanied by little or no increase in mental disorders," and the very important statement is finally made, "Mental disorder is interpreted as a phase of personal disorganization arising out of maladjustment in the social relations of the person." This latter statement is open to the criticism that the organic mental disorders certainly are not yet where they can so be interpreted, and the functional nature of dementia præcox and manic-depressive psychosis has yet to be established as a fact and is at present merely the absence of a well established organic basis.

Drs. Faris and Dunham entitle their research "An Ecological Study of Schizophrenia and Other Psychoses," and this is, in truth, an apt and important description. They follow in essence the classical description of the pattern of a city as set forth by Professor Burgess and study the rates of mental diseases in the various zones of a city. The book must be read to understand the nature of these zones, and whether these zones have importance for psychiatry or not, they have importance for those who wish to understand the city.

In these "natural" areas of the city, which range all the way from the high-priced apartment to what the authors here call "hobohemia," there are varying rates for mental disease. Thus, in Community I, which is a high-rental apartment house district, there is a rate of 111 for schizophrenia, and in Community 32, the hobo and central business district, the rate is

1195. Obviously, however, there is no etiologic relationship here. Districts select their occupants; hoboes congregate in "hobohemia" and hoboes have an enormous percentage of schizophrenics among them. This is shown by the fact that the female schizophrenic rate in Community 32 is low, yet while there are somewhat more male schizophrenics than female, the difference is not striking. "Hobohemia" attracts the male schizophrenic, but since there are few female hoboes, there are few female schizophrenics in "hobohemia." In other words, the relationship is not vital. Flies congregate where there is molasses, but the molasses does not create the flies.

Similarly the authors establish that in the rooming house districts, there is a large and disproportionate increase of paranoids. But the paranoid, in his natural evolution from social living to retreat, finally lands in the rooming house district where people live in contiguity but not in community. There the paranoid can be as isolated as a great city will permit him to be. It would make an interesting study to ascertain the number of brilliant young men and women who have passed upward from the rooming house districts to the better houses of the city, for by its very cheapness and isolation the rooming house is the place where the people going up come in casual and non-organic contact with the people going down. There is no causation whatsoever in the correlation that exists between paranoia and the rooming house district. On the way to the hospital for mental diseases, the paranoid reaches the rooming house as his next to last stop.

That alcohol and general paresis are associated with vice areas needs no special subtle analysis. Vice is largely described in terms of excessive alcoholism and sexual delinquency. An interesting correlation is that of the old age psychoses with areas of tenancy and, in fact, the increase of the senile psychoses in the admission rates of the state hospitals has become a hugely important social matter. It is not likely that people in tenement districts live longer than people in single family houses. It is much more likely that under the conditions of urban life, a mentally sick old person becomes difficult or impossible to care for in a small tenement. The social root of the commitment rate in this case, as in most, gives us no clue to the underlying etiology or fundamental factor in the disease process. The reactions of relatives in tenement houses to the helpless old man or woman who is quarrelsome, untidy and disturbed are different than those of relatives living in single houses with more facilities and more room.

Drs. Faris and Dunham make a little excursion of interesting type into psychopathology in the chapter on "Hypotheses and Interpretations of Distributions," and in one place they state as follows (p. 175), "If seclusiveness is the key trait of the schizophrenic, the explanation of the disorder lies in the cause of the development of this trait. It is important to determine whether the seclusiveness is due to an innate lack of sociability or to experiences which destroy the sociability."

It is probable that if seclusiveness is the key trait to the schizophrenic, then there is both an innate lack of sociability and experiences which help to destroy sociability, since individuals who lack this trait elicit experiences and find for themselves situations which injure their social relations. This I

have called the "evocative" theory, that is to say, in a certain measure people tend by their nature to evoke the kind of experiences they meet. A paranoid certainly will elicit persecution on the part of others. He asks for it, so to speak, and he gets it. The over-sexed find sexual experiences everywhere. I knew a man who, when he was in a city half a day knew the assessible brothels and the illicit liquor dealers of the community, and in a very short time had experiences corresponding to his penchant.

Whatever criticism may be made of the interpretations, these are modestly advanced and are buttressed or, at any rate, accompanied by important facts. The psychiatrist who is eager for the advancement of psychiatry will make this book a part of his library and will welcome these sociologists and the discipline they represent as his allies in the campaign for the conquest of mental disease.

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CHILDREN IN FOSTER HOMES. By *Marie Skodak, Ph. D.* Iowa: The State University of Iowa Studies, Volume 16, No. 1, 1939.)

Director Stoddard of the Iowa Child Welfare Research Station in a pertinent foreword explains: "The present study traces in some detail the mental growth of a large group of (pre-school) children placed early in foster homes, relating the work not only to other studies in the Station, but to the publications of Burks, Freeman, Jones and Leahy." The main finding of the study in significant: "In general the intelligence quotient of a foster child during the pre-school years is related not to the genetic constitution as derived from true mother and true father, but to the quality of the home in which he now lives."

This raises the continuing problem of heredity versus environment. The latter supposedly determines the mental equipment of these young children as measured by their intelligence quotients. This ignoring of the biological origin of the child is contrary to the great mass of traditional knowledge and commonsense. Hence we would question the validity of the intelligence quotients given as offering a true criterion of real ability. In this connection it is worthy of note that intelligence ratings of young children vary considerably from time to time.

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THE HYPOTHALAMUS. MORPHOLOGICAL, FUNCTIONAL, CLINICAL AND SURGICAL ASPECTS. By *W. E. LeGross Clark, John Beattie, George Riddoch and Norman M. Dott.* (Edinburgh and London: Oliver and Boyd, 1938.)

In this publication the four authors have done a valuable service in assessing present knowledge of the structure and functions of the hypothalamus, as they view it from their different angles of approach.

Professor Le Gros Clark's summary of the morphology of the hypothalamic nuclei is very clearly written and includes many valuable original contributions which he has made to the subject from his studies of human material.

Professor John Beattie's contribution to the book deals mainly with the experimental physiology of the subject. He draws some general conclusions as to function which may be of value as working hypotheses in clarifying knowledge of the intricate mechanisms of the hypothalamus.

Dr. Riddoch deals with the subject from the point of view of the clinical neurologist. He gives a very satisfying description of the many clinical syndromes which have been attributed to hypothalamic dysfunction.

The surgeon's point of view as set forth by Mr. Dott, necessarily overlaps, to some extent, that of Dr. Riddoch. He adds to the book technical information as to the operative approach to hypothalamic lesions.

Dr. Riddoch's and Mr. Dott's sections of the book are well illustrated with clinical case reports.

The bibliography, to which all the authors contribute, adds to the value of the work as a book of reference.

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SOUTH ITALIAN FOLKWAYS IN EUROPE AND AMERICA. By *Phyllis H. Williams.* (New Haven: Yale University Press; London: Oxford University Press, 1938.)

This is the second in a series of handbooks embodying the results of studies made by the Institute of Human Relations into the urban population of New Haven, Conn. It is concerned with one ethnic group of the population of that city, namely the South Italians, and is largely a compilation of facts about the culture of that group. Culture is treated in the rather formal sense established as the Yale tradition by Sumner, and the folkways are set forth in detail as being important in themselves. That is to say, this study was made neither as part of the solution of any theoretical problem in sociology nor as an antiquarian investigation, but as a necessary aid to those practical people mentioned in the subtitle, who carry on their work among the foreign communities of North American cities and who therefore need accurate information as to wherein those foreign communities differ from communities with "Anglo-Saxon" or "old American" backgrounds. This purpose Miss Williams' book serves admirably. The material is arranged under such chapter heads as "Diet and Household Economy," Religion and Superstition," "Recreation and Hospitality," "Care of the Aged," etc., and there is a good index.

The author is so true to her main purpose of providing useful information that she does not follow up any sidetracks, no matter how alluring they may be. Hence the only criticism one can offer of her book is that such pertinent questions as the extent to which European folkways fall into disuse with increasing Americanization of the younger generations, are not discussed. She does indicate, in several places, that certain folkways, prominent in the first generation immigrants' behavior, persist almost unmodified in later generations. It may be that this is the case often enough, not only among South Italians in New Haven, but among foreign groups generally, to cause

us to question whether recent "assimilation" literature has not been too inclined to exaggerate the gap between first and second generation immigrants, and hence to overlook the importance of the ancestral folkways in the behavior of American-born children and grandchildren of European immigrants. This book should do something to counteract such a tendency, and further books in the series, dealing with other European groups in New Haven, will be welcomed. Badly needed, however, are concrete studies of particular individuals of foreign parentage or grandparentage, to ascertain how persistent in particular lives are the folkways of European countries.

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Mental Health. *Forest Ray Moulton*, Editor, and *Paul O. Komora*, Associate Editor. (Lancaster, Pa.: The Science Press, 1939.)

This volume contains the fourth symposium of The American Association for the Advancement of Science in the field of important problems of public health. It is the first time that the subject of mental health and ill health has been dealt with as a major issue by this premier scientific body.

The symposium which was under the immediate supervision of Dr. Walter L. Treadway, Assistant Surgeon General, U. S. Public Health Service, was organized in collaboration with The American Psychiatric Association and with the cooperation of the U. S. Public Health Service, The National Committee for Mental Hygiene and the Mental Hospitals Survey Committee, and formed part of the regular mid-winter program of the A. A. A. S. at its meeting in Richmond, Va., December 28-30, 1938.

The 49 papers and 41 discussions, summaries and critiques fairly represent American psychiatric and mental hygiene findings and opinions of today. In his address of introduction to the symposium Dr. Thomas M. Rivers, medical director of the Hospital of the Rockefeller Institute for Medical Research, made this significant statement:

"It may be the great contribution of psychiatry in the future to describe and define the art of medicine in more precise and useful terms by insisting upon the comprehension of the physician as one of the factors in the prevention and cure of disease."

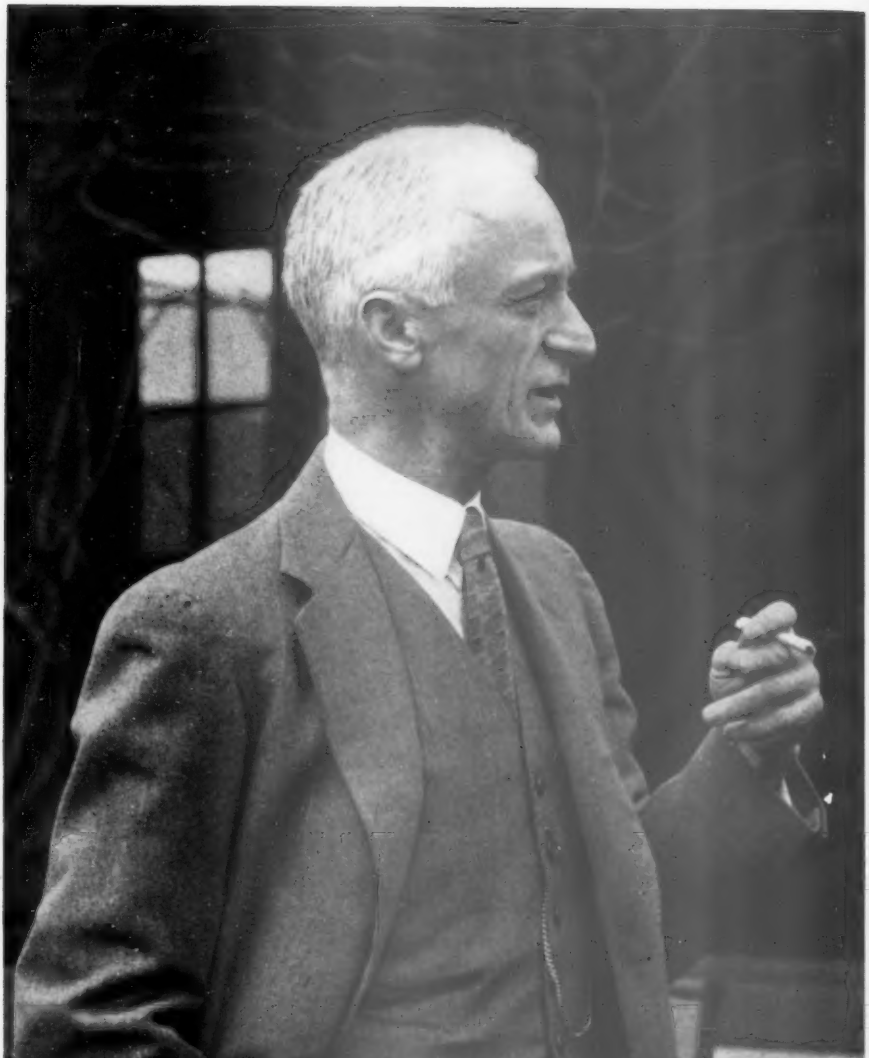
The material of the symposium is arranged in six sections, (1) psychiatric research, (2) sources of mental disorder and disease, (3) economic aspects of mental health, (4) physical and cultural environment, (5) mental health administration, (6) professional and technical education.

Under Dr. Treadway's admirable direction comprehensive programs were built up under each of these sections with authoritative contributors representing the various fields. After the papers in each section had been presented critical summaries were provided in invited formal discussions. Dr. C. Macfie Campbell closed the symposium with an address on "Human Needs and Social Resources" wherein he reviewed present outstanding issues in the psychiatric field, the problems which confront us and the resources with which they are being met.

The volume is a sizeable one containing 470 double column pages. Individual contributions include selected bibliographies which add to its value as a work of reference.

Reading this book recalls an earlier world report on mental hygiene, namely, the Proceedings of the First International Congress held in Washington in 1930 and issued by The National Committee for Mental Hygiene. The two works side by side on the library shelf will supply the student, research worker or social historian with authoritative material upon any phase of this all important public welfare problem.

C. B. F.



DR. HARVEY CUSHING.

UNIV. OF MICH.

In Memoriam.

HARVEY CUSHING.

1869-1939.

Harvey Cushing, the fourth in a direct line of a family of physicians, was born in Cleveland, April 8, 1869. He died October 7, 1939, from a sudden coronary occlusion, at the age of 70.

Dr. Cushing graduated from Yale in 1891, and from Harvard Medical School in 1895. After an interneship at the Massachusetts General Hospital he went to the recently opened Johns Hopkins Hospital at Baltimore; here he was resident in surgery for three years under the great Halsted.

Those were the days when the wards of a hospital were filled with typhoid fever. Dr. Cushing has told me how he and the resident in medicine would take turns sitting by the bed of a typhoid patient hoping to operate within an hour after a perforation of the small intestine. A patient with a gunshot wound of the cervical spine and cord damage came under his care. Fortunately the patient gradually recovered and Cushing spent hours charting the return of sensation and motor function. Also on this patient he helped make some of the earliest x-rays used on this continent. Thus did Cushing the hospital interne become marked as a worker.

Knowing him as I did later I am sure he was on the wards morning, noon and night working on his patients and seeing that they were looked after by nurses, junior internes and orderlies. There was nothing having to do with the care of one of his patients that was foreign to his interest. It would have been quite characteristic of him to interrupt and instruct an orderly giving an enema. His medical reading was stimulated by problems that his patients presented. He learned to sketch, and his drawings added greatly to his records and histories making them stand out above the average. Very early in his career he must have sensed that good records were all important not only for the welfare of the patient but also as a future basis for publication, either by himself or some one else. I remember him remarking that an interne only needed to leave the

hospital for a few hours once a month; he did advocate an hour a day at tennis or squash on the hospital grounds. He himself played tennis until well over fifty, when the blood supply to his lower legs became inadequate.

It has been said that Halsted recognized a brilliant pupil—rather would I say that Halsted recognized a man who worked unusually hard and more efficiently than the average. Dr. Cushing's genius lay in the fact that with average mentality and health he set his course for the top and never became sidetracked or slackened the pace. This keeping up the pace could be sensed even in such a simple affair as playing a game of tennis. In his medical life the pace was terrific, it must have meant refusal to explore many a bypath for fear of losing the high ridge he had chosen to travel.

At the end of his interneship, which had thoroughly qualified him in general surgery, Cushing was 31. He then went abroad with the intention of learning what was to be known about the surgery of the nervous system. After a few months with Horsley, the outstanding pioneer in neurosurgery, he proceeded to Berne. There working in the laboratory under Kocher's direction, he studied the problem of intracranial pressure—a work which brought him widespread recognition. He then spent a few months with Sherrington at Liverpool. Thus equipped he returned to Baltimore and petitioned for a post as neurosurgeon in the clinic. Dr. Halsted was staggered by the proposal, he felt that there was no possible source of livelihood in neurological surgery, even Horsley did not limit himself to any such specialty. Orthopoedic surgery was suggested. However for the young Cushing it was to be neurological surgery—he had hitched his wagon to a star. If he could not start a clinical service in Johns Hopkins, he could at least work in a laboratory. This he did and with such good effect that he was able to write his book "The Pituitary Gland and Its Disorders." Neurological cases began to accumulate. Cerebral surgery at this time was in its infancy, it was still the day of mallet and chisel for entrance; of wound drainage and imperfect closure—fungus cerebri with ultimate infection was the almost inevitable consequence of most interventions for tumor. Dr. Cushing applied Halsted's use of interrupted fine silk sutures and demonstrated that suture lines in the scalp would stand up and heal, even in the face of intracranial pressure and tension on the suture line, when closure was made with a deep and superficial

layer of fine silk. When this principle was established he was able to operate on cases and to escape the previously staggering mortality rate from cerebral fungus and infection. He had reached the point where the medical profession could be told that an exposure for brain tumor was reasonably safe. The possibilities of removing brain tumors were becoming apparent. A big neurosurgical field was opening up.

The opportunity was there—a man of high calibre was needed. One can imagine no one better fitted than Dr. Harvey Cushing—a young man with courage, vision and great energy. Once he had set his mind on the problem nothing was to deter him. He continued to travel the road until his last monograph "The Meningiomas" was published shortly before his death.

In September 1912, he became professor of surgery at Harvard and head of the surgical service at the Peter Bent Brigham Hospital. This was a general surgery appointment but Dr. Cushing continued to limit his personal work to neurosurgery.

In 1914 war broke out, and Dr. Cushing immediately let it be known that his sympathies were with the allied cause. He organized and headed Base Hospital No. 5, from Boston. From then until his return to the Brigham in 1919, all his energies and abilities were centered on the various war neurosurgical problems that he encountered in France. During this period he kept a voluminous diary, part of which has been published.

On his return to the Brigham Hospital, neurosurgical patients rapidly accumulated. When I became Dr. Cushing's resident in 1922-23, the service was busy and well established with an average of 25 to 30 patients, the majority of them suffering from brain tumor. They came from all corners of the globe, many of them referred by leading medical neurologists. The time had not arrived when the general practitioner had become familiar with the diagnosis of a brain tumor. Today the general practitioner appreciates the frequency of such a lesion and as a result we have seen the establishment of neurosurgical clinics in all university and medical centers.

My appointment as Dr. Cushing's resident needs telling both for the opportunity of expressing gratitude for the privilege of working with him for a year, and also to put on record Dr. Cushing's dis-

posal of the prize money which goes with the Mickle Fellowship. The story is as follows:

In 1922, the Charles Mickle Fellowship of the University of Toronto was awarded to Dr. Cushing as the member of the medical profession anywhere in the world who was considered by the medical faculty of the University of Toronto "to have done most during the previous ten years to advance sound knowledge of a practical kind in Medical Art or Science." Dr. Cushing accepted the award of $1000.00 and requested the University of Toronto to send to Harvard one of its young graduates to work with him for a year. In this way I became his resident. During that year Dr. Cushing passed the Mickle prize fund on to me, evidently realizing how helpful financial assistance often is to a young doctor.

The first contact with "the chief" in his office in the Peter Bent Brigham Hospital in Boston still stands out clearly—it was a hurried interview, only a few minutes, but long enough for me to realize that the faster I got to work and the harder I worked, the better. He had a few words of advice: above all, histories were to be kept up, examinations were to be complete. This was to be the resident's real contribution for the opportunity of working on his cases. I was to make sure that he would never look up one of my case histories in future years and find it inadequate. Later in that year (1922), I can recall showing some of my case histories with considerable pride to Dr. Fred Banting when he spent a few hours at the hospital before giving one of his early presentations before the Harvard medical students, on the discovery of insulin.

The first few weeks as resident were difficult to say the least. I had never before to my knowledge seen a patient suffering from a brain tumor. There were 20 or more such patients in Cushing's wards at all times. The first problem was to master an ophthalmoscope and learn to recognize the normal and pathological optic nerve head; this took some weeks. At the same time a simple but complete, neurological examination had to be applied; this included the taking of eye fields on a perimeter. Examination of a patient took hours during the first few weeks, later one learned to do it in a matter of minutes.

In looking back on my year with Cushing, I recall a man who must have been an extraordinary master of his time. His day's

routine seemed to be set weeks ahead and seldom did anything interfere with his plans. His office was in the hospital next to his laboratories, where his pathological material was worked up. At that time Dr. Bailey was working over his material for their book on "The Gliomas." There was also a room for making photographs of patients as Dr. Cushing had difficulty in recalling a patient when reading the history unless a photograph was attached. His day's routine was somewhat as follows: he arrived at the hospital about ten, having already spent two hours at home working on Osler's Biography. His study at home was filled with copies of the London Times, covering the period that Osler spent in England. Each copy was scanned for some item of news having to do with Osler. Having come across some such item as: Osler attended such and such a medical meeting, he would then write to men, whose names were mentioned as also attending, asking for further information. In this way he built up the second volume of Osler's Biography, a painstaking, tiring and probably a never-ending task for most men. When Dr. Cushing arrived at his office at ten, he spent the next hour or longer at his desk dealing with routine correspondence. His desk was kept clean. Besides answering letters to doctors and patients he sent off each day a number of follow-up letters keeping his brain tumor records up to date.

At about 11.30 I would come down to tell him that the patient to be operated upon that day was anaesthetized and ready. He might ask his associate, Dr. Horrax, to commence the operation, more frequently however, he would commence himself. There were usually visitors in the gallery. His team consisted of nurse anaesthetist, two junior house surgeons, one as second assistant and the other passing instruments (a training our junior internes seldom get today), a scrub nurse and his resident surgeon, who acted as first assistant. His operations were a delight to watch, careful, accurate work, without fuss. In those days cranial operations took longer than they do now. Not infrequently Dr. Cushing would put in the last suture himself and not leave the operating room until three or four o'clock. He then made himself tea and toast, had a shower, dictated an operative note, often two pages in length. Seldom did he plan a luncheon period. Then on with the next job—usually it meant seeing two or three office patients that had been worked up by his associate, Dr. Horrax. Perhaps a

patient had died and relatives had to be seen and permission obtained for an autopsy. He was always available for such a duty when his resident could not control the situation. Then an hour in the laboratory working on the glioma material with Dr. Bailey. About five his resident would succeed in getting him up to the ward to see a few special patients, especially the patient that was worked up for operation the next day. The case might be straightforward or might require a good deal of discussion and examination by Dr. Cushing. One watched him work to size up a case; ventriculograms were not common then. Often it seemed to be intuition on his part when he decided to expose the brain at a point well removed from the area where his resident had localized the lesion. He made mistakes of course but more often than not be was right. He bemoaned the fact that he did not have more time to spend on the case. His contact with patients was a delight to watch. Just a moment with a few, but he made the most of it. Often he would tell the patient that his pillow did not look comfortable, give it a punch, ask if that did not feel better, and be out of the room before the surprised patient could start a long string of questions. Following these necessarily brief rounds there was sometimes a half hour of tennis with some housesurgeons whom he would take home to dinner. After dinner the housesurgeons shifted for themselves while Dr. Cushing worked through until eleven or twelve on the Biography. On Saturday morning there was an additional routine: he personally checked over the week's histories and wrote letters to the doctors referring the patients. There was also a weekly clinical lecture to students, although I imagine that Dr. Cushing never had much use for the didactic lecture. I think he felt that a student's job was to work on patients and to read anything and everything that would help in looking after the patient concerned.

In sketching one of Cushing's typical days I have made it clear that there were few spare moments. Many men put in just as full a day, yet few I venture to say make each work period count as he did. There was an underlying plan and a long view plan. The ultimate goal was a knowledge of brain tumors, and the passing on of that knowledge to the medical profession. Each day something was accomplished toward this end, perhaps only a follow-up note on a beautifully worked up case history, perhaps a paper completed and published. If Dr. Cushing addressed a medical meeting his paper was always written beforehand and subsequently published.

Outside consultations were not accepted. Invitations to speak were only accepted when something worthwhile was ready for publication. In his publications he has covered the field of brain tumors so thoroughly that there seems little else to be said on the subject. Furthermore, he arranged for the follow-up of his two thousand verified brain tumor cases after his death. Dr. Louise Eisenhardt who had worked with him for years on his records, will ultimately complete this life work when all of these patients have died.

I would say that Dr. Cushing early in his career made up his mind to be a better housesurgeon than the average, this meant working harder than the next man. I heard him frequently tell an interne: "You have got to keep your eye on the ball." After establishing a record for hard intelligent work he had the genius to grasp an opportunity in a hitherto unexplored field. The driving force was certainly never financial gain, nor was it merely a desire to be the leader in his chosen field. He was the leader because of his burning enthusiasm to hand on to the medical profession a sound knowledge of brain tumors.

Dr. Cushing's biography is being written by Dr. John Fulton, professor of physiology at Yale. Trying to write such a short sketch as this gives one an inkling of what a task it will be. Yet it will be a labor of love and when completed will continue the stimulus which Dr. Cushing gave to a succession of young doctors in this country and abroad who set their sails under his guidance.

K. G. McKenzie, M. D.

PSYCHIATRIC APPROACH TO THE TREATMENT OF EPILEPSY.*

By STANLEY COBB, M. D., Boston.

Introduction.

Before discussing psychotherapy I consider it imperative to define two terms, "epilepsy" and "hysteria" because much confusion exists in the minds of clinicians about the relationship of these two disorders, and because I believe that there is no need for such confusion. Moreover, if any of the cases herein cited could be diagnosed as "hysteria," this communication would lose its point.

It is difficult to write a definition of epilepsy because it is not a disease entity; it is a symptom of many cerebral diseases. If one tries to divide all cases into "essential" ("idiopathic," "true") and "symptomatic," one is merely stating that in the former the etiology is unknown and in the latter it happens to be known. This is not classifying epilepsy; it is classifying the knowledge of the classifier. At present it seems probable that there are two main groups:

(1) Inherited cerebral dysrhythmia, where abnormal electroencephalograms are found in the parents of the patients who show clinical fits. This may well be a disease entity, and after more is known about it, the term "true epilepsy" may well be reserved for this group. Certainly the clinical work of Schreck [1] suggested this, and the important discovery by Lennox and Gibbs [2] of the familial occurrence of cerebral dysrhythmia, proves that the inherited disorder is much more specific and more common than was formerly supposed. Three varieties of this disorder may be postulated, (a) dysrhythmia discovered only by electroencephalogram, the disorder never becoming clinically manifest, (b) dysrhythmia with clinical

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fits but no cerebral lesion as precipitant (3) cases where trauma or other cerebral lesion seem to bring out the subclinical disorder and make it overt.

(2) Epilepsy caused by cerebral lesion, without inherited dysrhythmia. Here belong all those cases of repeated fits where trauma, inflammation, degeneration and tumor cause pathological alteration discoverable at autopsy. Not enough research has yet been done with the electroencephalograph to determine whether the inherited cerebral degenerations known to be accompanied by fits are to be classified with (1) or (2).

In the light of the above classification I will describe for the purpose of this communication what I mean by the term EPILEPSY:

A disorder of the brain that causes repeated fits in which the usual functional abnormalities are: (1) changes in the electrical potentials as seen in the electroencephalogram; (2) partial or complete loss of consciousness; (3) nervous discharge into smooth muscle, striated muscle or glands, causing involuntary visceral or motor behavior.

Having thus described as well as I can what these cases are, I wish to explain what they are not, *i. e.*, HYSTERIA:

A syndrome with widespread disturbances occurring usually in women of childish habitus and immature personality. The essential symptoms are psychological conversion and amnesia.

I thus try to make it perfectly clear that hysteria is a diagnosis to be arrived at by positive observation, not by elimination. Lindemann [3] has well described the syndrome and this point of view. None of the cases here recorded had hysteria. The two conditions are distinct and clinically recognizable; they should cause no confusion; of course, like any other two disorders they may occasionally occur together in the same patient.

The term "hystero-epilepsy" should be dropped. It is usually used either to describe an hysterical patient whose conversion symptom happens to be spasms of the striated musculature, or (more commonly) to describe epileptic patients who have emotional disturbances, anxiety and other psychological symptoms in addition to their epileptic fits. In fact, most people subject to seizures have anxiety as a result of the danger and the social stigma. Moreover, in many epileptic patients anxiety, specific fears, and even concrete phobias may act as precipitating causes for the epileptic attacks.

This is understandable if one realizes that the cerebral disorder of epilepsy is almost always accompanied by a lowering of the sensory-motor threshold, so that in some patients simple stimuli, such as a loud noise, a bright light, constipation, phimosis or acute infection may precipitate seizures. The low threshold and the fears are phenomena associated with epilepsy; they may be called the psychoneurotic concomitants of a chronic disease, but they have nothing to do with hysteria.

Historical.

Although much has been written on psychological methods in the treatment of epileptic patients,[4, 5] most of the material presented by the authors is difficult to evaluate accurately because not enough facts are given. It is said to be "well known" that anxiety, emotional stress[6, 7] and even simple sensory stimuli in excess[8] can increase the number of seizures. Lennox and Gibbs[9] have definite electroencephalographic evidence that increasing the use of the brain, as in mental arithmetic, decreases the number of abnormal waves. Therefore occupation in large institutions is used not only as a "pass time" but as therapy.

Observations.

During the past 20 years I have treated many persons suffering from fits. This material I have reviewed, and for the purpose of making an approximate evaluation of the use of psychotherapy I found it expedient to select 45 patients who were chosen because (1) they had been seen within the last three years, (2) they had been seen by me repeatedly and (3) they had received somewhat intensive therapy. These all are listed in three tables. All cases are listed that conform to these criteria.

The patients who received *psycho*therapy show data on the tables under the heading, "Treatment, Psychological." This is of four sorts:

(1) "Social, corrective" means that difficulties in the environment were ameliorated; often the mother was taught to show less apprehension and to be less obsessively solicitous about the patient's symptoms. Irritating siblings were kept away. The whole atmosphere of "invalidism" was combated in many ways.

(2) "Social, constructive" indicates that the patients were actually put into new situations with the aim of improving mental hygiene. Some were taken out of the home and sent to appropriate schools (cases 3, 9, 19, 22); others were put at special work; some were given small jobs in the hospital where they could have particular supervision and be away from the old atmosphere of invalidism and defeat. The variety of social reconstruction was almost equal to the number of the personalities concerned. These two categories of treatment are recorded in months, estimated from the record.

(3) Psychiatric "interviews" as a form of "TREATMENT, Psychological" means that the patients discussed their problems with me or one of my assistants in a more or less thorough way. This is recorded in number of hours.

(4) "Psychoanalysis" is used to mean a regular Freudian procedure by an analyst to whom the patient was referred. The number of hours is estimated and recorded.

Under "TREATMENT, Drugs" there are three headings, "Ph. Barb." meaning phenobarbital; "Brom.," bromide; and "Dilant.," dilantin. These are the three drugs that were mostly used and in the record of each patient on this table the signs "+, −, and o" are used to mean that the drug was increased in amount, reduced in amount, or that it was not used at all, respectively. Thus in case 12 there was an operation which was only moderately successful. Phenobarbital had been used for years before the operation; it was increased after the operation, but when the seizures continued in spite of it, I instituted social and psychological therapy and reduced the amount of the drug radically. It is obvious that the results were beneficial. This case was reported at length in 1936.[10] On the other hand, case 40 is an example where phenobarbital improved the patient without any psychological treatment, and case 42 was taken off phenobarbital and put on neoprontosil with remarkably beneficial results, though the picture is confused by the use of psychiatric interviews along with reduction in the number of seizures.

The column marked "Special" under "TREATMENT" is used to indicate when there were special procedures like operations, the use of neoprontosil as above, or the use of acidosis by the ketogenic diet. Case 21 is an example of marked improvement through the use of ketogenic diet with some improvement in home conditions.

Looking at Table I and comparing it with Tables II and III, it is obvious that in the last two columns the reduction in number of seizures (indicated by a minus sign in front of the number indicating per cent of reduction), and the improvement in personality (indicated by plus signs) is much more marked in the patients on whom many months of social treatment and many hours of psychotherapy are applied. As one would expect from the listing according to hours of treatment, five out of the first six patients were the ones who had psychoanalysis. Case 1 has only recently started analysis, so the improvement which has been marked for the last 10 years is not due to the analytical work, but to the social work, psychological interviews and luminal that were begun in 1921. The dilantin has only been used recently and the amount of luminal has been reduced in recent years. This is an excellent example of how mixed these data are, but at any rate the result has been gratifying in that the patient has not had a convulsion for almost five years and now only suffers from myoclonia, which is much less than it used to be. The other four patients who have had analysis are of especial interest because three out of the four have had a marked reduction in the number of seizures and one has had a remarkable improvement in social adaptation (case 3). Cases 5 and 6 have also improved in their economic efficiency. Case 3 is of especial interest because she inherited Friedreich's disease of a mild form and had the convulsions that are sometimes associated therewith. There was extreme apprehension and an atmosphere of invalidism, continually surrounding her all through her youth until she was taken away from home at the age of 15 and put in a school where she immediately had many less seizures and improved in her outlook on life. After three years of school she went to a distant city and had two years of analysis. She returned home having at last shaken off the invalidism and with an ability to meet her own problems. She now only has a seizure about once in six months at the time of her menstruation. Dilantin has been used in the last six months.

Case 5 is that of an English youth of 30 who had two convulsions as an infant, and no more until at college. Then he had four major convulsions each year for three years associated with athletics, late parties and alcohol. He occasionally had attacks of vertigo like his aura. During his last two years in college he went to a psychia-

TABLE I.

Case No. Age-Sex.	Clinical Type	Etiology.	Treatment.								Results.	
			Special.	Drugs.			Psychological.				Seizures.	Personality.
				Ph. Barb.	Brom.	Dilant.	Social. Cor. Con. Mos.		Int'w Analy's Hrs.		% Dec. − Inc. +	
44 1 F	Myoclonic (Conv.)	Heredity (? Enceph.)	Colectomy	−	0	+	180	160	60	80	−80	+++
66 2 M	Conv.	?		−	0	0	160	120	250	0	−95	+++
23 3 F	Lapses (Postural)	Friedreichs		−	0	+	72	60	65	550	−90	+++
48 4 F	Automatic	?		+	±	+	0	0	150	400	±	−
30 5 M	Conv.	?		−	0	0	0	0	40	600	−75	++
31 6 M	Confusion	?		−	−	0	24	24	0	500	−40	++
15 7 M	Rages Conv.	Trauma	Acid.	+	+	+	18	14	200	0	∓	+
8 F	Lapses (Conv.)	Trauma		−	..	+	7	7	140	0	−90	++
20 9 F	Confusion (Conv.)	Heredity		−	0	0	36	50	65	0	−50	∓
24 10 F	Conv.	? Trauma		+	0	0	12	0	60	0	−30	∓
26 11 F	Confusion (Conv.)	? Enceph.		+	−	+	4	5	60	0	+30	−
27 12 F	Conv.	Trauma	Operation	−	0	0	60	18	30	0	−80	+++
42 13 M	Conv.	Trauma		∓	0	0	0	0	35	0	+40	∓
38 14 M	Confusion (Conv.)	? Enceph.		+	0	+	0	0	40	0	−60	−
47 15 F	Confusion Fugues (Conv.)	Enceph.	Hospital	0	0	0	60	60	40	0	+	+
16 M	Conv.	Aplasia	Operation									

trist for help, having about 20 interviews a year. These helped him to understand his difficult family situation, his extraordinary drive to be a success in athletics and the college world. All his forbears had an aristocratic tradition of this sort. During his last year in college he had only two convulsions. Next year he entered a technological school in America and began psychoanalysis which continued for 20 months. The attacks dropped in frequency to two per year and finally, since analysis he has had a period of three years with only two attacks. The analysis brought out facts concerning homosexuality which were closely connected with his drive for success at school and college. Each of the last attacks since analysis has been associated with a reversion to homosexual temptation. The patient is perfectly convinced that he can avoid attacks by avoiding this adolescent form of sexuality. Throughout his illness he has been a good student, and since analysis he has been a steady worker. At times of stress during the year following analysis he took a small dose of phenobarbital at night.

Case 6 is that of a man who had many confusion attacks associated with an abnormal electroencephalogram and usually precipitated by emotional upsets. He had two years' analysis during which time he was away from a hard home situation. He has now returned home, has about 40 per cent less spells, and his personality has improved conspicuously, as is indicated by his ability to do effective work.

Case 4, which showed no improvement after analysis and has been steadily deteriorating, is that of a woman of 48 with seizures of the automatic-locomotor and dream-state type. They are of unknown etiology, but they are frequent, sometimes every day, and the electroencephalogram shows almost continuous abnormal waves. All sorts of drugs have been tried. She is a confirmed hypochondriac, is extremely stubborn and might be expected to be unsuccessful under any form of treatment.

From these cases it seems that the use of psychoanalysis in well selected patients is a desirable form of treatment.

Case 2 is of interest because in a man of 50 who had become an absolute invalid through anxiety over his rare convulsions, the most remarkable improvement was made by intensive psychotherapy carried on by means of psychiatric interviews and by taking him

away from his work for a year and then returning him to a new job where he was really happy and felt of real use in the world.

The last half of Table I and the first half of Table II show a number of patients who had little drug therapy and a large amount of social and psychiatric treatment. On the whole the results in this group are very encouraging, only four (7, 15, 21, and 31) are complete failures. In order to feel any conviction, however, that the results were because of the psychological treatment and not because of the drugs used, we must eliminate all in which the use of drugs or of special treatments was a factor. After eliminating these, we have left patients 9, 12, 17, 18, 19, 20, 21, 22, 23, 24, 25, and 26, in whom there was a decrease in the amount of drugs along with the use of psychological and social measures. It will be observed that most of these improved.

One of the best examples is case 8, a 25-year-old girl, single, began to have petit mal in early childhood. With the onset of menstruation she began to have grand mal; each attack occurred at about the beginning or end of the menstruation, and the attacks were quite frequent. She had a complex and tense home situation and craved affection. Although her only convulsive episodes appeared at the time of menstruation, they usually occurred when she had been under some emotional stress and there were frequent minor "lapses." Her grand and petit mal increased in frequency. She was interested in nothing but herself and was consumed with bitterness and self-pity. She was sent away from home to live in an apartment with a young psychiatrically trained nurse of her own age who works in close harmony with the psychiatrist. She was given opportunities to do volunteer social work at which she has been successful. For the first time in her life she has a sense of being worth while. Under moderate dilantin and phenobarbital medication she has remained free of seizures for six months, and her petit mal rarely appears.

Another eminently successful case is 12 who has been already mentioned and who was reported at a meeting of this association in 1933.[10] Case 18 is a young woman who was living in a rushing social and frivolous life with irregular hours and a moderate amount of alcohol and many cigarettes. Luminal had been used with no avail. On moving away from home, and later marrying a steady and devoted young man, she has improved remarkably in

TABLE II.

Case No. Age-Sex.	Clinical Type	Etiology.	Treatment. Special.	Drugs. Ph. Barb.	Brom.	Dilant.	Psychological. Social. Cor. Con. Mos.		Int'w Analy's Hrs.		Results. Seizures. % Dec. − Inc. +	Personality.
45 17 M	Conv.	Arachnoid.	Operation	−	0	0	36	12	15	0	−80	++
23 18 F	Conv. Myoclonic	?		−	−	0	10	36	12	0	−100	+++
26 19 F	Conv.	? Aplasia		−	0	0	24	16	10	0	−75	++
25 20 F	Confusion (Conv.)	Trauma Cyst	Operation X-Ray	−	−	0	30	2	30	0	∓	−
15 21 F	Lapses (Postural)	? Trauma	Aid.	−	−	0	30	0	0	0	−60	+
14 22 M	Lapses Conv.	? Aplasia	Acid.	−	−	0	16	16	0	0	−80	+
10 23 F	Lapses	?	Acid.	−	0	0	30	?	0	0	−60	+
21 24 F	Conv.	?		0	0	0	?	28	3	0	−30	+
23 25 F	Lapses Auto.	? Enceph.		−	0	0	15	2	20	0	−50	+
16 26 F	Lapses Conv.	Heredity ? Trauma		−	−	+	48	18	0	0	−90	+
36 27 F	Conv.	Trauma		+	0	0	28	36	3	0	−75	+
37 28 M	Conv.	Trauma	Operation	+	+	+	55	0	6	0	−90	++
19 29 F	Lapses Conv.	? Trauma	Acid.	−	0	+	18	12	6	0	−80	+
66 30 M	Confusion Postural Auto.	? Enceph.		−	−	+	36	2	10	0	−30	+
30 31 M	Conv. Rages	?		+	0	0	20	14	18	0	∓	−−

point of view and has only had one convulsion. Case 22 is that of a boy with petit mal who had three older brothers, all of whom were extremely active and he, although intellectually their equal, and perhaps their superior, was always stimulated enormously by the riotous household. He was away at school for two years and there had almost none of his lapses. On returning home for vacation with all of the Christmas excitement he had more and more lapses until one morning these became almost a status and ended in a major convulsion. On returning to his quiet school he once more had few lapses. In the light of this experience he is now growing up under wise supervision and has had no noticeable spells for over a year.

Table III, because it is made up of the higher numbers and therefore of those patients who had little or no psychological treatment, has varied forms of treatment under "Special" and "Drugs." In the upper half of this chart are some of our worst results, for instance, case 39 was a willful young woman with many psychological precipitants for her attacks. She insisted upon leading her own life in her own way, even though it was extremely unhygienic; she became worse and eventually committed suicide. Case 34 was obviously improved by the operations. Case 38 had all of her seizures at the menstrual period and was much improved by a régime of ovarian extract plus ammonium chloride for a week before each menstruation. The last four cases are patients on whom successful operations were performed and on whom the psychological therapy was either nothing or incidental.

Discussion.

Even a superficial study of the data presented in the charts shows that the treatment situation in almost every case was too complex for satisfactory analysis. The evaluation of any one procedure is impossible. It is of interest that social and psychiatric measures were employed in all but one of the cases, in varying degrees of intensiveness, but in only two (15 and 24) was psychotherapy the sole form of treatment. On the other hand, in some of those cases where drug treatment was definitely the mainstay, the decrease in seizures cannot be laid alone to the medication. For example case 42 was taken off phenobarbital and put on neoprontosil with excellent

TABLE III.

Treatment.		Results.
Drugs.	Psychological.	Seizures.

TABLE III.

Case No. Age-Sex.	Clinical Type	Etiology.	Treatment.								Results.	
			Special.	Drugs.			Psychological.				Seizures.	Personality.
				Ph. Barb.	Brom.	Dilant.	Social. Cor. Con. Mos.		Int'w Analy's Hrs.		% Dec. − Inc. +	
20 32 F	Confusion Conv.	Trauma	Operation Red Dye	−	+	0	0	24	4	0	+50	− − −
29 33 M	Lapses Myoclonic	Heredity		+	0	0	0	20	15	0	−50	+
27 34 M	Conv.	Trauma	2 Operations	−	0	+	0	20	5	0	−100	+
18 35 F	Lapses	? Trauma	Acid.	−	0	0	18	?	0	0	−70	++
20 36 M	Conv.	Trauma	Operation	+	+	+	0	20	4	0	+200	
22 37 M	Lapses Conv.	? Trauma	Acid.	+	+	0	18	0	5	0	+50	− −
33 38 F	Lapses Conv.	? Endocrine	Ovary. Acid.	0	0	0	0	0	10	0	−80	∓
30 39 F	Lapses (I Conv.)	Heredity		+	0	0	0	0	8	0	±	− − − −
12 40 F	Coordinated	?		+	0	0	2	0	0	0	+50	−
15 41 F	Conv.	? Trauma	Operation	+	−	0	5	0	0	0	∓	+
19 42 F	Confusion Conv.	Heredity	Pront.	−	0	0	2	1	25	0	−85	++
21 43 M	Lapses Conv.	Enceph.	Operation	+	0	0	0	0	8	0	−50	+
18 44 M	Conv. (Lapses)	Trauma	Operation	−	0	0	0	0	0	0	−75	±
81 45 M	Lapses C̄ Motor	Tumor	Operation	+	0	0	1	0	0	0	−100	±
64 46 M	Conv.	Tumor	Operation	0	0	0	0	0	0	0	−100	+

results and the improvement in social adaptation that came with the clearing of the phenobarbital dullness changed the patient's mood from dull and sad to alert and happy.

Tables I and II show those cases that were treated largely by psychotherapy; drugs were often reduced or not used; operations were exploratory only or unsuccessful. In other words the patient was passed on to me after the surgeon felt he could do no more, and seizures were still continuing. In 67 per cent of these cases there was marked improvement, in 10 per cent slight improvement, and 23 per cent were not benefited.

In conclusion I would like to give two quotations. The first is from James Jackson,[11] a wise physician of the first half of the nineteenth century. Writing in 1855 he said:

> The exciting causes of epilepsy are better known than the proximate cause; yet we cannot, in every instance, satisfy ourselves as to those. It would seem that any cause, disturbing the body or the mind, in an epileptic subject, may give rise to a paroxysm.

That holds true today. We have found the "proximate" cause of fits in many cases by the advances in our knowledge of pathology, but in most cases we still have to rely on treating the exciting causes, which may be varied and difficult to discover. Eighty years later at a meeting of the Association for Research in Nervous and Mental Disease, L. Pierce Clark[5] said:

> It is not possible to shirk the task of attempting continually to solve the problem of epilepsy. . . . . The epileptic and not the disease must be the main concern of therapeutics.

Little as I agree with Clark's concept of the "pre-epileptic character" and his interpretation of the fit as "a flight from reality," I admire his attitude towards the patient, and commend it to all physicians. It is obvious that one must attack a symptom such as the epileptic fit from a number of angles and not limit therapy to any one line. Although this may not give data that is easily analyzed by the epileptologist, yet all data on the treatment of human beings is so complex that conclusions cannot be drawn from a small number of cases, but must of necessity come from wide experience with a large number of similar cases observed over a period of years.

BIBLIOGRAPHY.

1. Schreck, Eugen: Die Epilepsie des Kindesalters, Ferdinand Enke Verlag Stuttgart, 1937, 173 pp.
2. Lennox, W. G., Gibbs, E. L., Gibbs, F. A.: J. A. M. A., 113: 1002, 1939.
3. Lindemann, E.: Med. Cl. N. A., May, 1938.
4. Rows, R. G., and Bond, W. E.: Epilepsy, London, H. K. Lewis, 1926.
5. Clark, L. P.: Psychobiologic concept of essential epilepsy. A. R. N. M. D., VII, p. 65, 1922.
6. Fremont-Smith, F.: Am. J. Psychiat., 90: 717, 1934.
7. Kardiner: Psychoanalytic Quart., Vol. 1, No. 3, Oct. 1932.
8. Goodkind, R.: Arch. Neurol. and Psychiat., 35: 868, 1936.
9. Lennox, W. G., Gibbs, F. A., and Gibbs, E. L.: Arch. Neurol. and Psychiat., 36: 1236, 1936.
10. Cobb, S.: Med. Cl. N. A., p. 1583, March, 1936.
11. Jackson, James: Letters to a young physician just entering upon practice. 344 pp. Boston, Phillips, Sampson & Co., 1855.

LEGEND FOR TABLES I, II AND III.

As described in the text these tables give an abstract of the record of 46 epileptic patients showing especially the kind and amount of treatment and under "RESULTS" showing an estimate of the effect of treatment. In these two columns under the heading "Seizures" is indicated an estimate of the percentage of decrease (Dec.—) or of the increase (Inc.+) in the number of seizures. Under the heading "Personality" plus signs indicate roughly the amount of improvement in social adjustment and minus signs the amount of deterioration.

The patients are arranged from 1 to 46 in accordance with the number of hours spent on each in psychological treatment. This is divided into "Social," "Corrective," (Cor.) or "Constructive" (Con.) as estimated in months. In the second two columns the number of hours spent in psychiatric interviews (Int'w) and the number of hours in psychoanalysis (Analy.) is indicated. The age of the patient and sex is given in the first column along with the number of the case. Clinical types are descriptive and are mostly well described in the literature. "Convulsions" (Conv.) means the motor outbursts often described as "grand mal." "Lapses" are the brief losses of consciousness usually called "petit mal." "Postural" means gross, slow movements of the trunk and limbs. "Automatic" is used to indicate the motor behavior during fugues and states of confusion. "Coordinated" means the type of motor activity described by Rosett that is seen in major seizures but is much more orderly and meaningful than the motor phenomena of the ordinary convulsion. "Lapse with motor" means a petit mal in which besides the loss of consciousness there is some brief stereotyped muscular movement.

DISCUSSION.

DR. ADOLF MEYER (Baltimore, Md.).—It is a real pleasure to see a physician put down a series of patients and try to analyze to some extent what

factors he has brought to bear in dealing with them. I think that epilepsy is particularly interesting because of the history of its management and the way it has fared and the history of the present day in its treatment.

I think it was about 1905 that Moses Allen Starr made the neurologist, and the medical profession especially, conscious of the fact that we were dealing with a brain disease. I am inclined to think that by that emphasis one came to forget to some extent that it also figured very definitely as a disease of the personality, with the brain included, to be sure.

When one speaks of psychiatric approach, one has to remember that there again we speak of something that we expect of every physician in every condition where problems arise that may pertain to involvement of the brain, or involvement of the person. What Dr. Cobb has put before us in his presentation has come to very clear expression. He describes that ever recurring problem of using the personality as the background and as the field in which one has to work.

I am particularly historically inclined today because of the place where we meet. Dr. Cobb has emphasized that activity is a very important issue in the consideration of the disease. That reminds me of the days 46 years ago when I had an opportunity and an urge to present before the Chicago Pathological Society (I included the pathology) the statement of the psychoneuroses with psychotherapy (principally as a form of occupation) as a reactivation of the personality.

Then I think of the tendency that existed in my environment at Chicago University, particularly when Watson was here, that tendency to look upon the brain as something of a telephone exchange with practically all the issues in the periphery and very little thought of the brain being capable of doing anything on its own, not of course just by itself but, nevertheless, on its own. That was the high tide of behaviorism in which the brain was left out.

Then I am reminded of a fact that Dr. Cobb did take pains to consider the terminology. After all, it is very important to have a clear idea of the terms one meets and there again we speak of psychiatric approach, I would say the psychiatric facts, the human facts, the facts that take up the whole personality. Certainly here in the problem of epilepsy it is very important to recede a little from the attitude that Moses Allen Starr promulgated to make sure that when we speak of an affection of the brain we do not forget the person. We must remember some of those cases of epilepsy in which there was a known lesion of the brain and where we then have to have the courage to meet the personality and to treat the case from the personality angle. Perhaps not only from the point of view of the 500 or 600 interviews, but sometimes from the point of view of what one is able to do in patients of that sort in one hour, in two hours, in a few interviews. I would like to say that if we would take up today the forms of disease that we have to treat and treat them in this way (as Dr. Cobb has treated his epileptics) we would have a very interesting experience. The interest in the person, the use of the person, the service to the person, is very important and we do not forget that the pharmacology of the person is in the ascendency and not to be disparaged.

FURTHER EXPERIENCES WITH THE USE OF SODIUM DIPHENYL HYDANTOINATE † IN THE TREATMENT OF CONVULSIVE DISORDERS.*

By H. HOUSTON MERRITT, M. D., AND TRACY J. PUTNAM, M. D.

Approximately one year ago we reported [1] on the results of the use of sodium diphenyl hydantoinate in the treatment of convulsive disorders in 144 ambulatory patients who had been under treatment for periods varying from 2 to 11 months. The period of observation has been extended in these and an additional 123 cases have been added. We now wish to report the results on these 267 cases.

Clinical Results.

In accordance with our original report, only those patients who had previously received other forms of therapy (mainly phenobarbital) were accepted for treatment.

The duration of treatment with sodium diphenyl hydantoinate in these 267 cases was as follows:

	Patients.
Two to six months	73
Six to twelve months	71
Twelve to eighteen months	102
Eighteen to twenty-two months	21
Total	267

The ages of these patients varied between 2 and 68 years, divided into decades as follows:

	Per cent.
Two to ten years	12
Ten to twenty years	32
Twenty to thirty years	24
Thirty to fifty years	28
Fifty to seventy years	4
Total	100

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, May 8-12, 1939.

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This study was aided by a grant from Parke, Davis & Co., of Detroit, Mich.

† Sodium diphenyl hydantoinate has been accepted by the Council on Pharmacy and Chemistry of the American Medical Association for inclusion in New and Non-Official Remedies under the name Dilantin Sodium.

The time which had elapsed since seizures began varied from less than one year to more than 40 years. One hundred and sixty-four of these patients were subject to only one type of attack at the time this treatment was started: grand mal, 124; petit mal, 21; psychic equivalent or psycho-motor attacks, 19. The remaining 103 patients suffered with a combination of the three.

TABLE 1.

EFFECT OF SODIUM DIPHENYL HYDANTOINATE ON PSYCHIC EQUIVALENT SEIZURES IN 39 PATIENTS WITH FREQUENT ATTACKS WHO HAVE BEEN TREATED FOR A PERIOD GREATER THAN TWO MONTHS.

	Psychic equivalents alone.	Psychic equivalents associated with grand mal.	Total.	Per cent.
Completely relieved	12	12	24	62
Greatly decreased	5	4	9	23
Moderately decreased	2	4	6	15
Total	19	20	39	100

TABLE 2.

EFFECT OF SODIUM DIPHENYL HYDANTOINATE ON GRAND MAL CONVULSIVE SEIZURES IN 227 PATIENTS WITH FREQUENT ATTACKS WHO HAVE BEEN TREATED FOR A PERIOD GREATER THAN TWO MONTHS.

	Grand mal alone.	Grand mal associated with		Total.	Per cent.
		Petit mal.	Psychic equivalents.		
Completely relieved	74	51	12	137	60
Greatly decreased	16	12	3	31	14
Moderately decreased	16	10	3	29	13
No change	18	10	2	30	13
Total	124	83	20	227	100

The results in this series agree in general with those previously reported. Sodium diphenyl hydantoinate is most effective in controlling "psychic equivalent" attacks, next most effective in preventing "grand mal" attacks, and least effective in the "petit mal" attacks, as shown in Tables 1, 2, and 3.

We have not as yet seen evidences of any tendency of the therapeutic effectiveness becoming less with prolonged administration of the drug. It is true that some of the patients who were classified

in our previous report as "completely relieved" of their attacks have been placed, on account of the subsequent occurrence of rare attacks, in the group with those in whom the attacks were classified as "greatly decreased" in frequency. On the other hand, equally as many have been transferred from the "greatly decreased" to the "completely controlled" group. It is too early as yet, however, to draw any conclusions in regard to the possible development of tolerance to the therapeutic action of the drug, since none of our patients have been under treatment longer than two years.

TABLE 3.

EFFECT OF SODIUM DIPHENYL HYDANTOINATE ON PETIT MAL CONVULSIVE SEIZURES IN 104 PATIENTS WITH FREQUENT ATTACKS WHO HAVE BEEN TREATED FOR A PERIOD GREATER THAN TWO MONTHS.

	Petit mal. alone.	Petit mal associated with grand mal.	Total.	Per cent.
Completely relieved	6	34	40	39
Greatly decreased	4	17	21	20
Moderately decreased	1	20	21	20
No change	10	12	22	21
Total	21	83	104	100

DOSAGE.

The therapeutic dose of sodium diphenyl hydantoinate varied from 0.2 to 0.6 gram daily for adults and 0.2 to 0.5 gram daily for children. The usual procedure was to start adults with 0.3 gram daily, given in dosage of 0.1 gram with each meal, continuing the previous medication also. After four or five days, the dosage of the previous medication was gradually reduced so that its administration was stopped after an additional four or five days. This was found necessary since the sudden withdrawal of the previous medication occasionally resulted in the precipitation of several or many convulsive seizures before a reservoir of sodium diphenyl hydantoinate was established. The original dose of 0.3 gram of sodium diphenyl hydantoinate was increased after a sufficient interval had elapsed to determine that this dose was ineffective. In a large number of cases, the frequency or severity of the attacks was only slightly changed or not affected at all by a dose of 0.3 gram daily, but the addition of an extra 0.1 or 0.2 gram produced complete relief of attacks.

Toxic Effects.

As a rule sodium diphenyl hydantoinate was well tolerated by most patients, and indeed many stated that they felt better than at any time previously. There have been no fatalities from its administration and no very serious reactions. The most troublesome symptoms in regard to the regulation of the dosage are those referable to the gastro-intestinal and central nervous system. Less frequent but more serious symptoms are those referable to the skin and gums.

The gastro-intestinal symptoms, nausea and vomiting, are probably due to the alkalinity of the drug. These symptoms can be prevented in most cases by giving the drug with the meals or by the administration of 15 minims of dilute hydrochloric acid before the meal.

Sodium diphenyl hydantoinate has very little or no sedative action but overdosage may produce drowsiness, ataxia, nystagmus and diplopia. These symptoms will disappear with reduction of the dosage, and in many instances they will not reappear if the dose is again gradually increased.

A toxic dermatitis occurs in approximately 5 per cent of our patients, usually in children. The rash is usually of an erythematous, morbilliform, scarlatiniform nature. In one case a mild exfoliative dermatitis developed and we have seen one patient not under our care that had a severe dermatitis with the formation of blebs with subsequent desquamation. As a rule the dermatitis is accompanied by fever and there may be an eosinophilia in the blood. The drug should be discontinued on the appearance of a rash and if previously effective further use, while not absolutely contraindicated, should be with caution. The reappearance of the rash indicates that the drug cannot be tolerated.

A mild or moderately severe hyperplasia of the gum was seen in 6 per cent of our patients, also usually in children. This swelling of the gum, firm and non-tender, was not noted by the patient and was found only on examination. The swelling is different from that seen in scurvy in that the gums are not spongy, there is no tendency to bleeding, and other symptoms of scurvy are missing. Kimball has reported [2] a reduction of ascorbutic acid in the serum of such patients, but values reported by him from a group of

children in an epileptic school are similar to those reported from state hospital patients in general,[3, 4] presumably due to diet low in vitamins.

Symptoms of toxic effects of the hemapoietic system or the kidneys have not as yet been noted by us. One patient, previously reported [1] had ecchymotic lesions on the buttocks, symptomatic of a non-thrombocytopenic purpura. The lesion cleared although the administration of the drug was continued.

Summary.

In 227 of 267 patients previously refractory to other forms of therapy, sodium diphenyl hydantoinate has been found to be an effective, non-sedative and relatively non-toxic means of controlling or reducing convulsive seizures over periods varying from two months to two years. A more prolonged study on a larger series of cases is necessary to establish its place in the treatment of patients with convulsive seizures.

BIBLIOGRAPHY.

1. Merritt, H. Houston, and Putnam, Tracy J.: Sodium diphenyl hydantoinate in the treatment of convulsive disorders. J. A. M. A., 111: 1068-1073, 1938 (Sept.).
2. Kimball, O. P.: Treatment of epilepsy. J. A. M. A., 112: 1244-1245, 1939 (Apr.).
3. Alexander, L., Pijoan, M., Schube, P. G., and Moore, M.: Cevitamic acid content of blood plasma in alcoholic psychoses. Arch. of Neurol. and Psychiat., 40: 58-65, 1938 (July).
4. Elgin papers. Published by Elgin State Hospital, Elgin, Illinois. 3: 225, 1939 (Jan.).

A COMPARATIVE STUDY OF THE EFFECTIVENESS OF DILANTIN SODIUM AND PHENOBARBITAL IN A GROUP OF EPILEPTICS.*

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Merritt and Putnam [1] in their search for anti-convulsant drugs, have, in a series of experiments, demonstrated the effectiveness of sodium phenyl hydantoinate in raising the convulsive threshold in normal cats. Further experimental evidence of the anti-convulsant properties in human subjects has likewise been demonstrated by the authors [2] in an article which is to be published in the Archives of Neurology and Psychiatry. Following the experimental demonstration of the anti-convulsant nature of dilantin sodium, the obvious application of the drug to treatment of epilepsy was undertaken by Merritt and Putnam.[3] According to their figures, they were able to completely control the seizures in 58 per cent of their cases who had failed to respond to any other type of therapy, viz.: phenobarbital, bromides, ketogenic diet, etc. Blair, Baily and McGregor [4] have compared clinically, the anti-convulsant properties of sodium diphenyl hydantoinate with those of luminal, prominal, bromides and chlorides. Kimball [5] reports complete amelioration of symptoms in 62.7 per cent of his 129 cases and definite improvement in 14.7 per cent.

The authors undertook to study clinically, the anti-convulsant properties of dilantin as compared to phenobarbital in a group of epileptic patients. Due to the relatively small number of patients suffering from convulsive disorders in the institution, the study was necessarily limited to a small group.

Method of Study.

Fifteen cases of idiopathic epilepsy were selected at random. Their age varied from 21 years to 50 years and the duration of their illness varied from 6 years to 38 years. Five or 33.3 per cent

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of the patients began having convulsive seizures at the beginning of their menstrual period. Three or 20.0 per cent dated the onset of their illness following pregnancy. Two or 12.3 per cent gave a history of trauma previous to the onset. Encephalograms on both of these patients failed to reveal any demonstrable pathology. Five or 33.3 per cent of the patients had no definite history relative to the onset of the epileptic attacks.

All these patients were under constant observation for one month, during that time all seizures as to type and duration were recorded. After one month, all patients were placed on 1½ grains of phenobarbital at bed time, and the frequency and types of seizures were further recorded. Having been on this type of medication for one month, they were then placed on ideal phenobarbital; *i. e.*, the convulsive reactions of the patients were studied as to time of occurrence and additional doses of phenobarbital were so timed as to be effective at the time of frequent occurrence of seizures.

The maximum dosage of phenobarbital given to one patient over a 24-hour period was 3 grains, the minimum being 2 grains. The above procedure was followed by gradually substituting dilantin sodium for phenobarbital. This transitional period was attended by a general increase in seizures which usually subsided in a few days. Thus, F. B. whose seizures were reduced by ideal phenobarbital to 11 a month, jumped to 10 convulsive responses in 10 days during the transitional period and then the seizures subsided completely. Thirteen of the 15 patients showed the above phenomenon.

Results.

As may be seen from Table I, 13 of the 15 cases showed a decrease in the number of seizures on doses of 1½ grains of phenobarbital daily. One patient showed no change and one actually had an increase in the number of seizures. On ideal phenobarbital there was a further general drop in the number of seizures or its equivalent. The best results, however, were obtained on dilantin-sodium. On this medication, 6 of the 15 patients or 40 per cent showed a complete cessation of epileptic seizures for periods of three to six months. Two or 13.3 per cent showed a diminution in the number of grand mal seizures. Six or 40 per cent showed no improvement

TABLE I.

Case No.	History				Attacks (Type)—Treatment—Results													
	Age (yrs.).	Sex.	Attacks started, age.	Etiology if known.	No. treatment. Ave. no. per month.			Phenobarbital. Ave. no. per month.			Daily dose	Ideal Phenobarbital. Ave. no. per month.			Daily dose.	Dilantin. Ave. no. per month.			Daily dose.
					Grand mal.	Petit mal.	Psych. equiv.	Grand mal.	Petit mal.	Psych. equiv.		Grand mal.	Petit mal.	Psych. equiv.		Grand mal.	Petit mal.	Psych. equiv.	
F. B. 1	33	F	13	Menses began	17	0	0	9	0	0	gr 1 ½	11	0	0	gr 3	0	0	0	Cap 5
G. D. 2	21	F	12	Menses began	7	3	0	14	0	0	gr 1 ½	10	0	0	gr 2¼	2	7	2	Cap 6
B. J. 3	39	F	21	?	5	2	0	0	5	0	gr 1 ½	Not Done				1	2	0	Cap 3
L. M 4	50	F	13	Menses at 14	8	2	0	4	0	0	gr 1 ½	3	0	0	gr 2	3	0	0	Cap 3
M. S. 5	29	F	15	Menses at 16	23	1	0	9	5	1	gr 1 ½	3	8	0	gr 2½	0	0	0	Cap 6
J. K. 6	32	F	17	Injury at 15	8	5	0	4	0	0	gr 1 ½	2	1	0	gr 2¼	1	2	1	Cap 3
R. E. 7	45	F	30	After birth second child	8	96	0	1	8	0	gr 1 ½	1	7	0	gr 2	3	2	0	Cap 5
M. M. 8	23	F	14	Auto injury at 12	16	0	0	8	0	0	gr 1 ½	7	0	0	gr 2¼	1.5	0	0	Cap 4
S. I. 9	31	F	25	?	6	2	3	1	1	4	gr 1 ½	2	0	3	gr 2¼	2	0	2.5	Cap 4
E. K. 10	35	F	23	After birth first child	6	0	0	6	0	0	gr 1 ½	4	1	0	gr 2½	5	0	0	Cap 4
M. B. 11	33	F	19	?	6	30	0	0	11	0	gr 1 ½	1	12	0	gr 2	1	6	1	Cap 3
T. B. 12	29	F	12	Menses began	1	1	0	0	0	0	gr 1 ½	Not Done				0	0	0	Cap 3
R. R. 13	32	F	14	?	3	0	0	1	1	0	gr 1 ½	1	0	0	gr 2	0.5	0	0	Cap 3
M. W. 14	38	F	28	?	12	0	0	3	1	0	gr 1 ½	2	2	0	gr 2½	2	0	0	Cap 5
B. B. 15	35	F	24	After birth third child	28	16	0	0	7	0	gr 1 ½	1	5	0	gr 2½	1.5	1.5	0	Cap 4

Remarks:

and one patient or 6.6 per cent was somewhat worse. On the whole, all patients showed improvement in affect and an increased interest in activity, though the increased interest in their well being and the special occupational and recreational classes organized for them may have had something to do with the changes in these irritable epileptic personalities.

An evaluation of the number of convulsive seizures in all patients was made by dividing the total number of grand mal seizures during a period of one month by the number of patients studied. Petit mal seizures were not studied because it was felt that a reliable index to the number of petit mal seizures was not possible. Using this method of evaluation of irritable reactivity for the patients studied, enabled us to tabulate (see Table II) results in terms of the number of seizures per patient, per month. On no medication,

TABLE II.

Type of medication.	Number of patients.	Average number grand mal seizures per month.	Average seizures per patient per month.
No medication	15	154	10.26
Phenobarbital grain 1½ per day	15	60	4.0
Ideal phenobarbital	15	45	3.0
Dilantin sodium	15	23.5	1.56

the average number of seizures per patient, per month, was 10.26 per cent. On phenobarbital, grain 1½, the number was four seizures per patient, per month. On ideal phenobarbital, the number fell to three and on dilantin sodium, to 1.56 per cent seizures per patient, per month.

Complications.

Of the complications that have arisen, the most frequent ones were a hyperplasia of the gums and psychogenic phenomena. Complications were not confined to the first 10 days of transition as reported by Merritt and Putnam.[3] Rather, as Blair, Bailey and McGregor[4] have reported some of the complications, especially those of psychogenic origin have arisen from three to six weeks following therapy. Hyperplasia of the gums occurred in five or 33.3 per cent of the patients. Kimball[5] observed that 57 per cent of the patients who used the drug two months or more, showed

some definite hyperplasia of the gums; which he believed was correlated with vitamin C deficiency. In consequence vitamin C was used in conjunction with dilantin sodium therapy by Merritt and Putnam and also by Robinson as reported by them at the last American Psychiatric Association meeting at Chicago.

Psychogenic phenomena were noted in two patients who went through a frank psychotic episode with acute hallucinatory experiences, delusional trends, disorientation, etc., a schizophrenic type of reaction which failed to clear even upon withdrawal of the drug. However, all our patients were psychotic upon admission to the institution but the two patients mentioned, seemed to be clear of any psychotic symptomatology before treatment was begun. Five other epileptics developed somatic complaints of no organic origin which increased in number as the seizures decreased.

Of the neurological manifestations attesting the toxicity of the drug against which Schlesinger [6] felt constrained to sound a word of warning, tremors, myoclonic twitchings and ataxia occurred in two patients. The ataxia was severe enough to cause patients to fall while the tremors were of such nature that they actually interfered with eating and work. None of the patients developed skin reactions. Most of these overt symptoms except those of psychotic nature, either disappeared after a number of days or were easily controlled by decreasing the dosage or removing the drug entirely.

Conclusion.

1. A comparative study of the effectiveness of phenobarbital and dilantin-sodium on a group of chronic epileptics whose average duration of illness has been 15.6 years was undertaken. While phenobarbital has proven beneficial in most cases, the best results were obtained by using dilantin-sodium, as on the latter drug 40 per cent showed a complete cessation of seizures for a period of three to six months and 13.3 per cent showed a marked diminution in the number of convulsive responses, or a total of 53.3 per cent of improvement.

2. Complications ranging from slight myoclonic tremors to frank psychotic episodes occurred. However, most of them cleared upon withdrawal of the drug or diminution of the dosage.

3. In the treatment of epileptics, the therapy of choice varies with the individual. In our series of cases, dilantin-sodium has been the medication of choice despite its complications and has proven its worth as a valuable adjunct in the treatment of epileptics.

BIBLIOGRAPHY.

1. Merritt, H. H., and Putnam, T. J.: A new series of anti-convulsant drugs tested by experiments on animals. Arch. Neurol. and Psych., 39: 1003 (May), 1938.
2. Goldstein, H. H., and Weinberg, J.: Experimental evidence of anti-convulsant properties of dilantin. To be published in the Archives of Neurology and Psychiatry.
3. Merritt, H. H., and Putnam, T. J.: Sodium diphenyl hydantoinate in the treatment of convulsive disorders. J. A. M. A., 111: 1068 (September 17), 1938.
4. Blair, Donald, Bailey, K. C., and McGregor, J. S.: Treatment of epilepsy with epanutin. Lancet, 2: 363, August 12, 1939.
5. Kimball, O. P.: The treatment of epilepsy with sodium diphenyl hydantoinate. J. A. M. A., 112: 1244 (April 1), 1939.
6. Schlesinger, B.: A new remedy for epilepsy? (Corres.). Lancet, 2: 1085 (Nov. 5), 1938.

A FOLLOW-UP STUDY OF NON-LUETIC PSYCHOTIC PATIENTS WITH ABNORMALITIES IN THE SPINAL FLUID.*

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Introduction.

This investigation was prompted by the not infrequent occurrence in mental hospitals of patients who have abnormalities in their spinal fluid not explainable on the basis of lues. In a few of these patients, brain tumor, brain abscess, multiple sclerosis or other organic disease of the central nervous system can be diagnosed. In others, the spinal fluid abnormalities are attributed to an associated alcoholism, drug intoxication, trauma or arteriosclerosis. A number of cases remain in which no explanation can be found for the spinal fluid findings. Such cases are usually discharged with the diagnosis of "Undiagnosed Psychosis," or "Psychosis with other brain or nervous disease, type undetermined." In some, diagnoses such as "dementia præcox," "manic-depressive psychosis," "involutional melancholia," and "paranoid state" are made in spite of the spinal fluid abnormalities.

It is well known that in a number of neurological diseases, such as brain tumor, multiple sclerosis, subdural hematoma, combined system disease and cord tumor, abnormalities in the spinal fluid may sometimes appear long before there are any outstanding signs or symptoms on which a clinical diagnosis can be based. In such cases only time and repeated examinations or autopsy can finally clarify the situation. In several of the above diseases, mental symptoms are of frequent occurrence. It is therefore to be expected that occasionally mental symptoms may be the first manifestation of a neurological disease in patients in whom there may be no other clinical evidence of an organic process except for abnormalities in the spinal fluid. A follow-up study of such cases

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seemed to us to be logically indicated, with a view to determining the progress of any organic disease that may have been present.

Procedure.

A study of the files in the spinal fluid laboratory at the Boston Psychopathic Hospital revealed that between the years 1924 and 1936 there were 368 patients admitted in whom the spinal fluid examination showed abnormalities not explainable on the basis of lues. During this same period there were approximately 12,000 psychotic patients admitted to the hospital. For the purposes of this study the following criteria were selected arbitrarily as the upper limits of normality: nine leucocytes per cubic millimeter, 59 milligrams of protein per 100 cubic centimeters, a gold sol curve containing 3's. These criteria were deliberately chosen higher than is usually accepted in clinical practice.

Of these 368 patients it has been possible to obtain follow-up data on 165. Most of these data were obtained through the kind cooperation of the staffs of other state hospitals in the Massachusetts State Hospital system. It is interesting to note that a form letter sent to 204 patients who were discharged home from the hospital elicited only six responses. Forty patients were examined by one of us in the follow-up study. The spinal fluid was re-examined for check-up in 26 patients. The average period of follow-up was 34 months.

Results.

It has been possible to place the 165 patients on whom follow-up data were obtained into four groups, as follows:

Group I.—This group consisted of patients in whom good reason was found to conclude that the so-called abnormal finding in the spinal fluid was due to an error in technique. Laboratory errors may occur in the spinal fluid examination as elsewhere. In six, or 4 per cent, of the cases in whom follow-up data were obtained, an early repetition of the lumbar puncture demonstrated that the abnormal finding was probably due to a technical error, or to contaminating blood.

Group II.—This group was made up of patients in whom an abnormal neurologic or metabolic state was suspected at the time

of the first lumbar puncture, and subsequent follow-up was able to establish this state as of probable etiological importance in the abnormal spinal fluid findings. Seventy of the patients, or 42 per cent of the total number followed, were placed in this group. The conditions suspected and number of patients to each were: cerebral vascular disease, 21; severe alcoholism, 21; other drug intoxication (bromide, luminal, veronal), seven; encephalitis, four; brain trauma, three; brain tumor, two; multiple sclerosis, two; pellagra, one; Huntington's chorea, one; undiagnosed disease of the central nervous system, seven. It is known that the above conditions may or may not be associated with abnormalities in the spinal fluid. The follow-up study was of value in this group because in some instances the diagnosis was at first not clear, while in others it was possible that the diagnosis made was not the true cause for the abnormalities in the spinal fluid. Through the subsequent clinical course of the disease, through repeated spinal fluid examinations, and in five cases, through autopsy findings, we were able to make the diagnoses given above more certain as the cause of the abnormalities in the spinal fluid.

Group III.—This group included those patients in whom organic disease was discovered in the follow-up study, though at the time of the first spinal fluid examination such disease passed unrecognized. These cases are, of course, of greatest interest. Nineteen patients, or 12 per cent of the total number followed, were placed in this group. The diagnoses and number of patients to each were: cerebral vascular disease, six; brain tumor, three; alcoholism, three; myxedema, two; encephalitis, one; multiple sclerosis, one; intracranial tuberculoma, one; Pott's disease of the spine, one; undiagnosed disease of the central nervous system, one. These diagnoses were made in the same manner as were those in Group II. Four cases came to autopsy.

Group IV.—This group contained those patients in whom the spinal fluid abnormalities remained unexplained. Seventy patients, or 42 per cent of the total number followed, were placed in this group. One case in which an autopsy revealed no disease of the central nervous system is included in this group.

Discussion.

The fact that errors in technique can produce spurious abnormal results in spinal fluid examinations makes it necessary that repeated examination of the spinal fluid be done whenever abnormal findings are not readily explainable on the basis of the patient's clinical picture. Had such repeated examinations been done in all our cases it is likely that Group IV would have been much smaller and Group I much larger. Probably one of the most important sources of error is the contamination of the spinal fluid by blood—the so-called "bloody tap." A method for obtaining reliable spinal fluid data in spite of contaminating blood has been given by one of us elsewhere.[1]

If we combine Groups II and III, we find that the organic condition that was most prominent in producing abnormalities in the spinal fluid in the total group was cerebral vascular disease. This was diagnosed in the follow-up study in 27 patients, or 30 per cent of the patients in whom the spinal fluid abnormalities were eventually explained. In 21, or 78 per cent of these 27 patients, the diagnosis was suspected at the time the first spinal fluid examination was made. It may be that some of these cases go unrecognized for a time because the lesions are small, occur in "silent areas," and yet may be near enough to the cerebral ventricles to produce changes in the cerebrospinal fluid. Whether the psychosis in these cases may also be explainable on similar grounds is open to conjecture. The diagnosis that was first made on these patients, in all but four cases, was "psychosis with cerebral arteriosclerosis," or "psychosis with other brain or central nervous system disease, type undetermined," or "undiagnosed psychosis." In the remaining four cases the diagnosis was "dementia præcox," "manic-depressive, manic," "manic-depressive, depressed" and "involutional melancholia," respectively.

The second most common condition which was held to be responsible for the spinal fluid abnormalities in Groups II and III was alcoholism. Twenty-four, or 27 per cent of the patients in these groups were chronic alcoholics suffering from some type of alcoholic psychosis. That the various forms of chronic alcoholism may be associated with abnormalities in the spinal fluid has been recognized.[2] The diagnosis that was first made on these patients in all but six cases was either "delirium tremens," "psy-

chosis with chronic alcoholism," "alcoholic hallucinosis," or "Korsakow's psychosis." In the remaining six cases the diagnosis was "undiagnosed psychosis" in three, "paranoid condition," "dementia præcox," and "psychosis with other brain or central nervous system disease, type undetermined" respectively. In three of these cases alcoholism was recognized but was not considered of importance in the diagnosis. In the other three alcoholism was not recognized, but was detected in the follow-up study.

The association of spinal fluid abnormalities in seven of our cases with drug intoxication brings up an important practical point. Since the psychosis in some instances may also be related to drugs, it seems worth while in all psychotic patients where spinal fluid abnormalities are not readily explainable, to conduct a special investigation into the possibility of drug ingestion.

Myxedema, which was diagnosed in two of our cases during the follow-up study, should always be considered when a high spinal fluid protein value occurs unexpectedly. Whether the psychosis in our two cases was etiologically connected with the myxedema is questionable. In one case, diagnosed "involutional melancholia," the myxedematous condition was discovered later in another state hospital and thyroid medication was begun. The patient improved from a psychiatric standpoint, but succumbed of cardiac decompensation. An autopsy corroborated the diagnosis. The other case was detected during this follow-up study, and is still under investigation. The diagnosis first made in this case was "dementia præcox, paranoid."

The remaining conditions which were diagnosed in Groups II and III are known to be frequently associated with spinal fluid abnormalities. These were encephalitis, brain tumor, brain trauma, multiple sclerosis, pellagra, Huntington's chorea, intracranial tuberculoma, and Pott's disease of the spine. In most of these cases the diagnosis on discharge from the Boston Psychopathic Hospital was such as to take into account an organic process in the central nervous system. The following, however, were exceptions: one patient with post-encephalitic Parkinsonism was diagnosed "dementia præcox"; one patient with a brain tumor was diagnosed "manic-depressive, depressed"; one patient with encephalitis was diagnosed "psychosis with mental deficiency"; the patient with the intracranial tuberculoma (which later caused a fatal tubercu-

lous meningitis) was diagnosed "psychosis with psychopathic personality." One may wish to conclude from these cases that in view of the definitely abnormal spinal fluid the above "functional" diagnoses should not have been made. However, in Group IV such diagnoses were made in 64 patients, and in these the follow-up study revealed no evidence of organic disease.

The fact that the abnormal spinal fluid in seventy cases still remained unexplained after the follow-up study is puzzling. The later diagnoses made on these cases give us little help. Thirty-five were diagnosed schizophrenia, seventeen manic-depressive, seven paranoid, seven psychosis with mental deficiency, two involutional melancholia, one psychosis with psychopathic personality, one psychoneurosis. As mentioned previously it is probable that some of the abnormal spinal fluid data in these cases were due to laboratory errors. Some may have been due to transient disease of the central nervous system which left little sign, such as brain injury, subarachnoid hemorrhage, or small "silent" vascular thrombosis. Some may have been due to unrecognized drug intoxication or myxedema. It is even conceivable that a few may yet show signs of neurological disease if the follow-up study is carried out for a longer period. However, the true answer to most of these problems must forever remain in doubt.

Perhaps the most significant result of this study is the fact that a follow-up study of these patients was able to explain the abnormal spinal fluid findings in over half the cases. Obviously, the moral to be derived is that abnormal spinal fluid findings in non-luetic psychotic patients are not to be taken lightly, even if there is no apparent explanation at hand at the moment. A further search is indicated, including a careful investigation as to the possibility of drug intoxication or alcoholism; the spinal fluid should be re-examined, if the protein is high a basal metabolism should be done, x-rays of the skull should be made, and the possibility of brain tumor should always be kept in mind. Patients should be under observation until either the spinal fluid findings are explained or it becomes reasonably certain that no neurological disease is present. An abnormal gold sol curve should be treated with especial respect. In the ten cases which we followed where an abnormal gold sol was the only abnormal spinal fluid finding, eight turned out to have organic central nervous system disease.

Summary.

A follow-up study was made at the Boston Psychopathic Hospital of 165 non-luetic psychotic patients in whom the spinal fluid contained abnormalities. The patients were followed for an average period of three years. (1) In six, or 4 per cent, the abnormal findings were traced to errors in technique (laboratory error, or "bloody tap"). (2) In 70, or 42 per cent, confirmation was obtained for neurologic or metabolic conditions which were suspected at the time of the lumbar puncture as being responsible for the spinal fluid abnormalities. These conditions were: cerebral vascular disease, alcoholism, drug intoxication, encephalitis, brain trauma, brain tumor, multiple sclerosis, pellagra, and Huntington's chorea. (3) In 19, or 12 per cent, organic diseases were discovered in the follow-up study though at the time of the first spinal fluid examination, these diseases were not recognized. They were: cerebral vascular disease, brain tumor, alcoholism, myxedema, encephalitis, multiple sclerosis, intracranial tuberculoma, and Pott's disease of the spine. (4) In 70, or 42 per cent, the abnormal spinal fluid findings still remained unexplained. Thirty-five of these patients were schizophrenic, 17 manic-depressive.

Conclusion.

Abnormal spinal fluid data in non-luetic psychotic patients should be regarded as highly significant even if no ready explanation is at hand. Every effort should be made through further examinations and prolonged observation to determine the cause for the spinal fluid abnormalities. Neurologic or metabolic disease may thus be discovered which will account not only for the spinal fluid abnormalities, but for the psychosis as well.

BIBLIOGRAPHY.

1. Solomon, Philip: The diagnosis in spinal fluid contaminated by blood; the "bloody tap." N. E. Jour. of Med., 212: 55-57, Jan. 10, 1935.
Solomon, Philip, Dailey, M. E., and Fremont-Smith, Frank: Contamination of the cerebrospinal fluid by blood. Arch. Neurol. and Psychiat., 31: 1222-1234, June, 1934.
2. Merritt, H. H., and Fremont-Smith, Frank: The cerebrospinal fluid, 214 pp. W. B. Saunders Co., Philadelphia, 1938.

DEMENTIA PARALYTICA ACCOMPANIED BY MANIC-DEPRESSIVE AND SCHIZOPHRENIC PSYCHOSES.*

The Significance of Their Co-Existence.

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It is well known that patients with dementia paralytica often present features which bear a resemblance to symptoms typically observed in manic-depressive and schizophrenic disorders. While these features are usually attributed to previous personality traits, they are considered part of the neurosyphilitic illness; the whole clinical picture is generally regarded as an organic psychosis in which the symptomatology has been conditioned or colored by personal factors. In some instances, however, the resemblance is striking enough to suggest the possibility that a true manic-depressive or schizophrenic psychosis is present. Cases of this type have received little attention in the American literature[1] but have been discussed frequently by European writers.[2, 3] Interest has been directed chiefly to cases which schizophrenic features, and divergent interpretations of the observations have been advanced.

Schube[1] has claimed that in most cases of general paresis two conditions exist side by side, namely, cerebral syphilis plus a second mental disorder which is precipitated by the neurosyphilitic process. Even though one may object to the wholesale application of this idea to such a large group, the conception may still be valid for certain cases and therefore deserves careful consideration. If it can be shown that dementia paralytica is sometimes accompanied by an affective or schizophrenic psychosis and that their occurrence together is not accidental, implications arise which are of interest not only for the neurosyphilitic disease for also for the associated conditions.

This problem was recently brought into sharp focus by a case of dementia paralytica in which a chronic hypomanic picture de-

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veloped. Death occurred from extraneous causes, and with anatomic observations available, the significance of the hypomanic symptoms could be evaluated in a clearer manner than has heretofore been possible in such cases. Because of these unusual features the case is reported in considerable detail.

CASE 1.—J. C., a woman, aged 60, was admitted to the Foxborough State Hospital on September 26, 1928. The patient was a member of a large family in which there was no history of mental disease. She received the equivalent of a grammar school education in a convent. She was described as an active and vivacious person, religious and sociable but quick-tempered and stingy. She was married twice. Her first marriage, which took place at the age of 16, was unhappy, her husband being excessively alcoholic, abusive, and unable to support her properly. She worked in factories during most of her married life. She had one child that died at the age of eight months and one miscarriage at six months. Her first husband died eight years ago. Four years previously he had sustained a fracture which failed to heal, and he had been given several injections in the arm "to change his blood." Four years prior to admission, the patient was married to a widower with two children. There was considerable bickering with her husband, chiefly over financial matters. For several years she had been suffering from diabetes, for which she was given dietary treatment. About two years ago, she began to grow jealous of her husband. It is probable that there was some real basis for this feeling. During the past three months her jealousy increased. She expressed vague somatic complaints. She had violent outbursts of temper. More recently she spent money lavishly in beauty parlors and she became extravagant in buying clothes. She expressed the idea that she was in love with a former employer. She attempted to sell her furniture and old clothes to neighbors, and she claimed that she had earned more than a thousand dollars in such a manner. Her memory showed impairment, and she spoke of angels and saints coming to her and singing hymns to her. She believed that angels had given her power to heal others. She talked of becoming a nurse and finally grew restless and unmanageable.

Examination.—The patient was overactive, overtalkative, and circumstantial. As a rule, she was elated, but she often became tearful when hardships in her past life were discussed. She believed that she possessed healing power and claimed that she had fifty thousand dollars. She had visual and auditory hallucinations of a religious and erotic nature. She was well oriented, but her memory for recent events was distinctly impaired. She made many mistakes in simple arithmetical calculations; otherwise her grasp of general information was fairly good. Her judgment was poor and insight was lacking. The patient was well developed and somewhat obese. The skin around the ears showed chronic eczema. The blood pressure was 155 systolic and 90 diastolic. The neurological examination was negative with the exception that the left pupil reacted sluggishly to light.

Laboratory Observations.—The Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 4 cells per cubic millimeter, and there was a pronounced increase of albumin and globulin. The colloidal gold curve was 5555555410. The urine contained sugar, and the blood sugar was 160 mg. per 100 cubic centimeters.

Course.—The patient became more restless and excited. She was noisy and obscene in her speech. At times she displayed a confused or delirium-like state, reacting actively to hallucinations of a religious and sexual nature. Malarial therapy was begun on November 5, 1928, and was discontinued after nine chills had occurred. On December 18, 1928, the spinal fluid changes were similar to those noted at the first examination. Following malarial therapy the patient improved considerably. The hallucinations disappeared and she no longer exhibited any impairment of memory. She became less restless and excited, presenting a picture which conformed to that of a hypomanic state. She displayed mild overactivity, distractibility, grandiose tendencies and mild elation, with occasional periods of irritability. She claimed that she could compose music and talk many languages. She dressed gaudily. Occasionally she expressed the idea that she was related to certain well-known persons and she spoke of opening a hospital in Florida. She was alert and well oriented. On February 15, 1929, she had five generalized convulsions, after which she was restless and confused for several days. Apart from this, she continued to present a hypomanic picture which persisted, with occasional fluctuations in the severity of the symptoms, throughout the rest of her stay in the hospital. On February 26, 1929, the Wassermann reactions of the blood and spinal fluid were still positive, and the colloidal gold curve was 0112331000. In June, 1929, the Wassermann reactions of the blood and spinal fluid were negative. The spinal fluid showed 2 cells per cubic millimeter, and a slight increase of globulin. The colloidal gold curve was 0112200000. Examination of the blood and spinal fluid six months later disclosed similar results, and subsequent examinations, several of which were performed, revealed normal findings in the spinal fluid except for a total protein which ranged from 35 to 70 mg. per 100 cubic centimeters. The patient received 12 injections of bismuth during 1929 and 15 of tryparsamide during 1932. At first, her diabetic condition was controlled by dietary measures, but she stole food from other patients with such regularity that insulin therapy was finally administered. However, her general physical condition remained good until January, 1937, when she developed acute coryza. Within a few days signs of pneumonia were noted, and death occurred two days later, on January 17, 1937.

Gross Pathological Observations.—Necropsy was performed six hours after death. The right lung showed extensive lobar pneumonia, and there were areas of beginning consolidation in the lower lobe of the left lung. The heart muscle was flabby. Coronary sclerosis of moderate severity and slight aortic sclerosis were observed. The liver exhibited fatty changes and the spleen was congested. The pancreas appeared somewhat atrophic. The brain weighed 1225 gm. Faint opacity of the pia-arachnoid was noted in the frontal and precentral regions. The frontal convolutions seemed

slightly shrunken. The arteries at the base of the brain showed a few atheromatous patches.

Microscopic Observations.—Large sections from the anterior frontal region, temporal lobe (including the cornu ammonis), parietal lobe, occipital lobe, basal ganglia, mid-brain and cerebellum were studied by the standard methods. With the Nissl stain the pia-arachnoid displayed slight fibrous thickening in a few places. In the frontal lobe (Figs. 1 and 2) the nerve cells were well preserved, and the cyto-architecture was not disturbed. No distinct increase of neuroglial cells was noted. Meningeal and perivascular infiltrations were completely lacking. Other parts of the brain (Figs. 3 and 4) showed the same normal appearance. Stains for microglia cells failed to disclose any abnormalities. No increase of iron was observed in sections stained by the Turnbull blue method. There was possibly a slight paucity of nerve cells in a few parts of the frontal lobe. In sections stained by the Perdrau method the vascular network of the cerebral cortex seemed rather prominent, though the small vessels were not thickened. Some of the large arteries presented atherosclerotic changes, but focal lesions of the nervous tissue were not encountered. Silver stains revealed a few areas of slightly increased density suggestive of young or incompletely developed forms of senile plaques. All these changes were considered to be well within the limits of normal for persons falling in the age period to which this patient belonged.

Comment.—In this case the clinical picture at first showed a mixture of manic and organic symptoms, and repeated examinations of the spinal fluid clearly indicated the presence of an active neurosyphilitic process. After malarial therapy, the special features which suggested organic disease of the brain, namely, impairment of memory, confusion, and delirium-like episodes, disappeared promptly and completely, and the spinal fluid soon returned to normal. Yet the patient remained in a chronic hypomanic state which persisted until death occurred from lobar pneumonia. This hypomanic condition cannot be regarded as a mere personal coloring of a neurosyphilitic psychosis, for the anatomic observations showed that a neurosyphilic disorder was no longer present. This was consistent with the fact that serological changes indicative of general paresis and mental disturbances of the type ordinarily observed in an organic psychosis were absent during the last eight years of the illness. One may therefore conclude that the patient had been cured of her neurosyphilitic disease and that she had another disorder, a hypomanic form of manic-depressive psychosis, which occurred side by side with dementia paralytica but ran a course independent of the latter.

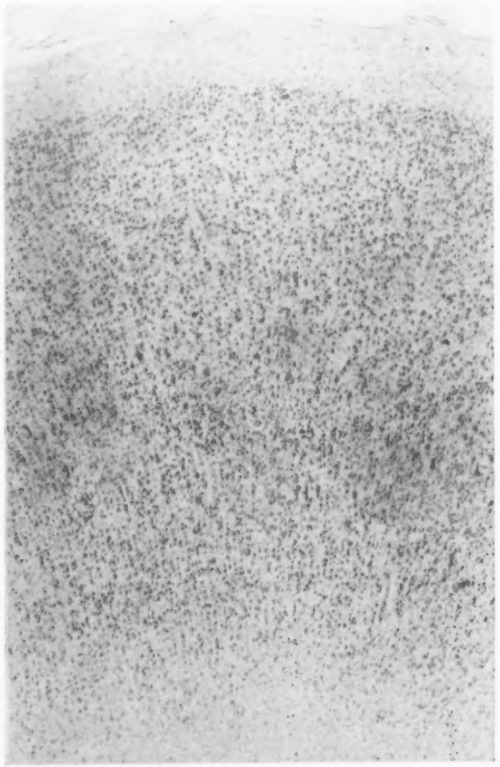


FIG. 1.—Section from the frontal lobe (case 1) showing the well preserved cerebral cortex. The cyto-architecture is normal and there are no vascular changes. Perivascular infiltrations are lacking in the pia-arachnoid and elsewhere. Thionin stain.

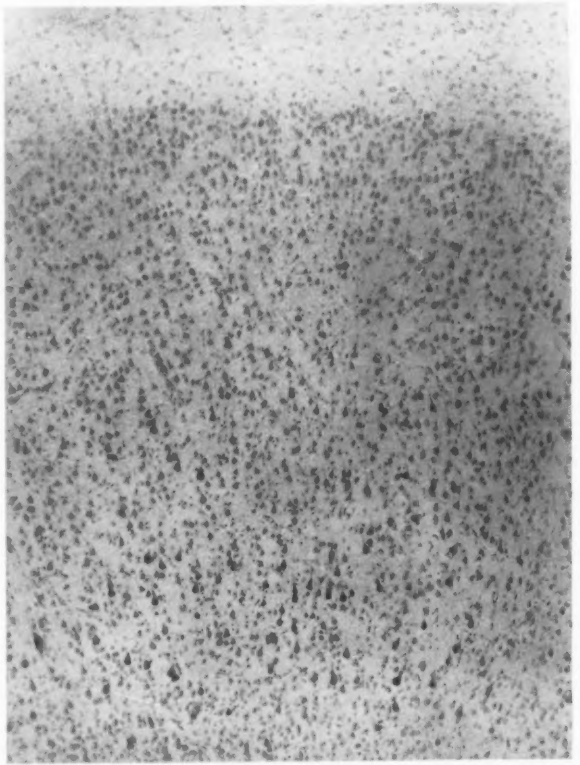
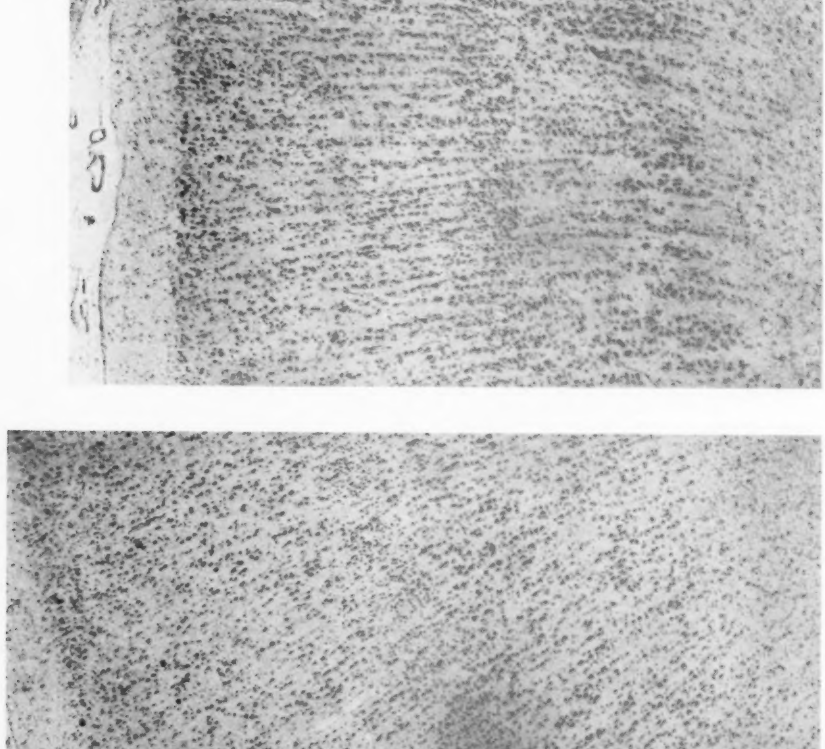


FIG. 2.—Higher power view of a section from the frontal lobe (case 1). The nerve cells are well preserved and pathologic changes of the other tissue elements are lacking. Thionin stain.



FIG. 4.



FIGS. 3 and 4.—Sections from the temporal and occipital lobes respectively (case 1), illustrating the normal appearance of the cortex and white matter. Thionin stain.

At this point, the question arises whether there was any causal relation between the two conditions. A predisposition to manic-depressive psychosis may well be postulated in view of the patient's physical make-up, which was of a distinct pyknic type, and her cyclothymic temperament. Nevertheless, the patient was able to make a successful adjustment during almost 60 years of a life beset by a variety of difficult situations, and the manic-depressive illness did not appear until an active neurosyphilis process had set in. However, one might still argue that the two conditions occurred together purely by chance. This idea can scarcely be maintained if it can be demonstrated that dementia paralytica is accompanied by other psychoses with considerable frequency. The following cases may throw light on the whole problem.

CASE 2.—F. D., a married man, aged 54, was admitted to the Foxborough State Hospital on March 7, 1932. The patient's mother died of senile dementia at the age of 82. The patient was described as a good mixer, capable and energetic, jovial, boisterous and excitable, subject to brief blue spells and intemperate in the use of alcholic drinks. His excitable traits led to considerable discord with his wife. On November 30, 1928, he was taken to the Boston Psychopathic Hospital because he had been irritable, restless and sleepless for six weeks. He had been drinking excessively, spending money extravagantly, and carrying on an affair with a morally degenerate woman. Examination showed that the patient was garrulous, noisy, blustering, facetious and euphoric. He was well oriented and his memory was not grossly impaired. His judgment was poor but he had partial insight. The Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 88 lymphocytic cells per cubic millimeter. The total protein was 48 mg. per 100 cubic centimeters and the colloidal gold curve 4432110000. A diagnosis of general paresis was made.

On December 11, 1928, the patient was given malarial therapy, which was terminated after 12 chills had occurred. Within a few weeks marked improvement was noted, and he was allowed to leave the hospital on February 7, 1929. Thereafter, he was given weekly injections of tryparsamide in the out-patient department. He was able to return to his previous occupation. Examination of the spinal fluid on June 3, 1929, disclosed a positive Wassermann reaction, 2 cells per cubic millimeter, a total protein of 38 mg. per 100 cubic centimeters and a colloidal gold curve of 4311000000. The patient remained in good condition until November, 1930, when he became overactive, irritable, and euphoric. He was therefore re-admitted to the Boston Psychopathic Hospital on November 25, 1930. At this time, the Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 4 cells per cubic millimeter. The total protein was 36 mg. per 100 cubic centimeters and the colloidal gold curve 1111000000. The patient was transferred to the Westborough State Hospital on Decem-

FIGS. 3 and 4.—Sections from the temporal and occipital lobes respectively (case 1), illustrating the normal appearance of the cortex and white matter. Thionin stain.

ber 11, 1930, and he was discharged on February 1, 1931. He resumed his previous work and adjusted well for about a year.

In February, 1932, the patient became overactive, elated, boastful and excitable, spending money extravagantly and showing poor judgment in the management of his business. He was committed to the Foxborough State Hospital on March 7, 1932. On admission he was voluble, noisy, excited and elated. He was boastful of his strength, but did not express outspoken grandiose delusions. He was irritable and antagonistic towards his wife and complained of mistreatment at other hospitals. He was well oriented and his memory was not impaired. Physically the patient showed an outspoken pyknic make-up. The blood pressure was 120 systolic and 88 diastolic. The left pupil was larger than the right, and its reaction to light was sluggish. The Wassermann reaction of the blood was positive and that of the spinal fluid was doubtful. The spinal fluid contained 3 cells per cubic millimeter, the total protein was 44 mg. per 100 cubic centimeters, and the colloidal gold curve 0012321000.

Within a few weeks the patient began to show improvement. He was allowed to leave the hospital on September 17, 1932, and he adjusted successfully during the next two years. During this period he attended the out-patient department, receiving 37 injections of tryparsamide and a similar number of bismuth injections. A mental examination performed on June 7, 1934, failed to disclose any abnormalities. Several months later, there was a gradual recurrence of mental symptoms, with elation and overactivity in the foreground, and the patient was taken to a hospital for mental diseases in December, 1934. During the following year temporary improvement occurred, and on five occasions he was able to return to his home for from one to two weeks. He was transferred to the Foxborough State Hospital on March 9, 1936, where he has remained to the present time. He has continued to exhibit a manic or hypomanic picture, with considerable fluctuations in the severity of the symptoms. There have been several periods of improvement, which were not of sufficient degree or duration to permit his discharge from the hospital. He has shown no evidence of physical or intellectual deterioration. The Hinton test of the blood has remained positive despite prolonged treatment with bismuth, but it has not been possible to obtain any additional specimens of spinal fluid for examination.

Comment.—In this case the patient developed a manic picture, and repeated examinations of the spinal fluid disclosed the presence of an active neurosyphilitic process. After malarial therapy a remission occurred, but subsequently the patient had several psychotic attacks with normal periods intervening. In all of the attacks, the symptoms were typical of the manic form of manic-depressive psychosis; in fact, the whole course of the illness was characteristic of that disorder and not of dementia paralytica. Ten years have elapsed, and the patient has failed to show any of the intel-

lectual disturbances or physical deterioration which one would expect to encounter in a prolonged paretic psychosis. These observations point to the conclusion that the early and intensive treatment was successful in healing the neurosyphilitic process and that the recurring psychotic outbreaks were expressions of a genuine manic-depressive psychosis. The first attack was associated with active neurosyphilis, but the later ones were apparently independent of any organic cerebral disease. It is possible that the patient was left with an increased vulnerability to further outbreaks. In any event, his pronounced cyclothymic temperament and outspoken pyknic make-up pointed to a strong predisposition to manic-depressive psychosis. At the same time, it should not be forgotten that he was able to adjust well until signs of a neurosyphilitic illness developed.

The psychotic pictures that finally emerged in the foregoing cases could be recognized as clear-cut affective conditions because they were not obscured by the associated neurosyphilitic disorder, which was treated successfully. However, it is sometimes possible, even when the paretic process remains active, to detect features which suggest that the organic psychosis is accompanied by another mental disorder. The following case may be given as an example.

CASE 3.—J. F., a man, aged 34, was admitted to the Foxborough State Hospital on June 3, 1929. The father died of unknown causes when the patient, an only child, was an infant. After graduating from grammar school, the patient helped support his mother, to whom he was closely attached. Later he worked as a salesman, earning as much as one hundred dollars a week. He was described as an energetic worker, a good mixer, serious, sensitive, and inclined to worry. He contracted syphilis when he was 17 years old and received an unknown amount of treatment. Five years ago he married a woman who have been divorced a year previously. They lived with the patient's mother, who quarreled with her daughter-in-law to such an extent that the latter soon returned to her own family. Four years ago, his mother died of kidney and heart disease. A few weeks prior to admission, the patient began to worry because he had been doing poorly in business for some time. He had spells of weeping. Finally, he refused to eat, stating that he had no money to pay for food and that he wanted to die. He was admitted to a hospital for mental diseases, where he was found to be depressed, agitated, hopeless, and despairing. The Wassermann reaction of the blood was positive, but he was discharged before a lumbar puncture could be performed. A diagnosis of manic-depressive psychosis, depressed type, was made. The patient was taken to another hospital and was soon transferred to the Foxborough State Hospital.

Examination.—The patient was underactive, depressed, and fearful. He was aware that he had syphilis and was greatly worried about it. His speech was somewhat dysarthric and his memory slightly impaired. The blood pressure was 115 systolic and 75 diastolic. The pupils reacted sluggishly to light. The patellar and Achilles reflexes were absent and there was impairment of postural sensation in the toes. The Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 85 cells per cubic millimeter; there was an increase of albumin and globulin and the colloidal gold curve was 1233441000.

Course.—The patient received treatment with bismuth. He remained in a depressed and underactive state. He complained of constipation and his appetite was poor. On several occasions it was necessary to give food by stomach tube. At times there was incontinence of urine and feces. In December, 1929, it was noted that the patient was still depressed. He was well oriented, but his memory was distinctly impaired. Towards the end of January, 1930, he began to show improvement and for several months presented essentially normal behavior. In March, 1930, the Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 20 cells per cubic millimeter; albumin and globulin were increased and a paretic type of colloidal gold curve was obtained. In June, 1930, the patient became restless, excited and overtalkative. He was elated but quarrelsome. This state of excitement grew steadily more pronounced. During September and October, 1930, he was extremely restless, noisy, and confused, and his speech became incoherent. His physical condition rapidly grew weaker and death occurred on October 27, 1930. Post mortem examination disclosed bronchopneumonia and acute pleurisy. Microscopic study of the brain revealed changes characteristic of general paresis in the pia-arachnoid and cerebral cortex.

Comment.—In this case the illness began with a depressive reaction which was typical enough to lead to a diagnosis of manic-depressive psychosis, depressed type, at another hospital where the spinal fluid could not be examined. The similarity to an affective psychosis became still more striking with the subsequent occurrence of an elated and excited state. At the same time, the patient showed from the start certain mental symptoms, such as impairment of memory, which were indicative of an organic disorder in the background. Ultimately this disorder came to the foreground in the form of profound restlessness and confusion, which led rapidly to a fatal termination. From an analysis of the whole picture, it seems likely that the neurosyphilitic disease was accompanied by a genuine manic-depressive psychosis, the symptoms of which were finally submerged by overwhelming manifestations of the paretic process.

Schizophrenic psychoses may also be associated with dementia paralytica. However, a clear-cut demonstration of their presence is beset with obstacles which were not encountered in the foregoing group. Owing to the nature of the symptomatology in manic-depressive psychoses, it was possible to show plainly in the first two cases that mental symptoms of the type one would expect to find in an organic condition were no longer to be observed. But schizophrenic disorders may lead to states of partial or complete inaccessibility in which it is difficult to determine whether symptoms pointing to a stationary or even an active organic process still play a rôle. Under such circumstances it may be argued that the psychosis is a neurosyphilitic one with merely a strong schizophrenic coloring. In spite of these difficulties, a careful consideration of the clinical picture and course of the illness indicates that this argument cannot be accepted in certain cases. The following cases may serve as illustrations:

CASE 4.—G. B., a man, aged 40, was admitted to the Foxborough State Hospital on April 8, 1923. The family history was negative for mental diseases with the exception that the father had a "nervous breakdown" in elderly life.* The patient had been married three times. Death of the first wife was attributed to childbirth and that of the second to tuberculosis. There were three children who had died of unknown causes. One child resulting from the third marriage was well. The patient was described as sociable but quick-tempered, stubborn and argumentative. His irritable temperament led to considerable marital discord. He worked regularly as a weaver. The present illness began gradually six months ago. He complained of exhaustion and seemed dull. At times he behaved in a peculiar manner. Two weeks before admission he stopped working. He became fearful and restless. He complained of pain in various parts of his body. He grew irritable and talked in a rambling manner. Finally, he started several fires by carelessly dropping lighted matches, and he began wandering aimlessly about the neighborhood.

Examination.—The patient was very overactive, overtalkative and noisy. He was emotionally unstable, his mood varying from elation to marked irritability and transient depression. His speech was incoherent and he occasionally expressed grandiose ideas of an absurd nature. He was disoriented, his memory was impaired and his judgment was defective. His speech showed dysarthria. The pupils did not react to light. The Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 65 cells per cubic millimeter; albumin and globulin were increased and the colloidal gold curve was 4445554000.

* It was discovered later that three paternal cousins have had schizophrenic psychoses.

Course.—Swift-Ellis treatment was given weekly until the end of August, 1923. During this period repeated examinations of the spinal fluid yielded results typical of dementia paralytica. A few weeks after treatment had been started, the patient began to improve greatly, becoming correctly oriented and less active. However, he soon began to express peculiar ideas. On July 16, 1923, he stated that poison was being put into his head in the form of medicine which was like the medicine given to African slaves to make warts grow. In September, 1923, he complained that other patients were referring to him as decaying and dying of syphilis. On several occasions he refused to eat because he thought the food was poisoned. He became restless, aggressive and incoherent in his speech, and at times he reacted to hallucinations. This was followed by a period of several months during which he gradually grew quieter. He then showed seclusive behavior, often hiding in various places. Occasionally he accused certain persons of poisoning him. In November, 1924, he complained that he was being infected with syphilis and that his mind was being taken away from him. During 1925 he was frequently restless and excited, apparently in reaction to hallucinations; at other times he was quiet, doing simple work on the ward. In June, 1926, the Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 5 cells per cubic millimeter; albumin and globulin were increased and the colloidal gold curve was 5555554100.

In October, 1926, the patient was given malarial therapy followed by tryparsamide. He seemed more friendly for a few months but thereafter became excited and aggressive in reaction to hallucinations. This condition continued during the next few years. In April, 1931, it was noted that he had numerous delusions to the effect that his body and personality had been transformed. He claimed that his teeth had been transferred to another person. Although he knew the name of the hospital, he regarded it as "a reserve bank, nothing but an insane incubator." He stated that he was 85 or 86 years old and that he had been here for 46 years. He explained that he did not look old because he had been transformed into a child. Similar ideas were noted in April, 1932. His speech was incoherent. When asked whether he was doing any work he replied, "Yes, I done every God damned thing that was necessary. Yes, I have a home in my own—when a man is taken in by the Foxborough constable—through the depths. I remember every act that was committed—done—there is no more of it." At times the patient shouted loudly in response to hallucinations. During the succeeding years there was no essential change in his condition except that his speech became more fragmentary and he grew more inaccessible. In April, 1937, he responded to a few questions as follows: (What is your age?) "1881-29 in December, give you 97 years of age." (Have you any money?) "I'm the bank man in the first place. The American gun number one." (Do you hear voices?) "Needles shot every night." (What is the name of this place?) "Everybody they call Foxborough State Hospital." (What is the date?) "1937, 1939, April 18th." (Are you happy?) "When you're in the bug-house, insane man number one." In spite of the severe mental disturbances, the patient has been able to do

routine work on the ward, and his general physical condition has been good. As to the spinal fluid, the Wassermann reaction remained positive till 1935, but since 1928 all other tests have yielded normal results with the exception that a slight increase of total protein was noted in 1931. The Wassermann reaction of the blood was doubtful in 1931 and negative in the succeeding years; the Hinton test was positive in 1935, becoming negative in 1938.

CASE 5.—B. K., a woman, aged 29, was admitted to the Foxborough State Hospital on March 22, 1932. The mother has been a patient in this hospital since 1916, the diagnosis in her case being dementia præcox, catatonic type. There was one sister who was well. The patient graduated from grammar school and received training in a business college. She worked as stenographer for two years, until her marriage at the age of 18. She had three children who were well. She was described as even-tempered and undemonstrative; she was an over-conscientious housekeeper with no outside interests. Her marital life was marked by considerable discord, and it was thought that her husband had been going with other women during the past year. At the age of 17, the patient was assaulted sexually and she contracted syphilis, for which she received 10 injections of arsphenamine and 16 of mercury. She was well until three months ago, when she had a convulsive seizure. She became forgetful and at times acted in a peculiar manner. Shortly before admission she became confused and restless, neglecting her household duties. Finally, she attacked one of her children, and was therefore taken to the Boston Psychopathic Hospital on March 8, 1932. She was disoriented, her memory was impaired, and the serologic reactions were typical of neurosyphilis. A diagnosis of general paresis was made, and the patient was transferred to this hospital.

Examination.—The patient was irritable, evasive and somewhat suspicious. She seemed mildly depressed or apathetic. She was partially disoriented and her memory was defective. Her physical make-up was of an asthenic type. The pupils were slightly irregular and they failed to react to light or in accommodation. The patellar and Achilles reflexes were absent. The blood pressure was 112 systolic and 70 diastolic. The Wassermann reactions of the blood and spinal fluid were positive. The spinal fluid contained 17 cells per cubic millimeter; the total protein was 129 mg. per 100 cubic centimeters and the colloidal gold curve 5555543210.

Course.—The patient was given weekly injections of tryparsamide, and gradual improvement occurred. In September, 1932, the spinal fluid showed a positive Wassermann reaction, 2 cells per cubic millimeter, a total protein of 130 mg. per 100 cubic centimeters and a colloidal gold curve of 5554321000. During the next few months she continued to improve. She was occupied regularly in the hospital laundry, and apart from slight impairment of memory, distinct mental abnormalities were not observed. However, in September, 1933, periods of preoccupation and periods in which she seemed to be reacting to hallucinations were noted. At this time the spinal fluid findings remained unchanged except for a return of the protein content to normal. Occasionally the patient was uneasy and tense, but she was in

good contact with her surroundings. Because of the persistence of these symptoms, malarial treatment was given in July, 1934. The patient had 11 chills. Shortly afterwards she became restless and impulsive. She heard voices coming from under her bed and at times grew irritable and refused to eat. She was correctly oriented in all fields. During the next few months her restlessness gradually decreased, and she became completely mute and inaccessible. She began to show frequent incontinence of urine. Occasionally she annoyed other patients by pulling them out of bed or attempting to remove their clothes. In August, 1935, incontinence of feces was noted. She was restless and resistive but remained in an inaccessible state. She developed a voracious appetite, and persistent coprophagia and smearing of feces were observed in February, 1936, and later. On one occasion during September, 1937, she answered a few questions. She was oriented as to time, and she gave her own name correctly and the names of her husband and children; further information could not be obtained. Apart from this, the patient has remained in a mute, restless and extremely untidy state. At the same time, she frequently acts in a manner which indicates complete comprehension of the situation. All examinations of the spinal fluid have yielded normal results since December, 1934, but the Hinton test of the blood is still positive.

Comment.—In both of these cases the mental disturbances were at first typical of dementia paralytica. Swift-Ellis treatment in one case and tryparsamide in the other led to marked improvement, but features reminiscent of schizophrenia made their appearance. Since that time both patients have presented clinical pictures which do not differ in any essential respect from those observed in certain severe forms of dementia præcox. In view of the inability to test in detail the mental functions typically affected in organic psychoses, the possibility that neurosyphilis still plays a part cannot be conclusively excluded. However, it can be said that progressive mental and physical deterioration of the type ordinarily associated with an active paretic process has not occurred, despite an illness lasting for sixteen years in one case and eight years in the other. If neurosyphilitic manifestations are present at all, they are evidently in the background. The long-standing mental disturbances in the foreground have not only taken on the appearance of a schizophrenic psychosis but have followed a course characteristic of that disorder and not of dementia paralytica. One cannot regard such disturbances simply as part of a neurosyphilitic disease when instead of being subordinate to the latter they have assumed an independent status. The only satisfactory explanation is that the

illness is really what it appears to be, namely, a genuine schizophrenic psychosis developing in association with general paresis.

General Discussion.

The foregoing observations were obtained from a hospital which does not receive a large number of neurosyphilitic patients. Furthermore, the cases selected do not exhaust the available material but merely represent outstanding illustrations of the pertinent data. Nor is there any reason to suppose that such cases are confined to one locality. Judging from the present investigation, they form a not inconsiderable proportion of the neurosyphilitic group which stays indefinitely in the hospital despite vigorous therapy. They stand out in sharp contrast to the other members of the hospital group in which one sees intellectual disturbances of a stationary or progressive nature, accompanied perhaps by euphoric traits but little else. The statement of Schube[1] that dementia paralytica is in most instances associated with another mental disorder seems too sweeping; yet there are probably cases in which a concomitant psychosis remains undetected because it is hidden or overshadowed by the organic disturbances. In view of these considerations, one may conclude that cases of the type under discussion occur too frequently to be explained on the basis of an accidental co-existence of two different conditions.

Postle[4] has recently called attention to the fact that general paresis may occur in patients who have previously had manic-depressive or schizophrenic psychoses. In her cases the neurosyphilis was associated with mental symptoms which were identical with those observed during the earlier mental illness. Postle[4] concluded that the organic brain disease serves as a release for previous personality tendencies or patterns of behavior. The present group differs from her cases in that none of the patients had shown psychotic disturbances prior to the onset of the neurosyphilitic disorder. Released patterns of behavior may still be regarded as part of the organic brain disease and not necessarily as a separate psychotic process. Therefore, it should be emphasized that in the cases reported here these patterns become independent of the neurosyphilitic condition and pursue their own lines of development, lines characteristically followed by manic-depressive and schizophrenic psychoses.

In cases of dementia paralytica in which malarial therapy is used, features suggestive of schizophrenia have been noted with special frequency. Dattner [5] pointed out that similar features occasionally occur when other therapeutic methods are employed. He claimed that all these conditions were due to toxic effects of the treatment and not to the neurosyphilitic disease. The conception of malaria as the etiologic factor has been widely accepted, though such close students of the subject as Bostroem [2] and von Angyal and Gyárfás [3] have refused to regard it as generally valid. This conception was based largely on a study of the paranoid-hallucinatory states that are commonly observed after fever therapy. Less attention has been devoted to cases with manic-depressive pictures, but here too the data have been interpreted in different ways by different workers.[2, 6]

Von Angyal and Gyárfás [3] believed that the aforementioned paranoid-hallucinatory states resembled schizophrenic psychoses but were really complex exogenous reactions produced by malaria or the neurosyphilitic disease. These authors pointed out that dementia paralytica was sometimes associated with other types of schizophrenic picture which were genuine schizophrenic psychoses precipitated by the paretic disorder. Cases 4 and 5 correspond to this group, the former showing many hebephrenic features and the latter an outspoken catatonic state. In both cases schizophrenic symptoms were already present when malarial treatment was given, becoming accentuated afterwards in case 5. In the cases with manic-depressive features, the affective symptoms were noted before treatment of any sort had been administered, though the hypomanic picture in case 1 appeared in a more clear-cut form subsequent to malarial therapy. Thus, it is evident that these disturbances cannot be attributed to any type of therapeutic procedure. Of course, it is possible that malaria may be the etiologic factor in other cases, but the present observations suggest that in many instances the treatment merely has the effect of diminishing or banishing the organic mental symptoms, as a result of which any concomitant psychosis appears in a more clearly recognizable form.* This is in agreement with the ideas of Bostroem.[2]

* Hechst [7] has suggested, on the basis of histologic observations, that relatively mild anatomic changes may be a prerequisite for the occurrence of schizophrenia-like features, and if this is so, the treatment may be indirectly

The question may be asked whether mental stress incident to syphilitic disease represents an important factor in the origin of these affective and schizophrenic conditions. As pointed out in an earlier paper on senile psychoses,[8] the existence of structural damage to the brain does not inevitably exclude the participation of psychologic influences in the disturbances. However, in the group under discussion it does not seem likely that such influences played a prominent rôle. Two of the patients (and possibly more) knew definitely that they had syphilis shortly after contracting the infection, and it is therefore necessary to explain why severe mental reactions thereto should occur only many years later. It is conceivable that difficulties which had hitherto been dealt with successfully could no longer be overcome with the handicap of organic brain disease. In this sense psychologic phenomena may perhaps play a part, but the neurosyphilitic process itself must still be regarded as the primary factor.

It is not claimed that neurosyphilitic processes contain solely within themselves the ability to produce manic-depressive or schizophrenic disorders. Such psychoses are generally looked on as types of reaction which are expressions of certain innate and perhaps acquired traits. The fact is, traits which suggested a strong predisposition to manic-depressive or schizophrenic conditions were observed in three of the cases. The fundamental significance of this aspect of the problem may be granted without minimizing the importance of the point that neurosyphilis can provoke so-called functional psychoses.

In the light of the foregoing considerations, it is necessary to scrutinize the paretic group more carefully. It is probable that in many instances an unfavorable outcome is to be attributed to a concomitant schizophrenic disorder and not to persistent or advancing organic mental changes. This is consistent with the experience of Solomon and Epstein [9] that paretic patients with a picture suggestive of a schizophrenic psychosis rarely make a good clinical adjustment, showing a disorganization of personality even when the syphilitic process is arrested. Similarly, some of the chronic elated states may well be traceable to an accompanying affective psychosis. The question whether manic-like forms of dementia

concerned in the development of such conditions by favorably influencing the neurosyphilitic process.

paralytica are in general expressions of a manic-depressive psychosis with organic admixtures also deserves serious consideration.* These problems have been largely neglected. Too much emphasis has been placed on studying dementia paralytica simply as an impersonal anatomic process and too little on studying it as a disease which may set into action powerful underlying tendencies of a more personal nature.

On the other hand, a tendency to over-emphasis in the opposite direction may perhaps be discerned in etiologic considerations of manic-depressive and schizophrenic psychoses. Of course, it is not a new observation that such conditions may owe their inception to structural damage to the brain. Cohen and Gavigan [10] described schizophrenic psychoses in cases of multiple sclerosis, and Schilder [11] believed that cerebral trauma could directly provoke a manic disorder. Somatic disturbances [12] which could well cause cerebral changes have often been mentioned as precipitating factors in so-called functional psychoses. Yet there has been a tendency to discount the importance of such factors or to disregard them completely. This has occurred in spite of the persistent belief that among schizophrenic disorders especially there is a group of cases produced on an organic basis.[13] The present observations suggest that greater attention should be given to the whole problem of possible organic factors in manic-depressive and schizophrenic psychoses. Histopathologic study may be unable to throw much direct light on this problem, but some of the newer methods of investigation give promise of yielding more fruitful results.[14]

Summary.

In five clinically and serologically typical cases of dementia paralytica, there developed features strongly suggestive of manic-depressive or schizophrenic psychoses.

Long-section studies of the cases pointed to the conclusion that these features were not merely a part of the neurosyphilitic disease but were manifestations of true manic-depressive and schizo-

* In the patients less burdened with cyclothymic tendencies one might postulate that the manic condition will tend to disappear if the structural integrity of the brain is restored by successful treatment; whereas with a more pronounced predisposition, the manic-depressive state, once started, may continue along its own independent course as in the cases recorded here.

phrenic psychoses which pursued their own lines of development independent of the organic disease. Anatomic observations available in one case supported this view.

It is believed that manic-depressive or schizophrenic psychoses accompany dementia paralytica too frequently to be regarded as accidental concomitants of the latter. Apparently they are directly provoked by the organic brain disease.

BIBLIOGRAPHY.

1. Schube, P. G.: Emotional states of general paresis. Am. J. Psychiat., 91: 625, Nov. 1934.
2. Bostroem, A.: Über die Auslösung endogener Psychosen durch beginnende paralytische Hirnprozesse. Arch. f. Psychiat., 86: 151, 1929; Über organisch provozierte endogene Psychosen. Ztschr. f. d. ges. Neurol. u. Psychiat., 131: 1, 1930.
3. von Angyal, L., and Gyárfás, K.: Die Prognose der schizophrenen Form der progressiven Paralyse. Ztschr. f. d. ges. Neurol. u. Psychiat., 153: 753, 1935.
4. Postle, B.: Pattern features and constitutional susceptibility. J. Nerv. Ment. Dis., 89: 26, Jan. 1939.
5. Dattner, B.: Moderne Therapie der Neurosyphilis, Vienna, Wilhelm Maudrich, 1933.
6. Kihn, B.: Über typische Rezidivbildingen und typische Verlaufsformen der Paralyse nach Abschluss der Infektionsbehandlung. Ztschr. f. d. ges. Neurol. u. Psychiat., 131: 124, 1930.
7. Hechst, B.: Histopathologische Untersuchungen bei der sog. schizophrenen Form der progressiven Paralyse. Arch. f. Psychiat., 102: 25, 1934.
8. Rothschild, D.: Pathologic changes in senile psychoses and their psychobiologic significance. Am. J. Psychiat., 93: 757, Jan. 1937.
9. Solomon, H. C., and Epstein, S. H.: Dementia paralytica: Results of treatment with malaria in association with other forms of therapy. Arch. Neurol. and Psychiat., 33: 1008, May 1935.
10. Cohen, L. H., and Gavigan, A. J.: Psychiatric manifestations associated with disease of the central nervous system with special reference to multiple sclerosis. J. Nerv. Ment. Dis., 85: 266, March 1937.
11. Schilder, P.: Psychic disturbances after head injuries. Am. J. Psychiat., 91: 155, July 1934.
12. Menninger, K. A.: The schizophrenic syndrome as a product of acute infectious disease. A. Research Nerv. and Ment. Dis., Proc., 5: 182, 1925.
13. Rosanoff, A. J., Handy, L. M., Plesset, I. R., and Brush, S.: The etiology of so-called schizophrenic psychoses. Am. J. Psychiat., 91: 247, Sept. 1934.

14. Jasper, H. H., Fitzpatrick, C. P., and Solomon, P.: Analogies and opposites in schizophrenia and epilepsy. Am. J. Psychiat., 95:835, Jan. 1939.

DISCUSSION.

DR. CLARENCE A. NEYMANN (Chicago, Ill.).—The paper by Dr. Rothschild was interesting because it brought out many points that have been observed in the last few years. The most interesting factor in the whole paper are his slides which show very definitely that dementia paralytica can be cured by fever. From our way of looking at this problem it makes little difference what the agency that produces fever may be, whether this is produced by a toxin injected, protein, or even organic substance, by inoculations or in an artificial manner by physical means. I have seen many patients who have recovered from dementia paralytica by means of fever.

When these patients are examined after their recovery, it is clear to the investigating psychiatrist that they even after recovery show the type of mental symptoms that they demonstrated during the psychosis or during the psychotic period. We cannot make a distinct differentiation between a psychosis and a personality change. Introversion leads by the way of the schizothemic personality into a personality showing a schizophrenic reaction type and finally a schizophrenia, and between the introvert, as we meet him in ordinary life, and the outspoken schizophrenic confined in one of the state hospitals for the psychotic, there are only grades of differentiation, and no one can say, "This individual is psychotic and belongs in this pigeon-hole and this individual is non-psychotic and belongs in another pigeon-hole."

In the same manner, extroverted personalities lead stage by stage into outspoken manic-depressive reactions. The change is too gradual to be able to say here insanity or psychosis begins and here normalcy ends. Since the formulation of the concept of dementia paralytica by Bayle in 1822, its preliminary description by Haslam in 1798, the introduction of the spinal puncture by Quincke, the proof that this is a *Treponema pallidum* disease by Noguchi and Moore about 1916, we have witnessed a clinical expansion of this concept and we know now that many patients formerly diagnosed as senile dementia, arteriosclerotic dementia, and all sorts of diseases, were basically affected with treponema. In other words, today about 35 per cent of all general paretics are classified either as ambivalent, expansive, depressed or schizoid reaction or the neurostatic reactions and 65 per cent are now clearly classified as belonging to dementia. It has been shown that with this expanding concept of the disease, the idea of dementia has expanded and it is perhaps not too radical to assume that the outstanding factors of the disease are dementia colored by the reaction type or, if you will, the psychosis of the patient.

DR. DAVID ROTHSCHILD (Foxborough, Mass.).—I do not think there is essential disagreement in Dr. Neymann's and my own attitude. I just

want to emphasize that in my cases the mental disturbances were much beyond anything that we could call a personality disturbance. I think they would have to be regarded as outspoken psychoses, and if one did not know the past history I do not think anyone would have hesitated to diagnose them as manic-depressive or schizophrenic psychoses respectively, and I have tried to show that it is possible to get such a mixture of organic disease with the so-called functional psychosis.

A CASE OF PARTIAL BILATERAL FRONTAL LOBECTOMY.

A Psychopathological Study.

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AND

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The study of the frontal lobes of the brain has engaged the increasing attention of medical men over the past 75 years. The first article bearing directly on the subject in this country was Harlow's [1] report on a man who survived the experience of having a crow-bar pass through his frontal lobes. It is not within the province of this article to trace the development of medical thought on this subject. Suffice it to say that the leading students of the brain have all left their mark upon the subject.

The earlier work was done on cases who went on to die from their frontal tumors plus a few cases who survived accidental injury. More recently the boldness and skill of the brain surgeon has supplied us with a group of cases who have survived frontal lobectomies. Penfield and Evans [2] have collected a group of these cases; Ackerley [3] reported a case which revealed a very interesting symptomatology; Brickner [4] has made an exhaustive study which is reported in detail.

As to the general symptomatology of these cases a wide variety of findings have been reported. Some of the more outstanding follow: superficiality, deterioration of moral and ethical judgment, silly superficial humor, lack of initiative, passivity, loss of normal anxiety, irritability, motor restlessness, decreased motor expression, aphasias, etc. It is seen that some of the above symptoms are at opposite poles and there is no doubt that the cases are ill assorted as to the extent and nature of their lesions and as to their previous personalities. There have of course been many attempts to express these losses as the result of some common factor. Ackerley felt that the lack of "normal anxiety" in his case was due to loss of

the ability to be "normally" distracted. Brickner believes that the symptoms can be explained by a loss of the ability to synthesize simple engrams into complex patterns which loss destroys the individual's ability to organize his life at a normal cultural and "intellectual" level. Goldstein,[5] who has worked extensively with frontal lesions and has formulated a group of tests, describes the deficit as a loss of the "abstract attitude." On first reading the two last mentioned authors one gets the feeling that they have different concepts of the fundamental psychopathology, for "abstract" and "synthesize" seem to be antonyms; but Goldstein is using abstract in the sense of "to form abstract ideas" and since these must be formed by elaboration and synthesis it appears to us that these two are writing of the same thing in different words.

The experimental laboratories have not been idle in this field. Bianchi[6] concluded that pre-frontal lobe damage in monkeys tends to "disaggregate the personality, and incapacitate for serializing and synthesizing (*cf.* Brickner) groups of representations." Franz[7] summarized the deficits in cats and monkeys in terms of deterioration of recently formed habits. Probably the most careful and adequate analysis of the function of the frontal lobes in animals is to be found in the work of C. F. Jacobsen,[8] who has worked with several of the lower primates. In a repetition of Franz's work on these animals he has failed to confirm the claim that recently acquired habits were lost after frontal injury. No amnesia appeared for simple problem-box and visual discrimination habits, and the animals learned new habits of this sort at the normal rate. In later work, Jacobsen established the fact experimentally that "destruction of the frontal areas is followed by failure to adjust to delayed-response tests."[9] In these tests the monkey's task consisted in making a selection between two cups to secure food which it had seen concealed. The problem could still be solved after delays up to 90 seconds. Unilateral pre-frontal lobectomy did not damage the level of this performance, but bilateral destruction of the frontal lobes abolished the capacity to select the correct cup after a delay of even five seconds. The same animals exhibited no post-operative deficits in problem-box situations nor in the learning of visual discrimination habits. Moreover, when damage of similar extent was done to other regions of the cortex, no deficit concerned with delayed reaction resulted and losses on other tasks did appear.

Thus, the frontal areas appear to mediate a fairly circumscribed type of psychological capacity in the lower primates, namely, the capacity for temporal integration.

Although the loss following bilateral pre-frontal lobectomy was not generalized, Jacobsen does report that the operated animals were more restless than they had been before operation. Their behavior was more stereotyped in the learning of puzzle-box problems. They repeated their errors more frequently than they had before operation. The operated animals seemed more distractible when contrasted with the sustained, directed behavior of the normal subject. And finally, the operated animals exhibited less disturbance following mistakes; they were impervious to any frustrating effects of errors. Jacobsen was cautious about extending his findings concerning the function of the frontal association areas in monkeys to man, since the homologies of the areas in man and in the lower primates are not clearly established.

The most recent development in this field is the wilful destruction of large portions of the pre-frontal lobes to cure chronic states of extreme psychomotor agitation accompanying the major psychoses. Moniz [10] had observed individuals in whom organic lesions of the frontal regions were followed by varying degrees of loss of tension and the production of a more happy, easy-going personality (this type of case is perhaps best typified by Ackerley's patient who showed the loss of a "normal" degree of anxiety). He, therefore, entertained the hypothesis that doubt, indecision, worry and anxiety were mediated through arcs in the frontal regions, and argued that if these pathways could be destroyed the individual could then exist on a much more comfortable level. He tested this hypothesis on a group of extremely agitated patients and found them improved post-operatively in that they could live more easily with themselves and others.

In this country this surgical approach to mental disease has been tested by Watts and Freeman [11] and by Lyerly.[12] The point should be stressed that in thinking of psychosurgery we must, in the authors' opinion, realize we are substituting one disease for another, and although this may be a perfectly justifiable procedure in severe cases, it can never be used in those individuals whom we wish to return to a reasonably high economic and social level. The patient

to be reported had a relatively small loss of frontal tissue yet his efficiency has been markedly reduced.

Case Report.

W. T. G. A white male, American, aged 45 (1939). Employed as executive secretary for a large organization. Admitted to Butler Hospital December 29, 1934.

The first symptom noted was a failing sense of smell which made its appearance in 1922. Anosmia was complete three years later. At or about this time he began to have attacks of petit mal epilepsy characterized by pallor, confusion and the performance of rather bizarre automatic acts. These attacks occurred at irregular intervals averaging about one every two months. In 1928 he noted that the vision of his left eye was failing slowly but progressively. About 1930 he began to show a heightened irritability and would go into rages upon slight provocation. During the next four years his symptoms slowly became more severe, and his recent memory seemed to suffer some impairment. He continued with his job until 1932 when, due to the economic depression and his irritability, his resignation was requested.

In June, 1934, he entered the Massachusetts General Hospital on the service of Dr. W. Jason Mixter.*

On June 23 a left frontal craniotomy revealed a compressed but not markedly atrophic frontal lobe. On lifting the lobe a large mesially located tumor was seen which had all the characteristics of a parasagittal olfactory groove meningioma (confirmed by pathologist's report). There was an extension into the middle fossa. The size of this portion of the tumor could not be ascertained. It was realized early in the operation that an adequate exposure could not be obtained unless frontal lobe tissue were sacrificed. Therefore, with the cutting cautery the antero-inferior portion of the left frontal lobe was removed piecemeal. Due to distortion from the tumor it was difficult to determine the line of incision according to the anatomical markings and relationships to the skull. The surgeon in reconstructing the incision on a normal anatomic chart drew an elliptical line which started at the frontal pole, at the anterior limit of the superior frontal sulcus, and then curved postero-inferiorly to strike the base just anterior to the point of origin of the Sylvian fissure. From this incision line the cut was extended mesially until the falx was reached. (See Fig. 1.) With this increased exposure the tumor tissue occupying the anterior cranial fossa to the left of the mid-line was removed. The patient made a good recovery from the operation. His symptomatology, however, did not show much change. In September of the same year he returned to the hospital and on the 24th of that month a right frontal craniotomy was performed. At this operation the same difficulty in gaining adequate exposure was present. Therefore a

* The authors wish to express their thanks to Dr. Mixter for permission to report the operative findings, and for his help in the preparation of this section.

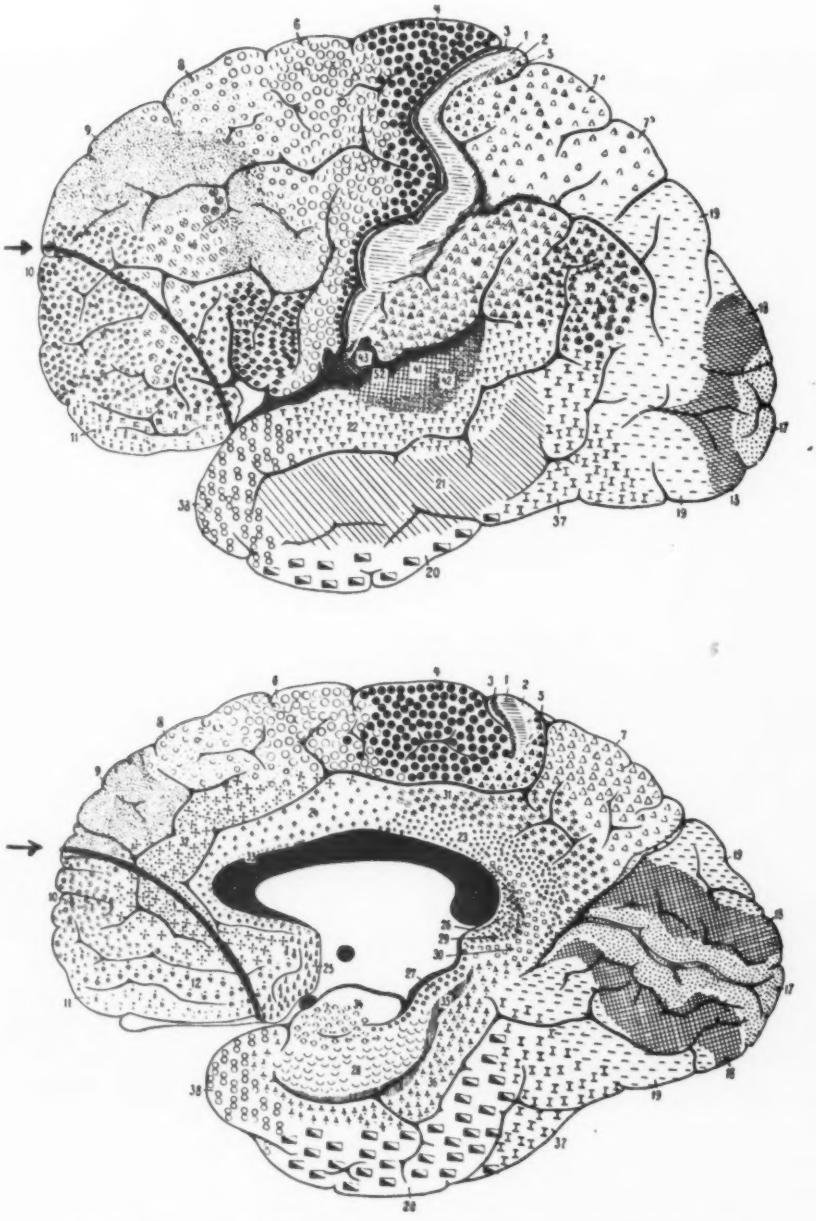


FIG. 1.—The line of incision projected onto lateral and mesial views of Brodmann's schema.



FIG. 2.—Pneumoencephalogram done March 28, 1939. Note the enlarged frontal horns of both lateral ventricles. The distortion on the left indicates regrowth of the tumor, but there is still a great deal of space available for further expansion before pressure signs develop.

similar incision was made in the right frontal pole and approximately the same amount of cerebral tissue was removed as had been taken out on the left. The tumor was then attacked and all the growth removed except for a small amount in the region of the optic chiasm and whatever may have been present in the middle cranial fossa. His post-operative recovery was again satisfactory.

He seemed somewhat improved after the second operation but he was still very irritable and continued to have petit mal epileptic attacks. When it was realized that, due to his irritability and the occurrence of spells, he could not return to his home, he was transferred directly to Butler Hospital December 29, 1934, and has remained there continuously since that time except for a few days at home in December, 1938.

Space does not permit a detailed description of his behavior at this latter institution. He fitted into the routine easily and seemed to have no difficulty in finding his way about.

The physical examination revealed the frontal scars, complete anosmia, bilateral optic atrophy most marked on the left, and a defect in the nasal field of the left eye. The other cranial nerves were negative. The tendon reflexes were bilaterally symmetrical and perhaps somewhat heightened in activity. There has been no change in these findings during his hospital stay. He is right handed.

All laboratory findings, including Wassermann test of the blood and spinal fluid, were negative.

The patient's extreme irritability which was so marked at the time of admission slowly subsided over a period of about three years. The petit mal episodes, also, followed a similar course. He now appears well and has gained in weight and physical vigor during his stay in this hospital. He shows a strong tendency to become stereotyped in his activities. Once established on a routine he follows it almost automatically. In the occupational therapy shop he prefers routine work and has steadfastly refused to start any project of his own. The patient is best handled by direct specific orders rather than by requests preceded by, "Wouldn't you like to —." He definitely dislikes being offered choices. In group card games his play is routine and colorless. In bridge he was never known to initiate a bid. When drawn into discussion he can talk easily, bringing in material from previous experience and showing a good knowledge of events of the day. He is fond of doing cross-word puzzles and can complete them with a speed and accuracy above the average for a highly educated group.

A recent encephalogram (March 28, 1939) shows a general dilatation of the ventricles which is very marked in the anterior portions of lateral ventricles. The basal cisternæ also contain more than an average amount of air. There is evidence of recurrence of the tumor along the left sphenoid wing but it has as yet occupied but little of the available space.* (See Fig. 2.)

*The authors wish to express their thanks to Dr. Isaac Gerber of Providence, R. I., for his help with the encephalographic study and for permission to use this material.

Psychological Studies.

The confusion evident in the wide variety of symptoms described as consequences of frontal-lobe injury attests the obvious need for test-situations which will define the nature and extent of the deficit in various cases. Verbal descriptions of behavior lack precision as an attempt to compare our patient with Ackerley's or Brickner's patient will demonstrate. It seemed particularly important to study this case because his level of ability and social status are unusually high and because the damaged areas are limited and fairly well defined.

The psychological analysis of this case is based upon three groups of tests. The first group (with no regard for the chronology of their administration) consists of a series of standard psychometric examinations administered by the staff psychometrician * and by one of the writers (J. McV. H.). The second group consists of tests in current use designed to test loss of cerebral function. The third group comprises tests specifically adapted to this patient. Most of the situations in the last group derived from an attempt to adapt Jacobsen's procedures to the level of human capacity.

I. PSYCHOMETRIC EXAMINATIONS.

The general intelligence of this patient following his operation was determined by the Otis self-administering examination. Three trials gave the following results: January, 1935, Form A, I. Q. 118; January, 1936, Form B, I. Q. 122; November, 1937, Form A, I. Q. 118. The patient's performance has been consistent and superior-adult ability is indicated.

It is of course difficult to determine what this patient's original level of ability was. A fine college record, mastery of four languages, and a good vocational adjustment in a very difficult field suggest even higher original ability than the I. Q. obtained. Our patient defined 89 of the list of 100 words in the Stanford-Binet scale, which is a very superior performance. Since vocabulary is considered to stand up best in face of a dementing process, Babcock [13] designed a test to measure deterioration which compares

* The authors wish to express thanks to Miss Margaret Keller, M. A., for her capable assistance in this work.

word knowledge with tests of speed, learning and motor efficiency. The Babcock indices for three administrations of the test to our patient appear in Table I.

The negative indices obtained from the Babcock examination also point toward some loss of ability. The indices are relatively stable from administration to administration, except for that concerned with learning. At first the deficit was considered as a loss of immediate memory, but later study indicated that it might be a lack of organization. In an absurdity test our patient performed excellently. Since this involved the repetition of most of the points in a narrative after a single reading, it would also argue against a deficit involving immediate memory.

TABLE I.

INDICES OF DEFICIT FOR THREE ADMINISTRATIONS OF THE BABCOCK EXAMINATION.

	January, 1936.	June, 1936.	August, 1937.
Efficiency index	−2.8	−2.4	−2.2
Learning index	−7.0	−4.2	−2.2
Motor index	−1.9	−1.3	−1.1
Repetition index	−0.0	−2.0	−2.3

A marked deficit was uncovered with the Knox cube test. In this test the examiner taps a series of four black wooden cubes in various orders which the subject must repeat. Our patient's performance (six lines) coincided with the median for seven- and eight-year-old children. The apparent discrepancy between results from the Babcock examination and the Knox-cube test and those from the absurdities test are probably explained in terms of organizing procedures, as tests reported below will show. His interest in the absurdities led him to organize them in the logical framework of the narrative, but lacking any such framework for the paired associates and the lines of taps on the Knox-cubes, he failed to organize them sufficiently to recall them. An "apparent" deficit for immediate memory is demonstrated.

II. STANDARD TESTS OF DEFICIT IN CASES OF CEREBRAL DAMAGE.

1. *Bender's Visual Motor Gestalt Test.*—Bender [14] has devised a test in which principles of perceptual organization are made

manifest through the manner in which the subject draws a series of standard figures. Certain cases of brain injury have been shown to revert to more primitive principles of perceptual organization, but our patient exhibited no evidence of such regression. He reproduced each figure precisely and in the adult manner. Moreover, he was able to reproduce six of the nine figures from memory with remarkable fidelity after the first reproductions had been completed. In this test he not only demonstrated an absence of the perceptual defect shown by this test, but also indicated that his immediate memory is adequate when he has been forced to organize the material at the time of its presentation.

2. *Goldstein's Tests of the Abstract Attitude.*—Goldstein [15] has devised a series of tests which have shown that the capacity to take an abstract attitude toward objects is commonly destroyed by damage to the frontal lobes, especially by damage to the speech areas (Brodman's areas 44, 45, and 46). Goldstein's concept of the abstract attitude is distinguished first of all from what he calls the "concrete" attitude. Objects viewed concretely are taken in their entirety for just what they appear to be. Viewed under the abstract attitude, on the other hand, certain aspects of the objects such as size, shape, color, design or relative position may be perceptually separated from the whole. Concrete objects may be synthesized from some single aspect which they have in common. The frontal-lobe deficit uncovered by Goldstein's tests consists in varying degrees of failure to use the abstract attitude and of inability to shift back and forth between the abstract and concrete attitudes. It was important to determine whether our patient exhibited this type of deficit for several reasons. His cerebral insult is smaller than that of most of Goldstein's patients, and does not involve the speech areas. Moreover, his intelligence is higher than any reported among Goldstein's cases. With these facts in view, one would expect a less pronounced deficit in the abstract attitude than has heretofore been uncovered.

We administered first Goldstein's adaptation of the Kohs-Block Test.[3] In this test the subject is ordinarily presented with a colored drawing of a yellow V on a blue background and asked to reproduce it with four of the Kohs blocks. The drawings presented at first are smaller in area than the area of the four blocks. Aphasic patients usually fail to reproduce the drawing until it is presented

in a size identical with the four blocks, or even until the lines representing the edges of the blocks are included. The smallest drawing in Goldstein's test is 1 in. square. To make it more difficult, we presented merely an outline drawing of a V on a ½-in. square; the V was labelled yellow and the background labelled blue. Our patient produced the figure indicated within one minute and with a minimum of manual manipulation. After this he was instructed to make the yellow V in white on a red background. This he did correctly in less than 30 sec. Moreover, when he was asked to make a yellow V on a red background, he replied aptly and almost immediately that this was impossible because none of the blocks was constructed with surfaces half yellow and half red. In this test there is no evidence of deficit.

Goldstein's other tests are based on sorting procedures. The subject either sorts a group of concrete objects in terms of abstracted aspect, or he is required to synthesize a category from his experience with concrete objects. As a test of the former sort, we had our patient sort the Holmgren wools. He was simply instructed to pick out all the skeins of a given color, say red. Here the color may be regarded either as some concrete quality, or may be regarded as a category of qualities varying from the light pinks to the dark maroons. With the blues, he selected a series of skeins varying from bluish white to something very near black. With yellow, he picked out six skeins and again a series was evident. His performance with other colors suggested color blindness. However, his performance with the blues was quite satisfactory evidence that a color word could represent a category containing a number of concrete objects.

To check the possibility of colorblindness, we administered the Ishihara test. In this test the subject is presented with a series of plates, asked what he sees. The plates consist of a series of colored dots so arranged that numbers or pathways are ordinarily perceived. If one has normal color vision, one sees one number or pathway; if one is colorblind, one sees another number or pathway on each of the plates. When our patient was presented with the first plate with the unspecific instruction to report what he saw, he said, "I see a big round spot of blue which is made up of small round blue dots, and some red dots." That was all, and because the examiner had already begun to formulate a theory of the nature of this

man's deficit, he turned immediately to other plates without further instructions. Our patient saw none of the figures at this first administration of the test. In a second administration of the Ishihara test some weeks later, he was instructed to look for figures, and then he reported the figures ordinarily reported by subjects who are red-green blind. This matter of "specific instructions" we consider important and will discuss later.

As a final test of the Goldstein type, we administered the adaptation of Vigotsky's test described by Hanfmann and Kasanin.[16] In this test the subject is required to sort 22 wooden blocks varying in color, shape, height and area of upper surface into four classes. The colors represented are five: red, blue, yellow, green and white; the shapes are six: circles, squares, triangles, trapezoids, hexagons and semi-circles. There are just two heights: *tall* and *flat;* and two areas of upper surface: *large* and *small.* The arbitrarily chosen correct solution is four groups consisting of *tall-small, tall-large, small-flat,* and *large-flat* blocks. The subject is confronted with these blocks, and told that there are four different kinds, and the fact that there is a logical solution is stressed. Errors are pointed out from time to time, but no positive suggestions are given. Three aspects of the task have been described by Hanfmann and Kasanin. The first is the *"abstract* or *categorical attitude"* of Goldstein, according to which the subject views the blocks not as individuals, but as bearers of certain general characteristics, or representatives of certain categories. The second aspect is termed *"insight into the multiple possibilities of the choice,"* or into the arbitrariness of the classification. The third aspect, considered the highest stage in conceptual thinking, is the consideration of the *total system* by the subject. While the qualitative aspects of the test are considered most important, a numerical score is obtained by multiplying the number of corrections by five and adding this product to the number of minutes spent at arriving at the correct solution.

On the basis of his previous performances, we anticipated that our patient would have little trouble with the first aspect of the solution, but we doubted whether he would ever reach a consideration of the total system. His performance justified these expectations. He began by putting all the circles together. When shown that a large-tall circular block was incorrect, he decided that the basis was color. Then he proceeded to alternate between the color

hypothesis and the shape hypothesis for about 10 attempts. Height was added to his bases during this time. Gradually he began to form a dual hypothesis combining color and height. Just what his color hypothesis was could not be determined. For several tries he wanted to get red, blue and white in each category. Finally after 18 corrections, he had achieved the correct classification, but it was done by selecting the matches for the tall-small, the tall-large and the large-flat blocks. The small-flat blocks were simply left in the center, and even when he had achieved this classification he did not realize it, for he asked what he was going to do with those left. Several times he forgot that there were only four classes and would start new classes when he already had four. His time was 40 minutes, and with the 18 corrections, this gave him a score of 130.

It was clear that our patient could maintain a categorical attitude. He also had insight into the multiple possibilities of the classification. Three types of deficiency stood out, however, in the course of his procedure. First, failure with a hypothesis did not lead him to discard it permanently. He alternated between shape and color for 10 corrections. Five corrections of the shape-hypothesis were made before he gave it up, and he held on to the color hypothesis until the end. Secondly, he tried unplanned placements. When stumped, he would hesitate, scanning the board until the impulse to do something appeared to have become so strong that he would try anything. Twice he admitted this, at other times he appeared to try to invent some far-fetched rationalization for his placement. Thirdly, he never appeared to appreciate the total system. At no time did he attempt to arrange a system of four classes simultaneously as most of the college students used as control-subjects did. Instead he worked with one or two at a time. The total system appeared to be too large and too complicated for him to master, and this is true in spite of the fact that he still has an I. Q. of 120 as measured by the Otis.

3. *Arithmetic Progressions and Absurdities.*—Hunt [17] has published a study which showed a sharp distinction between the performances of paretics and schizophrenics. The test material consisted of 13 groups of arithmetic progressions of increasing difficulty and a narrative containing several obvious contradictions. Paretic patients failed badly with the arithmetic progressions, but

they saw the absurdities upon the first reading without special instruction to look for them. On the other hand, schizophrenics did relatively well with the progressions but failed to find any of the contradictions until told to look for them. We administered this test to our patient. His performance was similar to that of the paretic patients. On the arithmetic progressions he succeeded with only four groups. He could continue a progression with a constant increment (2—4—6—8—) or decrement (11—9—7—5—) or one which involved simple doubling (1—1—2—2—3—3—) or one which involved the interspersing of a constant (2—1—4—1—6—), but he failed when the increment was varied (1—2—5—6—9—10—) or where two series were combined (17—1—16—2—15—3—), and with all more difficult forms. Nevertheless, as mentioned above, he read the narrative and recalled nine of the absurdities without further reference to the paper. His failure with the progressions appears to involve a capacity similar to that in the Vigotsky test. He could not analyse the system when it got beyond his present limited integrative capacity. We feel that the absurdities were recognized because they conflicted with previously learned patterns which we have demonstrated have been well retained.

III. TESTS ESPECIALLY DEVISED FOR THIS PATIENT.

1. *Adaptations of the Knox-Cube Test.*—The Knox-Cube test lends itself to an adaptation which appears to reproduce the essential features of the situation in Jacobsen's delayed reaction experiment fairly well. The subject watches while the experimenter taps four black, wooden cubes in a particular order, then he in turn must tap them in the same order. The number of taps, called a line, can be varied from 1 to any number sufficient to measure the subject's ability to reproduce them. In the standard form of the Knox-Cube test our patient reproduced 6 lines, a performance equivalent to the median for 7- and 8-year-olds. In our adaptation of this test 10 lines of each length were presented. At the first administration of this adaptation, our patient reproduced all 10 of the lines of 4 taps, but none of 5 or 6 taps. Students in elementary psychology courses ordinarily succeed with lines of 6 taps and many of them succeed with longer lines. When delays of 10 or 20 sec. were inter-

posed between the stimulus-line and the reproduction, our patient still succeeded in reproducing lines of 4 correctly almost every time, but never did he succeed with lines of 5 or 6 taps.

At the end of this test-hour the examiner questioned our patient concerning his method of recalling the lines of taps. He reported, as do almost all subjects, that he had numbered the blocks verbally, that he numbered each tap in the stimulus-line and recalled the numbers. It was interesting that he made no mention of the technique of grouping the numbers, a technique which most normal individuals hit upon when they begin to fail. Our patient had never acknowledged failure, however, for he reported subjective certainty after his reproduction of almost every line. During the discussion, the examiner mentioned the mnemonic device of grouping, and illustrated it verbally. When the test was repeated, his successful repetitions under the condition without delay increased to lines of 6 taps. The all-or-nothing aspect disappeared, for he succeeded with a progressively smaller percentage of the lines beyond 4 taps in length (5 taps—90 per cent, 6 taps—60 per cent, 7 taps—20 per cent). With the instruction to group the separate taps, the apparent defect in immediate memory is decreased, and the essential nature of the deficit begins to emerge. Our patient fails to devise spontaneously little tricks of technique when the method he happens to be using fails.

On this second test-day, delays were introduced again. With delays of 20 sec. our patient's efficiency in this performance was reduced (4 taps—90 per cent, 5 taps—20 per cent, 6 taps—0). When a question was interposed during the delay, his performance dropped back to where it had been in the beginning, namely, to lines of 4 taps (4 taps—80 per cent, 5 taps—0, 6 taps—0). In a normal control subject with ability similar to our patient's original ability the effects of similar delays produced no decrease in the efficiency of performance at all. Neither did interposing conversation damage this normal subject's performance to as great an extent (6 taps—90 per cent to 80 per cent, 7 taps—70 per cent to 50 per cent). There is just a bare hint that delay will decrease our patient's performance in this task, but further study will be necessary before we can affirm a deficit of temporal integration such as Jacobsen found in lower primates.

2. *Alternation Tests.*—In another attempt to adapt Jacobsen's procedures to our patient, we set him the task of learning a double-alternation problem. Jacobsen's procedure consisted in having the monkey learn to obtain his food from each of two food-boxes alternately. By interposing various delays between trials, he was able to measure the length of delay with which the animal could perform successfully. In the adaptation we employed, five playing cards, among which was the ace of spades, were dealt face down before the subject. Our patient was instructed to turn the ace of spades with as few turns as possible. The dealing proceeded with the ace appearing twice in successive deals at the patient's left, then twice in succession at his right. In the first test-hour with this procedure, he was presented with 200 successive deals without evidence of mastering the problem. Some little learning occurred, however, for after 60 deals he no longer looked for the ace among the three middle cards. Otherwise his performance was a chance affair. In preliminary tests, three high school students learned this problem in 12, 16, and 24 deals, respectively. Our patient's deficit on this test looms large.

On a later day the problem was simplified to simple alternation. The ace was dealt alternately on the left, then on the right. After 100 deals, our patient's performance was again still a chance affair. As there appeared to be little reason to proceed further in the same rut, a new instruction was introduced. The experimenter asked him *whether he had found the system.* He was quite surprised by this remark and claimed never to have considered the possibility of a system. Most individuals with much poorer I. Q.'s than our patient will look for new modes of attack in any problem when the one employed fails. Our patient failed to exhibit this type of search. Once it was supplied by the experimenter, however, he succeeded in mastering the system of single alternation in eight deals. The students mentioned required only four deals. When he was presented with the double-alternation problem again he also succeeded with it, although he required 36 deals. With this "set" to look for the system established, he went on to master several patterns of serial presentation. Usually the number of deals required was approximately three times as many as required by a control subject of similar ability.

It appears that our patient does not profit from his errors by searching for and inventing new modes of attack. So far this deficit has manifested itself in failure to look for a system in order to pick out the ace of spades from the five cards dealt, in failing to invent and utilize such a technique as verbal groupings in the Knox-Cube test, and the failure to perceive figures on the Ishihara plates may be a result of the same deficit. To what extent this deficit is a consequence of failure to appreciate his own failure, a consequence of failure to abstract the aspect of the situation causing difficulty, or a consequence of inability to invent a new mode of attack is not shown so far. The last alternative is unlikely, however, in view of his intelligence. Let us consider other evidence before defining the deficit further.

3. *Checkers.*—On two occasions, separated by approximately six months, the examiner played checkers with our patient. On the first occasion his performance was very poor. He exhibited no evidence of planning. He missed jumps, and when given a choice of two jumps it seemed a matter of chance whether he took the one most advantageous to him or not. During this session, which was approximately $2\frac{1}{2}$ hours long, he failed spontaneously to make any improvement in his approach to the game. Here again we see his characteristic defect—a lack of search for a more successful method of handling a problem. At the end of this session, he was quizzed concerning his method of play and incidentally given a few pointers. In the course of this discussion the examiner mentioned that one must jump at each opportunity, that jumps can be taken in such a way that they contribute to one's own advantage, and he described two of the standard methods of attack. When checkers were introduced again six months later, although he claimed that he had not played in the meantime, his game was entirely different. At the beginning he remarked, as if recalling the conversation mentioned, "One must jump when there is an opportunity." Unlike the first game when he had allowed jump after jump to pass without taking them, he took the first opportunity to jump. He considered alternatives and invariably took the better one. When he had more "men" than the examiner, he said, "Now I can trade even with you and win." While he was still no expert, he showed an appreciation of the strategy of the game.

4. *Arithmetic Progressions.*—Further evidence concerning the effects of appreciation of his own failure and of instructions as to method were obtainable with the arithmetic progressions. We determined to motivate our patient by telling him that due to his marked improvement, he should be able to do much better with the progressions than he had previously. Furthermore, we would let him know each time he failed. An attempt with the progressions under these conditions produced no improvement at all. He simply could not analyse the more complicated systems. It appeared definite, therefore, that appreciation of failure could do little by itself to improve his performance. We administered the arithmetic progressions a third time to determine how much instruction was required before he could succeed in continuing them. First the examiner described his analysis of the patient's general difficulty, telling him that he failed to see what factors were causing his failure, that he did not analyze the situations and then devise procedures which would work. This was not enough in this case, he still failed to treat the system in a progression as a whole. Next the examiner told him to write increments or decrements between each successive pair of numbers, study these and try to keep the progressions going. With this he succeeded in doing all of the type where the increment varied (1—2—5—6—9—10—). When he was presented with (17—1—16—2—15—3—) again, he employed the method given before and succeeded, but he was slow, and the task was obviously difficult. To ease the situation, the examiner told him to look at alternate numbers, and immediately he perceived the application, and employed it with all other progressions of this type. In succeeding groups of progressions, the increment or decrement was made to vary systematically. In the first one, *e. g.* (1—2—4—7—11—), the increment increased by one. In his first attempt with this progression, which he had always given up before, he tried first the procedure involving alternates which he had used just previously. This failed so he wrote in the differences between succeeding pairs, then he saw the principle and succeeded in continuing it. He continued his success until he had reached groups in which the progressions involved series within series, *e. g.,* 1—3—6—11—19—31. With these he failed again because he could not keep the various series separated. He confused the main progression with the figures he had written in,

and these with those he had written in to master the series of the differences. Supplying a method of attack which abstracts for him that aspect of the task which has caused his difficulty helped him considerably, but even this fails when the task is made sufficiently complicated. He seems to have lost the capacity to sustain several parallel lines of activity separately.

RÉSUMÉ OF PSYCHOLOGICAL STUDIES.

We have not settled the question as to whether Jacobsen's finding of failure of temporal integration can be extended to this human case of frontal-lobe injury. In the Knox-Cube test, in the alternation procedures, and in checkers a temporal factor is present. There is also evidence that interposing delays decreases the efficiency of our patient's performance in the Knox-Cube test more than similar delays affect the efficiency of a normal person of similar ability. On the other hand, however, there is failure to perceive the figures in the Ishihara plates until told to look for them, and here the temporal factor is reduced to a minimum.

We also have some evidence for increased distractibility in our patient. In the Knox-Cube test he lost more when a question was interposed between the stimulus-line and the reproduction than did the control subjects. In another situation involving the alternation procedures not previously described his performance was reduced to chance by conversation between successive deals. This finding would appear opposed to Ackerley's findings in his case, but as the evidence comes from entirely different situations, it is not comparable for the two cases. This finding would appear to be in line with Jacobsen's reports that his animals were more distractible during problem-box tests than were normal animals, but again the situational source of the evidence differs.

Several times we have mentioned an "apparent" defect in immediate memory. The term "apparent" derived from the fact that whenever the patient was supplied with the instruction to organize the material presented or with a situation in which organization arose spontaneously from the nature of the material, his memory span appeared fairly adequate.

From this study the deficit suffered by our patient appears to have four related aspects. First, he fails to supply spontaneously

fresh modes of attack on a problem in which his failure is obvious. This failure appears to result from an inability to abstract that aspect of the situation which causes the difficulty. Secondly, he exhibits a rigidity in a situation where it is necessary to shift from one hypothesis to another. To use Goldstein's phraseology, he cannot shift readily from one "categorical attitude" to another. Thirdly, even when the abstracting is done for him and he is supplied with procedures, he is sharply limited in the number of lines of endeavor that he can successfully keep separated simultaneously. In the arithmetic progressions more than two series caused marked confusion. Fourthly, the deficit also shows up in procedures where several factors must be integrated. In the Vigotsky test he was unable to appreciate the total situation. All four of these aspects of our patient's deficit might well be included in Goldstein's concept of *abstract behavior*. In him the defect is manifested at a relatively higher level of this behavior than has been found in other frontal-lobe cases studied in similar fashion. This we anticipated from his original ability and from the amount of tissue destroyed. This defect might also be included under the conception of the "power" aspect of intelligence. The fact that this patient's I. Q. is 120 demonstrates how poorly this aspect of intelligence is estimated by the standard psychometric test.

From a clinical standpoint it is important to note that this patient has profited considerably from instruction. He has carried these instructions from situation to situation and many of them seem to have been generalized considerably. He made more than the usual clinical improvement during the time that the study was being made. Fox and German,[18] in writing of a local cerebral lesion, also note that much improvement can be gained by re-education plus spontaneous reintegration. It will be interesting to see whether we can rehabilitate our patient to the point where he can make a living.

Summary.

A case is presented in which the anterior-inferior portions of both frontal lobes were removed in 1934. The present (1939) functional activity of the remaining frontal structures is not definitely known but the nutrition of the tissue seemed not to be grossly impaired at the time of operation. There is no evidence from

encephalography of local or general pressure. As nearly as can be determined from the surgeon's maps the ablation involves all of Brodmann's areas 11 and 47, the lower three-quarters of area 10, and the inferior quarters of areas 45 and 46. This patient's present I. Q. is approximately 120. Nevertheless his behavior is far from normal. He lacks initiative and tends to become stereotyped in his routine. A careful psychological study was made using a number of standard and special tests, from which we draw the following conclusions:

1. The Babcock test indicates a loss in his efficiency.

2. Bender's visual-motor gestalt test gave no evidence of deficit.

3. Two of Goldstein's tests, namely, his adaptation of the Kohs-Block test and his adaptation of the Holmgren wool test, uncovered no defect, but the Vigotsky test brought out a rigidity in his categorical attitude and a failure to appreciate the total situation.

4. On the Knox-Cube test his performance approximated the median for children of seven and eight years. On our adaptation of this test, he could reproduce only lines of four taps. Following instruction to group them he reproduced lines of six taps. Interspersing a delay reduced his performance slightly, but not sufficiently to demonstrate that Jacobsen's findings concerning the deficit in temporal integration in monkeys with frontal-lobectomies can be extended to this man.

5. With Hunt's arithmetic progressions and narrative containing absurdities, his performance was similar to that of paretics. He failed with the progressions, but noted most of the absurdities at the first reading.

6. Increased motivation did not improve his performance with the arithmetic progression, but suggestions concerning procedure led to considerable improvement. His failure with more complicated progressions consisted in an inability to keep more than two series of numbers separated.

7. On the Ishihara test of color-vision he failed to see any figures until told to look for them.

8. These performances led the writers to formulate this patient's deficit into four aspects: Failure to supply spontaneously fresh modes of attack on a problem apparently resulting from an inability to abstract that aspect of the situation causing the difficulty, a

rigidity of the "categorical attitude," a sharp limitation in the number of lines of endeavor that he can successfully keep separated simultaneously, and inability to integrate several aspects of a situation. We were inclined to regard the deficit a defect of *abstract behavior* at a relatively high level.

9. The encouraging response of our patient to instruction gives hope of considerable rehabilitation.

10. We wish to emphasize that the frontal lobes cannot be considered as silent areas, and that in psychosurgery it must be remembered that one disease is substituted for another.

BIBLIOGRAPHY.

1. Harlow, J. M.: Recovery from the passage of an iron bar through the brain. Publ. of Mass. Med. Soc., 2: 329, 1868.
2. Penfield, W., and Evans, J.: The frontal lobe in man: a clinical study of maximum removals. Brain, 58: 115, 1935.
3. Ackerly, S.: Instinctive, emotional, and mental changes following prefrontal lobe extirpation. Am. J. Psychiat., 92: 717, 1935.
4. Brickner, R. M.: The intellectual functions of the frontal lobes: a study based upon observation of a man after partial bilateral frontal lobectomy. New York, The Macmillan Co., 1936.
5. Bolles, J., and Goldstein, K.: A study of the impairment of "abstract behavior" in schizophrenic patients. Psychiat. Quart., 12: 42, 1938.
6. Bianchi, L.: The mechanism of the brain and the function of the frontal lobes. (Transl. by J. H. MacDonald). Edinburgh: E. and S. Livingston, 1922.
7. Franz, S. I.: On the functions of the cerebrum: The frontal lobes. Archiv. Psychol., 1: No. 2, 1907.
8. Jacobsen, C. F.: A study of cerebral function in learning. The frontal lobes. J. Comp. Neurol., 52: 271, 1931.
9. Jacobsen, C. F., and Nissen, H. W.: Studies in cerebral function in primates. IV. The effects of frontal lobe lesions on the delayed alternation habit in monkeys. J. Comp. Psychol., 23: 101, 1937.
10. Moniz, E.: Tentative opératoires dans le traitement de certaines psychoses. Masson & Cie, Paris, 1936.
11. Watts, J., and Freeman, W.: Psychosurgery: Effect on certain mental symptoms of surgical interruption of pathways in the frontal lobe. J. of Nerv. & Ment. Dis., 88: 589, 1938.
12. Lyerley, J.: Prefrontal lobotomy in involutional melancholia. J. of Flor. Med. Assoc., 25: 225, 1938.
13. Babcock, H.: An experiment in the measurement of mental deterioration. Arch. of Psychol., 18: No. 117, 1930.
14. Bender, L.: A visual-motor gestalt test and its clinical use. New York: Amer. Orthopsychiat. Assn., Research Monog. No. 3, 1938.

15. Goldstein, K.: The problem of the meaning of words based on the observations of aphasic patients. J. Psychol., 2: 301, 1936.
16. Hanfmann, E., and Kasanin, J.: A method for the study of concept formation. J. Psychol., 3: 521, 1937.
17. Hunt, J. McV.: Psychological loss in paretics and schizophrenics. Amer. J. Psychol., 47: 458, 1935.
18. Fox, J. C., and German, W. J.: Observations following left (dominant) temporal lobectomy. Arch. of Neur. & Psychiat., 33: 791, 1935.

DISCUSSION.

DR. ADOLF MEYER (Baltimore, Md.).—In going over the literature of frontal disorders, one is confronted with a tremendous mixture of data that at first sight, and perhaps also in the end, appear to make the topic one of considerable disappointment, if one expects very simple solutions. It seems to me as if we had still to realize that we are only in the beginning of such interpretation of human functioning as would make possible the correlation of the functioning with the brain structure, and if one follows a great many of our present-day moves in the direction of psychobiology and psychology, the complexity will have to be recognized and reduced to greater simplicity.

In the present case, there was an effort made to find essential deficiencies, and undoubtedly, with the application of some of the current testing methods, one can find data that are marking some deficiencies greater than others, and we may be satisfied, then, in speaking of deficiencies of memory, deficiencies of various categories, and also, finally, the deficiency of the categorical thinking.

Cases of this kind, had best be grouped according to their purity and simplicity and their complexity. There would be a great advantage in learning to single out first the patient's familiarity with his own reaction, and that has to be trained. Of course, the average familiarity leads to the statement that one must not expect any abnormality, superficially or as my colleague in brain surgery occasionally has indicated, one should realize that it may be best not to examine, because it would embarrass the patient without bringing help. That sort of embarrassment and injured feeling in one's perfectionistic attitude can create a great deal of confusion and lead to a somewhat bewildering picture.

In this particular case that Dr. Nichols has brought before us, the outstanding thing is an irritability on the part of the patient when confronted with a task that he absolutely refuses to tackle, and one sees then how one might develop tests that would separate the issue of management and mismanagement in handling material before one uses the standardized test of the psychobiological laboratory.

In the problem of aphasia, we have that tremendous difficulty at the present time of discouragement about being able to do some of the elementary things. In my view of aphasia, I have insisted on starting always from that chapter of the so-called projection areas, where the fundamental processes

can first be studied, and then to proceed to the more highly specialized interfunctional disturbances. One then gets into a field in which I would say the individual reactive capacity and tendency have got to be tried out, and one has to find where the individual shows the greatest deficiencies and also the greatest capacities, and where the general capacity, the emotional attitude, begins to play a very important rôle in the way of complicating the issue.

I was very much impressed by the general behavior of the patient, whom I had a chance to see for ten minutes on one occasion, just in an informal fashion. The possibility of pointing very directly to alterations is exceedingly small. The man has enough resources in general to be able to defend himself against being drawn into difficulties. Evidently some of the combinatory processes of having to make decisions, of having to project himself into the future, seemed to be exceedingly bewildering to him. The various available intelligence and other function tests have given some differentiations, but to go further, we shall no doubt have to learn to get a larger range of categories of testing, those that are really adapted to the patient's capacity to function, satisfactorily and also less satisfactorily. Out of that we shall undoubtedly get possibilities of formulating test methods that can be used in a general way, and then also lead to those generalizations which Dr. Ackerly would have to cultivate in contrast to what Dr. Halstead would like to give.

The whole question is of tremendous practical importance. We are now confronted with the fact that patients are delivered of anxieties by lobectomies, by interference in an organ which is very complex, which is not just in simple layers, but in curved layers, so that one does not know what happens in the so-called lobectomy experiments. The lesions that I have seen from this operation are very general and diffuse, if not hit or miss. The frontal lobe is not a simple organ. I think we have to see whether it is not possible to focus more definitely on those cortical areas that Brodmann has furnished us, but we also have to know something about basic topographic and functional correlations, and we are still very far away from that.

Dr. Raymond W. Waggoner (Ann Arbor, Mich.).—It is indeed an opportunity to discuss a paper of this sort. As Dr. Meyer has pointed out, this particular type of study is of great importance in increasing our knowledge of integrated human function. As we all know, the early and persistent activation of this mode of approach has been made by the psychobiological studies of Dr. Meyer. It seems to me that all processes of thinking must have both the so-called abstract and concrete components, and both seem to be disturbed in lesions of this sort. To be sure, in most cases that have been described, the abstract seems to be much more disturbed.

In this particular patient, according to the operative notes, the tumor mass extended into the temporal fossa. It involved not only the inferior surface of the frontal lobe, but the inferior surface of the temporal lobe as well. It is to be wondered how much actual destruction of a general and diffuse

character occurred involving not only the frontal lobe but the temporal lobe, since the lesion was present at least six or eight years before the operative procedure.

I was unable to find in the paper anything about abnormal movements which the patient might have presented, and I should like to ask whether such movements were present. As has been pointed out by Goldstein, there is great importance in the correlation of certain types of abnormal movements with mental states associated with frontal lesions. The inferior surface of the frontal lobe appears to be more important, perhaps, than the convex surface in the maintenance of abstract or categorical attitudes, and this was the part of the frontal lobe which was removed at the time of operation. As was seen in the diagram, most of the inferior surface of both frontal lobes was removed.

The inferior surface of the temporal lobe may also be of considerable importance in the production of symptoms such as were described in this paper, and as I have just mentioned, this was also involved, presumably by the tumor. The exact extent of the tumor, of course, we do not know.

I should like to take this occasion to agree with the authors that psychosurgery, as such, should be given a very small place in our therapeutic armamentarium; first, because we don't know what it does, and secondly, because of the obvious permanent damage to the brain.

One other tangential comment I might make. It seems to me that we are losing a great opportunity to make studies of this sort in the examination of various cases in the institutions for, let us say, the feeble-minded and convulsive disorders. I don't believe enough attention is being given to careful neurological and psychological studies of such cases. This should be done with a view to late neuro-pathological studies, and just the kind of study that Dr. Nichols has presented to us this morning is possible in a great number of such cases.

DR. FREDERICK J. KRUEGER (Lexington, Ky.).—When we introduced a method of equivalent and non-equivalent stimuli in investigations with human patients, we were rather pessimistic about finding a simple situation in man, chiefly because we found already in sub-human primates that the function that Dr. Meyer was speaking of was of such a complex nature.

The speaker referred here to the work of Jacobsen on the behavior of pre-frontal monkeys, and it seemed for a time, as if the analysis of the behavior of pre-frontal monkeys could be an entering wedge into the disturbances in man. He claims that it was chiefly a defect of the temporal organ of behavior that was found, although he really said that the experiment merely suggested that there was a defect of the temporal organ of behavior. The situation he used was visual. There were two cups before the monkey, or the ape, and it was found that the animal could not delay the response, although recently Dr. Jacobsen found certain situations which have this in common with the visual situations, that they require a temporal response, and it was found that the pre-frontal monkeys can delay, as well as the normal monkey.

In other words, you have even in sub-human primates the dilemma of finding twenty situations which look like temporal defects of behavior, but one situation which involves the same temporal features but in which the animal can delay, and which will show on further experiment that it must be a non-temporal feature of the situation. As has been pointed out, in monkeys as well as in man, we have the same situation in all cases of categories or basic functions, an interdependence of various functions that are at stake.

Dr. Kurt Goldstein (New York, N. Y.).—I would like to say a few words on one of the questions which was put in the discussion, namely the relation between the behavior of patients in test situations and outside of test situations in everyday life. When I began to make these studies about twenty years ago, with a test similar to that which Dr. Halstead has demonstrated, we studied at the same time the everyday life of the patient, and we found it is wrong to say that patients behave in everyday life in a normal way. Only if you observe the patient in a very superficial way will you come to the conclusion that he behaves in a normal way.

If you would read—I am sorry that most of the things are written in German—the very careful reports of our pupils who went through the city with patients and tried to find out what they are saying or what they are doing, their relations with family and friends and in the community, then you would see that the patient had the same acts in everyday life, only in everyday life they didn't come out so well merged, because most of the things we do in everyday life are done on a less concrete basis, and all of the time when the patient has to do something in everyday life on an abstract basis, there is the same mistake. It concerns knowledge, emotion, volition and all the various activities of the human being at the same time.

So I think it would be very important to study these patients, not only with tests, which I have used for many years and esteem very highly, but with things in everyday life, and then we will find the real differences between a normal individual and a sick patient in everyday life.

Dr. Nichols.—I wish to thank all the discussants for their comments. I feel that Dr. Meyer thinks that we didn't give quite enough attention to the emotionality in the situation. I think that as time has gone on, our patient made a better adjustment. His rages became fewer, perhaps, as he began to reintegrate certain things and to see his way through a bit.

Dr. Waggoner asked a question in regard to abnormal movements. We tested him for those abnormal movements—the position of the arms, for instance—and he showed no disorder of that sort.

In regard to the question of temporal lobe involvement in this case, that it is a difficult question to answer. The surgeon said that he felt there was additional tumor over in the middle fossa; how much, he couldn't ascertain. In the pneumoencephalograms taken recently, there was air in the basal cisternae, so things were not too crowded in that district.

Dr. Krueger in his discussion spoke of the correlation with animal experimentation in trying to compare our work with Jacobsen's temporal defect. Our evidence was equivocal and we couldn't come to any definite conclusion.

In regard to Dr. Goldstein's remarks on the life of the patient at home and in the community, of course this patient is still in the hospital, he is not in the community. He hasn't organized himself sufficiently to get back into it.

Penfield and Evans, in reporting cases of maximum removals of the prefrontal lobes, report a very interesting case which is probably world famous, in which the individual was unable to organize herself completely in the home. The situation that was most difficult for her was to organize a whole dinner and get everything coming to the stove at the right time and getting it properly related. With a little help and a little planning added by someone outside the situation, she could go through and do all these various things. Socially in the home she was very well integrated, but in these more complex situations, her adaptation broke down.

SOCIAL PSYCHIATRY—OUR TASK OR A NEW PROFESSION.*

By SAMUEL W. HARTWELL, M. D., Buffalo, N. Y.

Very often, when important events happen or when social changes, having far-reaching effects, occur, those nearest to the situation are least aware of their significance. So, perhaps, while it seems paradoxical to suggest it, the institutional psychiatrist does not realize how eagerly the general public awaits announcements of advances in the cure and prevention of mental disease.

He is blind who says that the discoveries and working hypotheses of the modern clinical and research psychiatrist and psychologist are not important and that they do not prove that there are great possibilities ahead for mental hygiene and social psychiatry. We see but vaguely, as yet, the way in which the emotional past of the individual relates itself to many forms of mental disease, but we know now that it does so relate itself. How tremendously this enlarges the field of psychiatry!

We have learned to think of the mentally-ill person as one who, like the mentally-healthy person, has many factors and forces, both external and internal that are meeting in him and making him what he is. Again, how the field of psychiatry is enlarged by this knowledge!

Today, many people are calling to us for help. The clergyman, the jurist, the criminologist, the sociologist, the teacher, the parent, the individual and the medical clinician are seeking our aid.

Some short 20 years ago, a new profession appeared on the scene. Its members were not called nurses of the mind, perhaps it would have been a good name. The psychiatric social worker of the first period was thought of as being the psychiatrist's assistant and ally. We are today confronted with decisions that will determine whether or not these psychiatric social workers are, indeed, to be our allies and assistants—our nurses of the mind—or are they to develop into an independent group who will be

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functioning in the field of social psychiatry and preventive mental medicine more or less independently of our profession and in some measure, at least, turning to others for training, leadership and working relationships. During the past 20 years the modern treatment of neurosis, mental hygiene in all its meaning, forensic psychiatry and child guidance have been accepted by our profession as part of our function. We must have assistants adequately trained. We have them in clinical psychologists and psychiatric social workers.

Is psychiatry—is this society—going to accept the responsibility of leadership in this field? If we are, we must have, first, an informed medical profession back of us; second, more psychiatrists; and third, we must have groups of truly professional assistants to help us. These groups still consider themselves as our assistants. They still are looking to us for the more technical part of their training and are still asking us for directions and supervision of their work.

Fellow members of The American Psychiatric Association, I am today warning you that, unless we, all of us, as individuals and as an organization, do more to hold and further develop these relationships, the time is not far distant when our opportunity will be gone. I do not need to present the arguments that show us so clearly that without non-medical assistants we shall not be able to enlarge the scope of our activities as psychiatrists, as we must enlarge them if we are to remain leaders in the field of mental health as well as leaders in the field of the treatment of mental disease.

I wish to discuss with you some of the facts that the Committee on Social Relationships of this society feel you should be thinking about. I wish to pass on to the membership of our Association some of the problems the American Association of Psychiatric Social Workers now have that they believe they need our help to solve. And finally, with the approval of the Council of The American Psychiatric Association I wish to discuss very briefly with you some of the recommendations our committees are making in our annual report to the Council and to report to you about the plan of action we propose to take in meeting the problem of the education of psychiatric social workers so that a certificate of psychiatric social work will definitely designate a person care-

fully and adequately trained and qualified to work with us in a truly professional manner.

The first fact is that more and more as psychiatry advances, we realize that the mental past of the mentally sick person must be known and his mental future planned for if our therapy is to be successful. This truly professional task is the first function of the psychiatric social worker.

I wish I had time to review all the factors that are operative in creating the ever-increasing demand, both public and private, that is now being made on psychiatry and the allied professions and the extent that this demand has created the possibility and the actuality of people not adequately trained, functioning in our professional field. Social agencies and schools and many individuals, while realizing often the danger of non-trained people trying to do psychiatric work but being unable to find psychiatrists who had the time to help them, turned quite naturally to the psychiatric social workers who were the only group that had, in any way, received the endorsement of, and had been trained by, psychiatry. These workers were, at first, used largely as case consultants in mental hygiene; but later, often because of great need, they were, and still are, used as psychotherapists with little or no supervision by psychiatrists.

The American Association of Psychiatric Social Workers was formed soon after graduates began to function. At first the members were nearly all working in psychiatric agencies and mental hospitals. Now only a minority of them are so employed. I have no time to discuss, and most of the members of this group are well aware, how this has happened. We, as psychiatrists, and the psychiatric social workers, as a group, may well feel that it is an unfortunate situation. When graduates of schools of psychiatric social work with little experience in psychiatric situations cease to work in psychiatric agencies, there are two dangers—the first, that the worker will forget her technique and come more and more to approach her job in a superficial way, or second, that she, because of her training and her consequent ability to see the need of psychiatric diagnosis and treatment and in the absence of the opportunity to use psychiatrists as consultants will, herself, function essentially as a psychiatrist. It goes without saying that this is a professionally unfortunate and clinically dangerous dilemma. This

dilemma is one of the important problems confronting the American Association of Psychiatric Social Workers, as an organization, and many of the individual members personally.

There is yet another important problem. For many reasons, but chiefly because the demand for social workers with training in the "modern case approach" and the resulting ease with which graduates who have had courses in abnormal psychology, clinical psychology, psychiatric case work, elementary psychiatry for social workers, child guidance and mental hygiene can be placed, many schools of social work are offering such courses in increasing numbers. A rather large number of new schools have been established in the past few years. We, as psychiatrists, certainly know that in theory such courses should be a part of the social worker's training. However, very few schools have a field-training program that usefully supplements these courses. Perhaps, in some cases, schools are using teachers not adequately trained and clinically experienced enough to be safe individuals to impart to young social workers, who are soon to be given grave responsibilities, the factual material of mental hygiene and psychiatry that they should have, and at the same time, prevent them from thinking they are competent to function as psychiatric social workers.

It is of serious import that we as a professional group should realize that even though the psychiatric social workers have, themselves, set up good curriculum standards, as yet neither the Federal Government nor any of the state governments have established any standards of training requisite for a degree or certificate in psychiatric social work.

Because of the tendency for all schools of social work to introduce courses in the same subjects that are offered by the best formal courses of psychiatric social work, and for other reasons, there are now many agencies, schools and even some psychiatrists who are honestly asking the question as to what difference there is between a psychiatric social worker and a social worker. There is a difference. The better schools have, without too much encouragement or interest on the part of a large part of our profession, developed a discipline of thinking that is definitely more scientific and a technique of working that is more professional than the schools of ordinary social work. Our task as a profession is to cooperate with the schools and with organized groups of psy-

chiatric social workers to the end that this difference will be greater and will be maintained.

Psychiatric social workers are definitely established as a part of the staff of all out-patient, psychiatric, child-guidance and mental hygiene clinics. But in many mental hospitals there are few, or even no psychiatric social workers; and, with some notable exceptions, mental hospitals have not yet manifested any very general interest in accepting social workers for training and in tuning their staff to the training and use of psychiatric social workers. Why has psychiatric social work, now more than twenty years old, failed to be more important in mental hospitals? Psychiatric social work is too important a profession, and hospital psychiatry needs, as a part of its therapeutic set-up, psychiatric social workers too badly for us not to be interested in this problem.

Let us briefly mention some of the reasons why mental hospitals are not getting the help that psychiatric social work might give them, why the psychiatric social worker is not being given basic and required training in mental hospitals, and why she is going into other fields than formal psychiatry. In the beginning, the presence of "psychiatric teams" in extramural clinics and their usual absence in mental hospitals had much to do with the non-use of the mental hospitals as training centers.

But we have all had more experience, and psychiatry, as well as mental hospitals, has changed in the past 20 years. We know that a psychiatrist who functions in private practice or in the field of mental hygiene, child guidance, industrial or educational psychiatry will be more useful and proficient if he has had the experience and discipline of mental hospital training. Everyone now agrees that the young physician who plans to enter psychiatry in any field must have mental hospital experience. If this is true for the medical school graduate who has spent seven or eight years in his training, how obviously it must be true of the often young and often immature social worker who has spent, at most, three years in preparing herself to assume the responsibility of trying to help people retain, or regain, their mental health. There are too many improved or recovered patients, who are returning to the hospitals, who could remain well or be able to live in their homes if hospitals had workers who could control, treat or cure such social situations, and there are all too many unhappy human beings, who have diffi-

culties in their environment, who, understood in terms of its psychological meaning to them, can be helped to an adjustment to life they will otherwise miss, and there are too many children who are indulging in poor mental habits which will later result in mental breakdowns or personality disintegration who can, if their problems be understood by someone who can interpret them or plan for them, correct some of these habits to their eternal good. There are too many of all these for us to believe that we do not need psychiatric social workers trained as thoroughly as it is possible for the schools and for ourselves to train them.

Many psychiatric social workers of recent and of earlier years have had no experience in their training years with psychotic patients or with the relatives of psychotic patients in terms of the mentally sick individual. We may now assume that one of the reasons why psychiatric social workers are not more generally employed is the fact that there is little uniformity of their methods of procedure in case interpretation and in social therapy. Until there is a basic training to help the worker orient herself in her field and to help her adjust to new situations and new colleagues, the young worker who is trained largely or entirely in a clinic under the direction of one, or perhaps two, psychiatrists is in great danger of becoming a disciple to the psychiatrists' methods, if not the psychiatrists, themselves. How different it would be if a part of the basic training of every psychiatric social worker was training experience in a well-staffed and well-equipped mental hospital, for hospital therapy is, at least in some degree, standardized and there are always a number of different psychiatrists who will have a part in the training program.

Many of these factors that have created our present problems are controversial, but if psychiatry and psychiatric social work are to become more intimately associated and we are not only to have the help of the psychiatric social worker, but are to maintain leadership in the field of mental hygiene, these factors must be discussed frankly and evaluated. We must admit that many mental hospitals have very practical reasons why they have not trained psychiatric social workers. The most important one is that the schools have not urged them to, or helped the hospitals to make practical plans to do so.

In some cases, perhaps most often, this has been because of the financial problems created in setting up an adequate training program for psychiatric social workers. In others, and this is a most regrettable situation, it has been because the hospital has found that the great demand for trained workers by non-psychiatric agencies has made it impossible to hold in their employ the workers they have trained to work with their staff members. A larger salary, a greater freedom, and what the worker considers is a more interesting job, are offered the worker as soon as her training is completed.

The psychiatric profession is partly to blame, perhaps we have some good excuses for our shortcomings. It costs more money to employ a psychiatric social worker than it does a social worker with little or no training. Inevitably, jealousies and difficulties develop when a hospital tries to employ one or two psychiatric social workers who must work with others not so trained.

Schools of psychiatric social work have graduated students poorly prepared and poorly adjusted in their own personality and have presented them for employment or sent them for training in situations where this is quickly recognized and have thus brought disrepute both on the school and the idea of psychiatric social work. The problem of the poorly adjusted student, the one who comes to the school obviously in need of psychiatric help herself, is a very important one. If the courses and her teachers help her to a better adjustment, she will possibly be a more useful psychiatric social worker than one who has had no problems. If they fail or partially fail to help her, she brings discredit to her profession. The schools now recognize this as one of their greatest problems.

The schools have sometimes sent the less desirable students to the hospitals. Psychiatric social workers in training have sometimes felt that the psychiatric staffs in mental hospitals were not "up-to-date"; and, in more cases, the social worker has created a false impression of proficiency because of the polysyllabic vocabulary that she has been taught to use.

Despite all these excuses the Committee on Social Relationships feels that psychiatry is not justified in remaining aloof from the problem and duty of developing a discipline of training workers to the end that they will become and remain truly professional and genuinely loyal to psychiatry as a high calling and to psychiatrists

as their teachers and leaders. There are some very practical things that we have now learned. First, we have not realized, until recently, that it was only by giving psychiatric social workers training in mental hospitals and long experience in working with psychiatric patients and with their relatives, that we, as psychiatrists, could create a relationship with the worker that would make her a truly professional person, able to work with, or under the direction of, any psychiatrist. Already the psychiatric social workers have had a very wholesome influence on the public's attitude towards mental disease, the discharged or paroled mental patient, and towards the mental hospital. If we are ever to make progress in prevention, it will be largely through the education of parents, school teachers and the general public.

Second, we have learned, through much experience, that the person who is mentally ill usually has, as one of the etiological factors in his illness, a pathological environment, and that cures in the hospitals are often negated by the patient's return to his outside world. But we have often failed to realize how much our psychiatric social worker can do to cure this sick environment and how much more she will be able to do twenty-five years from now, if we work hand in hand with her in developing her skill and technique.

Third, we have sometimes been guilty of expecting too much of the psychiatric social worker and of giving her too great responsibilities, and conversely, we have sometimes thought of her as an ordinary social worker and used her too much for duties which might be performed by non-professionally trained persons.

Fourth, we have learned that our greatest fault has been our indifference. We have said we will wait and see if psychiatric social work has usefulness for us. Almost universally the extramural psychiatrists find the psychiatric social worker useful. The time should come when she will be even more useful to the intramural psychiatrists.

What can we do about it? First, and most important, is for all hospitals so situated as to make it practical to plan to train students from the accredited schools of psychiatric social work. Prejudices within the staff both social and medical must be broken down. Time must be spent not only in training the social workers, but in helping the younger, less experienced psychiatric

social worker to improve her technique just as we now do in the younger members of the medical staff. We must try to create the feeling within the profession of psychiatric social work that to work in a mental hospital is a true function of a psychiatric social worker.

"A house divided against itself cannot stand." The psychiatric profession is coming to the realization that, if we are to live up to the high possibilities of our profession in the United States and Canada during this period of social change, we must not let a breach develop between the extramural and intramural psychiatrists. The institutional psychiatrist, the psychiatrists in public clinics, the psychiatrists in private practice, and the teachers of psychiatry in medical schools must be a unified body if we are to attain to our best possibilities. Psychiatric social work has become important, and it will either help us bring this unification within our professions, or it will make the situation less unified.

Medical schools are seeing the need for the teaching of psychiatry in its broader sense. In the next few years, to our mental hospitals are going to come many young psychiatrists who will feel that it is as important to help individuals with their early conflicts, to make unhappy individuals happy, to warn them of weaknesses in their personality, and to have techniques to strengthen the weak places, as it is to make a diagnosis of the type of mental disease that has developed or to give the schizophrenic patient metrazol or insulin. The staff members of mental hospitals should be among the leaders in community education. Community clinics should be a part of the hospital function. Staff members should be consultants in the schools and the out-patient departments of general hospitals. Mental hospitals should all have an out-patient department that is thought of as a friendly refuge in time of need. One thing that will help all mental hospitals to achieve these very desirable goals is to have skillful and well-trained psychiatric social workers.

The American Association of Psychiatric Social Workers is justified in feeling that it is an old enough group for psychiatry to be able to make the decision as to whether or not it wishes a close affiliation with them. They hope and believe that they can become increasingly useful to psychiatry. They believe that their research in the field of psychiatric social work should be integrated

with the research in psychiatry, and they believe that if we, as a profession, will join with them, we can accomplish much for our mutual benefit.

If we, as an association, are to actively accept the task of making it possible for psychiatric social work to develop as a truly professional group closely allied to psychiatry, there are some immediate things that we must now do. First, we must as an organization give formal approval or disapproval of schools of psychiatric social work. This, your Committee feels, should be a joint action with the American Association of Psychiatric Social Workers. We must together set up minimum requirements both for formal courses and field training that must be followed by schools whose certificates or diplomas of psychiatric social work The American Psychiatric Association will officially recognize. A corollary of this is that we, not only as a society, but as individuals, must directly concern ourselves with knowing whether or not those employed in our vicinities as psychiatric social workers are adequately trained and are worthy of the title they are using, and we must be active in the educating of our communities to the dangers of inadequately prepared or experienced psychiatric social workers.

Second, mental hospitals, many of them, must be established as basic training places of psychiatric social workers. Not until these opportunities are available shall we be able to do what the Committee of Social Relationships feels is the goal towards which we should work. That goal is to establish as soon as possible as one of the requirements for graduation in psychiatric social work a period of adequate training in a mental hospital as a basic experience.

And finally, we must realize that the training of psychiatric social workers by the staff of a mental hospital is a thing that in many ways is sure to improve the technique of the staff members themselves, and enhance their ability to work together as therapeutic teams. And may we not venture to believe that the training and employment of psychiatric social workers will not only increase our cures and discharges, but finally will it not help us as institutional psychiatrists to go out into the community and decrease the rate of admissions?

DISCUSSION.

MRS. KATHARINE M. WICKMAN (New York).—It gives me great pleasure to discuss Dr. Hartwell's stimulating and in many respects challenging paper. As a psychiatric social worker I naturally turn to those aspects most directly concerned with social case work and its practice in psychiatric hospitals and clinics. What can the social worker with more practical experience in working in association with psychiatrists in clinics and hospitals contribute to those with less experience or no experience who have had theoretical courses from psychiatrists in schools of social work?

By definition, a psychiatric social worker is a social case worker (in contradistinction to one trained to deal with people in groups) who has had training, experience, or both, in a psychiatric clinic or hospital. According to this usage, which is in keeping with that of the American Association of Psychiatric Social Workers, as reflected to date in its membership requirements, the term signifies the kind of setting in which the social worker has had experience, a special type of background which can be defined objectively. As Dr. Hartwell has said, she may have worked with psychiatrists in either a hospital or clinic setting or perchance both. The experience in clinics *is* as different as are the individual psychiatric clinical services.

Currently, for full membership in the American Association of Psychiatric Social Workers there is a requirement of one year's experience in a psychiatric clinic or hospital for the worker after graduation from a specialized course in psychiatric social work in an accredited school of social work (which includes a period of supervised experience in a hospital or clinic). For those who have graduated from other than psychiatric social work courses, two years' similar experience, following graduation, is required, six months of which shall have been in a position under the supervision of a psychiatric social worker. A few members are still admitted without formal training if they have had four years' experience of a kind which is specified and if this experience began prior to 1929, when professional education was not readily available. Some weeding out of workers, poorly equipped because of personality, is attempted by the schools of social work but this still remains an important factor for individual consideration.

The psychiatric social worker who has gone from the clinic or hospital field to a regular staff position in a community social agency, is, in keeping with the above definition, practicing family social work, that with dependent children, or public welfare as the case may be. She is working in an independently organized agency having a specialized social function which is distinguished from that of medical settings in which psychiatric social work or other medical social work is carried on.

There is, as Dr. Hartwell suggests, great need for more supervision by psychiatrists of those activities of the psychiatric social worker in which she participates more closely with psychiatric treatment plans for the patient. I believe that it would be unfortunate, however, to think of psychiatric social work as requiring anything less than the best basic general social work training which is available because the more thorough the professional

education of the social worker the more useful she will be in extending the psychiatrist's function in the community. Whatever part she may take in the intensive psychiatric treatment of the patient, this should go hand in hand with her more generalized functions. Stress on the broader aspects of her work may serve as the best possible antidote for any naive attempts to function as a pseudo-psychiatrist. The psychiatric social worker tends to draw from as well as to contribute to work in community social agencies and I believe she can be no more isolated in her social work thinking than she can remain physically isolated within the hospital walls.

From such knowledge as I have of schools of social work, there seems every reason to believe that evidences of interest on the part of The American Psychiatric Association, in the content of professional education, should command immediate attention and should have an important influence upon future trends. The working relationship already provided for by this Association and established between Dr. Hartwell's Committee and the Committee on Relationships with Psychiatry of the American Association of Psychiatric Social Workers which was appointed in March, 1939, with Maida H. Solomon as chairman, may progressively do much to stimulate and enrich the thinking regarding minimum educational requirements for psychiatric social work. It should strengthen a beginning relationship of the American Association of Psychiatric Social Workers with the accrediting body for schools of social work, namely, the American Association of Schools of Social Work, as well as with the individual schools.

However much these schools may currently be stressing broad general training as against that for any special field, the situation may be different some years hence: the individual fields of social work may develop and contribute more which is recognized as distinctive; advanced courses may be established; the length of time required to secure a diploma, or, as in some schools a master's degree, may be extended beyond what is now, more generally, a two-year period. Psychiatric social work may be among the first of the special fields to focus thinking upon the responsibility for providing for its students adequate specialized preparation in addition to what has become in recent years an improved basic training, but which, as Dr. Hartwell suggests, in some instances, certainly has not been well balanced in psychiatric content.

Schools of social work in their individual ways are tending to teach all social case work students some understanding of the emotional life of the individual. Attention to specialized theoretical content for psychiatric social work preparation now becomes a matter for reconsideration in consultation with psychiatrists. Along the way there has, in some schools, appeared to be a lack of full appreciation of the importance, for social case workers in general, of sufficient knowledge of psychopathology to supplement other courses given in psychiatry. The lack in this respect in the training of psychiatric social workers is of outstanding immediate concern.

Developments in the interrelationships of the work of psychiatrists and psychiatric social workers need to be formulated and more effectively brought to bear upon professional education for social work. It still remains

for the American Association of Psychiatric Social Workers to fulfill one of its most important functions, namely, to prepare and successively revise a minimum curriculum and standards for field work, in discussion with psychiatrists, which will be acceptable to The American Psychiatric Association and command the respect of the schools and be useful to them.

Dr. Hartwell's references to preferred centers for the training of psychiatric social workers is important and the matter should be brought up for discussion locally among psychiatrists and psychiatric social workers so that facts may be accumulated. It seems that any generalizations as to the relative advantages and disadvantages of hospitals, child guidance or other clinics must, at this time, be based upon the standards of social work which obtain in each setting and the availability therein of a psychiatrist or psychiatrists able to participate in the training program, in keeping with the requirements of the schools of social work. Pressure by students for placements in psychiatric clinics and hospitals is great and it comes from students wishing to pursue psychiatric social work and also from certain more experienced workers who plan to return to their respective social work agencies. The schools are training workers for all fields and are using psychiatric clinics and hospitals wherever they can find them, sometimes paying the salaries of the psychiatric social workers responsible for the training of students. I should like to stress the hospital as tending to produce the much to be desired eclectic and conservative point of view, through the wider opportunity it offers for contact with scientific medical procedures with all varieties of mental illness, including those of organic origin. How soon serious consideration could be given by the schools to including mental hospital experience in the training of all students in psychiatric social work would depend upon many factors. One very practical consideration which Dr. Hartwell has mentioned is the dearth of mental hospital training centers.

To touch upon another part of Dr. Hartwell's paper, there are, I believe, many good reasons why psychiatrists and psychiatric social workers have not always gotten together and it is obvious that the discrepancy of 82 years in the ages of the two specialized professional fields lies behind much of what Dr. Hartwell has said. This is the ninety-fifth annual meeting of The American Psychiatric Association; the next, in the case of the American Association of Psychiatric Social Workers, will be but its thirteenth. I am glad Dr. Hartwell has mentioned terminology. Various terms, meaningless to anyone but the social worker, and often only to the individual social worker, have come and I know that some of them have gone. Terminology in social work is in an embryonic stage. Psychiatric social workers need to contribute more to such efforts at definition as are being made. "Social treatment," for example, is certainly vague. It covers the gamut of the things which a social worker does. There are no distinctive types of treatment. So-called "treatment" may be carried on in visits, telephone calls, casual social contacts or in interviews. Standardized criteria for the extent or kind of social study have never been formulated. Gauging of results is, by comparison, nebulous. The term "therapy," unfortunate in so many ways, reflected a stage through which social workers passed when they

were first feeling the influences, as it happened, of psychoanalysis. They suddenly came to the baffling realization of the extent to which certain of their methods in meeting social problems within families went counter to the emotional needs of their clients. It took some time to adapt the new knowledge to old functions and to attain perspective. There is much room for improvement in methods of keeping records so that these shall embody only terms which can be clearly defined and will permit reading at one sitting.

Training and practice in psychiatric social work must follow one upon the other. Social workers are not trained to do something about personality problems as such, but rather about social problems. It is true, however, that these social problems in families, the "sick environments" to use Dr. Hartwell's words, rarely exist without a concomitant of what may be called "problem feelings," on the part of one or more members of the family constellation. This is true whether the social problems are financial, or those concerned with physical or mental health, with a child needing foster home placement, or others. I like to think of the more basic "problem feelings" from the point of view of social work as feelings of difficulty in intrafamilial and other social relationships, including that with the worker. Social workers have found it necessary to reckon with the feelings of the individuals with whom they deal. The more concrete social problems, when the worker has some awareness of what psychiatry has to contribute concerning the emotional life of people, often prove to be the inevitable avenues to learning the inner feelings of the individuals concerned. To effect changes constructively, in the surface problems presented, it has been shown that it often may be necessary to do something about these subjective feelings along the way. Rarely is it feasible, even if the worker should so wish it, to devote any great share of her time to interviews not pertinent to practical factors. Surely, this is true wherever the function of the social agency or department is clearly formulated.

Social workers need to become more selective in their thinking in all phases of their work, and selectivity with regard to frankly psychotic or neurotic clients, to which Dr. Hartwell refers, is of paramount importance. I was particularly glad he emphasized the urgent need which social workers are feeling for an increase in the number of psychiatric outpatient clinic facilities for adults. I am sure this is felt everywhere by social workers in social agencies as well as by psychiatric social workers in child guidance clinics who see parents clearly in need of psychiatric diagnosis and treatment as well as those in whom psychiatric symptoms have been less obvious but who might, upon being referred, be considered eligible for psychiatric treatment.

To conclude, psychiatric social work needs the expressed ideas of groups of psychiatrists. The developments within individual psychiatric clinics and hospitals have been too isolated one from the other. Far more has appeared in the literature on the applications of psychiatric concepts in practice in allied fields of social work than on psychiatric social work in clinics and

hospitals. Psychiatric social work looks to psychiatry for help in outlining a suitable minimum curriculum and standards of field work training; for cooperation in developing training centers in mental hospitals; for opportunities to discuss the rôle and function of the psychiatric social worker; for help in providing adequate placement service, so that available openings (including those of lesser responsibility which do not require experience in addition to training and offer opportunities for experience under supervision), may be made known to current and former graduates in psychiatric social work in schools of social work.

PSYCHOSES WITH MYXEDEMA.*

By RALPH M. CROWLEY, M.D.,
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Of approximately 4,500 hospital admissions in 14 years (1923-1937), we find only two patients who fall into the classification, "psychosis with myxedema." There are several reasons for reporting these.

First of all, they are rare. H. Wegener [11] makes a statement quoted by Dunbar,[17] that besides his own case there appear in the literature only two other unequivocal instances of psychosis with myxedema, one reported by Kraepelin [2] and one by Pilcz.[3] While perusal of the American literature does not bear out Wegener's statement, such cases are by no means frequent. Ziegler [9, 1] in his review of the literature finds some 45 to 50 and adds three of his own. Beck [5] reports one of his and states that 26 per cent of his 194 patients with thyroid deficiency manifested hallucinations. Wagner von Jauregg states 15 per cent of myxedematics have psychoses. Reports of further cases are as follows: Hayward and Woods,[10] two; Courtois,[13] one; Parker,[12, 15] two; Karnosh and Stout,[16] report four more; Akelaitis,[18] Ruhberg [19] and Simon,[21] three each. With the two here reported, the total approximates 70.

This syndrome is particularly interesting in the light it sheds on psychosomatic problems. It combines both psychic and somatic symptomatology, and offers opportunity for studying the psychogenetic and organogenetic aspects of etiology.

Furthermore, these cases raise the question: "What is myxedema with psychosis?" Myxedema shades into hypothyroidism, and psychoses shade into neuroses, giving rise to many borderline pictures. For practical purposes we must recognize that they fall into the provinces of both endocrinology and psychiatry.

Among these cases some writers include those patients whose myxedema followed radiological or surgical destruction of the

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thyroid. Many which Ziegler reports from the literature and all three of his own, are post-radiated or post-operative. One each is reported by Karnosh and Stout, Hayward and Woods, and by Simon. In our series these cases are purposely omitted because of the artificial element in the production of the myxedema and because of further complicating factors, such as difficulties of diagnosis and the reasons for the operations in the first place. Was there before the operation a psychosis for which the thyroid was excised? Was there a psychosis masking itself as hyperthyroidism? Or was a Basedow's syndrome in some sense an alternative solution to a psychosis in a conflictural emotional situation? Among our patients were many instances of psychoses following operations on the thyroid gland, with various complicating emotional and physical factors. There was only one case, however, of definitely determined post-operative myxedema, and this was complicated by a simultaneous neoplasm of the lung from which the patient died.

On the basis of reports on two patients, some of the questions raised above will be discussed.

First Patient.—Mrs. A., a divorced woman of 38, entered the hospital in April, 1929. Following an attack of grippe four months previously, she complained of swelling of her face, neck, and hands, of palpitation, weakness, feeling cold, of dryness of her skin, choking sensations in her chest, depression, and of feeling strange things were going on about her. She thought people were following her.

Her mother died when she was three, and her father was murdered when she was five. Until 13, she lived with an aunt who was extremely strict. Then she lived with an uncle and his wife. The latter was very meticulous, and unduly blamed the patient for making the house untidy. To escape this situation, she eloped when 16 with a man, who in three years dissipated her inheritance, was alcoholic and openly promiscuous, and treated her cruelly. Since he took no responsibility for providing for his family, the patient went into the real estate business. She was quite successful until she was 31. Then she met financial reverses; her husband deserted her, and they were divorced. She ran a rooming house and later worked as saleslady. At 27, she had both tubes and her appendix removed. Menstruation stopped. From having been easy-going and indifferent, she became irritable. She had temper spells in which she demanded much of her children and screamed and struck at them. She did not work, as she claimed her health was too delicate. She stayed alone, would not talk with her children's friends, stared out of windows, and was suspicious of her own friends.

After the grippe in December, 1928, when 38, she often acted as if she had been drinking. She smoked excessively; misinterpreted what others said; suspected her daughter of immorality; was convinced she was being followed;

talked of intimate family affairs indiscriminately; was sensitive to noises, had pressure in her ears and was easily fatigued. Certain events bewildered her, and she complained of "imaginations," or what others told her were imaginations. These seemed to be more delusions than actual hallucinations. Her appearance changed, her face and eyelids puffed up, her voice thickened, her skin became dry. She sat for long periods totally preoccupied. Two weeks before admission she complained to the police that she was being poisoned. She thought that she was accused of murder. She went to the police about a purse being taken out of her apartment. Later this was found misplaced. She came willingly to the hospital.

Examination revealed restlessness; some defects in her recent memory; thick, slow speech; retarded movements; anemia; fatigue; shortness of breath; pulse of 68; sweating of the extremities; petechiæ of the lower limbs; albumen in the urine; P. S. P. excretion diminished (40 per cent in two hours); and a B. M. R. of minus 38. Blood pressure 112/85. On thyroid grains 3 to 5 each day, she quickly recovered from her myxedema and from her delusions. B. M. R. rose to — 6 in six weeks and thyroid was stabilized at 2 grains each day. On discharge in June, 1929, two months after admission, she was well except for many domestic worries.

The following year the patient broke off with her lover who drank excessively. She worked up in a department store to assistant buyer in two years, but was discharged because of a tendency to feel persecuted. There was constant quarrelling with her daughter with whom she lived. Disposition was variable. She continued taking thyroid until 1934, though how regularly could not be determined. In the fall of 1934 she complained of being cold, of her face changing in appearance, of dry hands, and thought people followed her in order to do her harm. She screamed and cried; left her daughter to live with a friend; accused other tenants of attempting to harm her, and interfered with their activities. In May, 1935, Phipps Outpatient Clinic diagnosed "myxedema with mild psychosis." B. M. R. was — 32. Thyroid was given but not taken regularly. She became more upset and was admitted for the second time to Sheppard Pratt Hospital in June, 1935.

When admitted, she was unkempt; speech was slow and slurred; mood varied between marked depression, and anger at the way she felt she was being treated. She showed some lapses in recent memory, and was disoriented for time. She complained of "odors" closing up her throat and stopping her heart beating. She thought she would get well if she took thyroid, which she claimed not to have had for the past eight months due to lack of money and thinking she could get along without it. She appeared obese; hair was dry; pulse 84; blood pressure 168/106; skin was dry and scaly; teeth infected; reflexes hyperactive; B. M. R.'s averaged — 18 per cent; blood cholesterol 310 mgms. per cent (normally 190 mgms. per cent). There was a slight secondary anemia and some albumen in the urine. On 3 grains of thyroid a day her B. M. R. became normal within a month. This was stabilized again at 2 grains daily. She was cooperative in the hospital setting. At first she felt that certain difficulties in getting her clothes were due to some purposeful

plot against her, but after a few days showed no paranoid trends. She continued to talk freely of difficulties with her daughter. She regarded them as the latter's fault and the cause of her trouble. She learned to paint and did excellent needlework. Nine months after admission, thyroid was first reduced and then discontinued for one week. She relapsed into a myxedematous condition. She became seclusive, irritable, untidy in appearance, retarded and depressed. Within six days of thyroid medication all of these symptoms disappeared. After 11 months in the hospital, she was discharged recovered. She learned to stay on thyroid, 2 grains a day, and remained well until fall, 1938, when she became upset and uncontrolled in her behavior. She was not taking her medicine. Periodically, there was much dissension between herself and her children. When last seen in March, 1939, she was much improved. However, her color was pasty, her eyes protruded, and she talked rapidly though coherently. She has been referred to a hospital clinic for a new basal and for proper regulation of dosage.

Summary.—A woman of 38, four years after an artificial menopause, showed physical changes typical of myxedema together with a paranoid state. She recovered under thyroid treatment, had a relapse five years later when she ceased taking it, recovered again, and partially relapsed in a week when it was discontinued in the hospital. Thyroid again caused the disappearance of her symptoms. She remained well outside the hospital for three years after leaving except for a period just before Christmas, 1938, when she did not take thyroid.

Second Patient.—Mrs. B., widow, 65, was admitted to the hospital in February, 1927, resistive, critical, feeling misused, talking of suicide, forgetting easily, fearful of losing her mind, having dreams of being in trouble, seeing faces on the wall, and complaining of palpitation, nervousness and a dry mouth.

Her father and four older brothers were ministers. She was the middle of nine children. Following her father's death at 10 she lived with her mother in a churchly atmosphere until the latter died when she was 39. Because of her temper tantrums everyone in the family gave in to her; yet between times she was very timid, liked to make herself a martyr, was secretive, selfish, sensitive, and felt inferior. She had a marked liking for one of her brothers, and disliked the sister who did the most for her. After the death of her mother, she kept house for another family, the B. family. The first Mrs. B. died. Our patient continued to keep house for Mr. B. Then she took up nurses' training for a few months. She stopped that to marry Mr. B. when she was 42. Her marriage lasted 11 years and was the happiest time of her life. When she was 53, her husband died after being shot by a burglar who in turn killed himself. The patient witnessed both deaths. For several months she was upset, then pulled herself together, but continued restless. For eight years, nothing of note occurred. At 61, temper tantrums

recurred with periods of agitation, particularly about money matters. She became concerned about a physician's statement that she should not let a bunion operation make her an invalid. In trying to keep from being an invalid, she became so restless that she thought there was something wrong with her mind, felt tired, had no appetite, talked "wildly." She was then admitted in September, 1926, to a private clinic, where she remained five months. There she complained of ringing in her ears, seeing shadows and mice on the floor, and hearing her name called when nobody was about.

Physical examination showed low blood pressure (105/60) and low pulse of 64, weight of 171 (about 30 pounds over her usual weight), myxedematous facies, chronic arthritis, increased knee jerks, pitting over the shins, dry wrinkled skin, adenoma of the thyroid, and low B. M. R. of −20. On 3 "tablets" of thyroid daily, B. M. R. increased to +8 per cent in three months. Weight decreased to 141 pounds, her expression brightened, hallucinations almost disappeared, and only easy fatigability remained.

After some visits with her sister and being urged to take a trip, she developed influenza, which was followed by depression, confusion, excitement, rebelliousness, terror dreams, hallucinations and refusal to eat. Final diagnosis was "myxedema and La Grippe followed by mental depression and hallucinations."

Because of her refusal to stay at the clinic, she was brought to the hospital protesting and paranoid. Here she was sensitive to noises and showed emotional instability, petulance, memory lapses especially for names, and some insight into her unreasonableness. She complained of dryness of her mouth, palpitation of her heart, chilliness and other somatic symptoms.

At first she was very rebellious and resistive to all care, threw food at the personnel, banged her head against the wall, and talked of suicide. A few days after entrance she said she had had frequent delusions and hallucinations, seeing signs and feeling everyone was against her. She had the feeling that the doctors were holding her in order to get her money, and that they were neglecting her care and treatment.

Sensorium examination showed slight loss of memory for names and past dates. On occasion she seemed disoriented for time, but not always. Her inability to do the 100 −7 test was apparently on the basis of fleeting attention.

Physical examination, to which she was uncooperative, showed irregular enlargement of the thyroid, enlarged cervical glands, blood pressure 145/85, pulse 88, weight 131, lifelong strabismus, tremors of her lips and hands, edema about her feet and eyes, and retinal blood vessels normal. Laboratory reactions were negative. B. M. R. results were too unsatisfactory to interpret. Diagnosis was psychosis with somatic disease in which senile elements, influenza and myxedema were stressed. In seven months, she improved markedly, becoming more agreeable, active and sociable, and had only occasional days of depression and some suspicion about the handling of her financial affairs. She had only hygienic care and symptomatic medication, and no thyroid.

Seven months after admission, she left the hospital (September, 1927) and voluntarily put all of her business affairs in the hands of a capable person. She took great pleasure in a trip to California (May, 1928), and became more reconciled to her stay in the hospital, although she still resented it as being unnecessary. A physical checkup at Christmas, 1938, showed her to be in good condition for her age of 78. She has never taken thyroid extract since her stay in the clinic. She is mentally alert; she lives alone, does her own shopping, and takes care of herself adequately. She is travelling to California this spring (1939).

Summary.—A woman of 65, tragically widowed 14 years before, became irrascible and agitated over a period of four years because of nightmares, hallucinations of her name being called, and of mice and shadows on the floor. She complained of tiredness, chilliness, and poor appetite. For five months she was in a competent private clinic, diagnosed myxedema, given thyroid, and improved until she had an attack of influenza. Then she became confused, hallucinated, was rebellious, refused food, refused to stay at the clinic, and thought the doctors were against her; so was transferred to the hospital where she stayed seven months, recovering without further thyroid treatment. For the past 11 years she has maintained good health outside the hospital without thyroid.

Discussion.

These two reports offer material for discussion of the question: What is psychosis with myxedema? Let us begin by discussing what it is not. It does not include those psychoses which occur coincidentally in the course of myxedema and are uninfluenced by the administration of thyroid extract. Such was the situation, reported by Jacobi,[6] of a patient who presented a typical picture of schizophrenia. Thyroid treatment improved the myxedema, but the schizophrenia remained. Akelaitis[18] reports two such patients and feels that it is pertinent to distinguish them from true psychosis with myxedema. However, there are enough reports in the literature like that of Mrs. A. to indicate that psychoses can be caused by, or at least be dependent upon, thyroid hypofunctioning.

Mrs. A. developed clear-cut myxedema associated with a paranoid psychosis. Under thyroid she recovered from both psychosis and myxedema. On three subsequent occasions, two of them outside the hospital, thyroid medication was reduced and omitted.

Each time there was a relapse, with subsequent recovery under thyroid treatment. On her second admission to the hospital she ascribed all of her difficulties to lack of thyroid, and asked for it. Mrs. A. had a true psychosis with somatic disease; that is, with myxedema there was a psychosis; without it, there was simply a worrisome person who felt that her difficulty was the fault of others. In other words, the psychosis arose in the course of myxedema and was dependent upon it.

Wegener [11] leaves unanswered the question of whether the psychosis is specific for myxedema, while Akelaitis [18] and Parker [15] answer it in the negative. From what we know of psychosomatic problems, we believe that there must be a relationship, but just what we are not able to say. The two episodes of our first patient were characterized by excitability, irritability, seclusiveness, preoccupation, paranoid ideas, suspicions, and feelings of strange happenings. Many of the cases in the literature show, in addition, depression, confusion, clouding of consciousness, disorientation, vivid hallucinations, particularly of small objects or animals.[5] While these are very frequent disturbances in myxedema, they are also frequent in other psychoses with somatic disease, in psychoses of the toxic-delirious type, and even in functional psychoses. In both of our patients similar psychic disturbances followed attacks of grippe.

Hypofunctioning of the thyroid gland as well as administration of thyroid substance does not create anything in the personality not already there, nor does it remove something from the personality. It does, however, accelerate or retard processes already going on. Without an already existing capacity to develop psychosis, myxedema could never bring it on.

Psychosis, however, must not be diagnosed merely on the basis of symptoms of depression and irritability. Anyone finding himself afflicted with marked reduction of his vital processes is likely to react to his condition with such symptoms. Among the cases reviewed, for example, was one diagnosed as myxedema without psychosis, in which there were personality changes with paranoid trends, but these were insufficient for the diagnosis of psychosis or to necessitate hospitalization for long.

To summarize, the physical findings and bodily symptoms in these cases are typical of and specific for thyroid hypofunction-

ing; the changes in behavior and mentation are typical of and specific for the particular individual and can be seen in the individual's reaction to other like illnesses; if these personality changes go so far as a psychosis it is the same sort of psychosis as might occur in this individual under some other influence. For example, both of our patients had influenza followed by emotional changes similar to those accompanying their myxedema. Hence, psychoses dependent on myxedema do occur, but they are due more to specific psychological characteristics of the individual than to organic changes.

In connection with Mrs. A. another question arises: why did she stop taking thyroid and relapse the second time? She gives as reasons the cost of the medicine and thinking she could get along without it, but these are obvious rationalizations. Were her relationships with her daughter and son, as well as with her lover, too much for her precariously balanced personality, so that there was some point in becoming ill again? I do not refer here to secondary gain in the usual sense, but to that general human feeling that it is more acceptable to fail with an excuse than without one. The social worker informs us that the patient's son expects too much of his mother in the way of cooperation in his ambitious money-making ventures. At these times, mother and son get along badly, and when the patient is upset, neither daughter nor son assumes responsibility for her care. Apparently it is at these times that she fails to take her medicine. In the last analysis, then, Mrs. A.'s second psychotic episode in June, 1935, and her "uncontrolled behavior" around Christmas, 1938, were psychogenic in the sense that there were emotional reasons for her becoming so sick that she did not take thyroid.

We hear much about the effect of thyroid hypofunctioning upon the personality, but it is just as pertinent to make inquiry into the sort of personalities which seem to develop myxedema spontaneously. In the two patients reported here, the myxedema itself arose in a setting of years of chronic dissatisfactions and obvious childhood maladjustments. There is a suggestion here for further and more definite correlations, in which the parental constellation is also of interest. The usual question asked by medicine is: What is the organic structure involved in the development of psychic illness? We can legitimately turn the question around to

ask: What are the psychological characteristics involved in the development of organic illness, such as myxedema? Unfortunately, we cannot answer this question from these two patients as they were not studied psychoanalytically.

Endocrine interrelationships are of importance in the development of myxedema inasmuch as the latter frequently develops in periods of biological stress, such as the menopause. Immediately after removal of her ovaries, Mrs. A. showed a change of disposition. Four years after artificial menopause, she developed myxedema. This fact confirms the interrelationship between ovarian and thyroid functioning. It suggests that when ovaries stop functioning either naturally or artificially, treatment with thyroid is in order, controlled of course by basal metabolic determinations.

Mrs. B.'s story is not so typical as that of Mrs. A. Evaluation of Mrs. B.'s situation was complicated by physical changes due to her age, and by the fact that she got over her psychosis and maintained her health without benefit of thyroid medication. Yet she was definitely diagnosed as having myxedema by an internist familiar with myxedema and its mental changes.[5] In fact, she got much better on thyroid medication before entering the hospital.

This makes all the more pertinent the personality of the individual and the treatment of that personality. In the therapy of both of our cases, the effect of separation from the family and the importance of the general hospital atmosphere and opportunities for recreational and occupational interests cannot be overestimated. It is a question whether Mrs. A. would have obtained as beneficial results by taking thyroid at home. In any case we know from the outpatient clinic that she had the opportunity to take it but frequently did not.

In the case of Mrs. B. senile changes were deemed important. This is interesting in the light of theories that waning thyroid secretion is responsible for atheromatous changes. However, she showed no definite arteriosclerotic changes on physical examination. Whether senile changes as indicated by memory lapses and occasional disorientation had any essential relationship to her psychosis is doubtful. She has since maintained herself outside the hospital for 11 years. Furthermore, recent pathological studies of the brain show no definite correlation between senile changes and mental symptoms.[20]

Conclusions.

1. Two cases of myxedema associated with psychosis are presented: in one, recovery seemed definitely dependent on thyroid medication, and in the other it did not.

2. Even in elderly and apparently senile individuals with myxedema, caution is needed in ascribing mental symptoms and disturbances in behavior to organic changes in the brain or even to hypofunctioning of the thyroid gland.

3. Emotional and personality factors need to be studied to understand the type of psychosis and personality reaction to the myxedema, and to understand the relapses due to neglect of medication.

4. Psychoanalytic investigation of persons with myxedema is desirable in order to answer the question as to what personality structure favors development of myxedema.

BIBLIOGRAPHY.

1. "Report on myxedema." Tr. Clin. Soc. London, 1888. 26 (supplement). Quoted by Ziegler.[9]
2. Kraepelin, Emil: Zur Myxödemfrage. Zentralbl. Neurol., 9: 65, 1890.
3. Pilcz, Alexander: Zur Frage des Myxödematosen Irreseins und der Schilddrüsentherapie bei Psychosen überhaupt. Jahrb. f. Psychiat. u. Neurol., 20: 77, 1901.
4. Sierau, M.: Zur Frage des Myxödematosen Irreseins. Mitt. a. d. Hamb. Staatskrankenanst., vii: 1-15, 1906. Also, Jahrb. d. Hamb. Staatskrankenanst., xi, pt. 2: 1-15, 1907.
5. Beck, H. G.: The hallucinations of myxedema. Med. Times, 54: 201, 1926.
6. Jacobi, Erich: Myxödem und Psychose. Archiv. f. Psychiat. u. Nervenh., 86: 426, 1928-29.
7. Shrijver, D.: Myxedema and psychosis, Nederl. Tijdschr. v. Geneesk., Haarlem, lxxiii, pt. 1: 3144-51, 1929.
8. Tas, J.: Mental depression in myxedema. Nederl. Tijdschr. v. Geneesk., Haarlem, lxxiii, pt. 2: 5093-5105, 1929.
9. Ziegler, Lloyd H.: Psychoses associated with myxedema. Jour. Neur. and Psychopath., 11: 20, 1930.
10. Hayward, E. P., and Woods, A. H.: Mental derangements in hypothyroidism. J. A. M. A., 97: 64, 1931.
11. Wegener, Hermann: Myxödem und Psychose. Zeitschr. f. d. ges. Neurol. u. Psychiat., 143: 542, 1933.
12. Parker, H. L.: Two cases of myxedema with nervous and mental features. Proc. Mayo Clinic, 8: 721-31, 1933.

13. Courtois, A., Pichard, H., Liber, A.: Myxoedèma acquis avec troubles mentaux. Annales Medico-psychologiques, xivs, tome 2: 206-9, 1933.
14. Bertolani Del Rio, M.: La Vita Affetiva nel Mixedema conclamato. Riv. pat. nerv., 41, 130-9, 1934.
15. Parker, H. L.: The nervous and mental manifestations of myxedema. Irish Journal of Medical Science, 6s, pt. 1: 124-7, 1935.
16. Karnosh, L. J., and Stout, R. E.: Psychoses of myxedema. Am. J. Psychiatr., 91: 1263, 1935.
17. Dunbar, H. Flanders: Emotions and bodily changes. P. 161. New York, 1935.
18. Akelaitis, Andrew J. E.: Psychiatric aspects of myxedema. J. Nerv. and Ment. Dis., 83: 22, 1936.
19. Ruhberg, George N.: Myxedema—its nervous and mental manifestations. Minnesota Medicine, 19: 637-41, 1936.
20. Rothschild, D.: Pathologic changes in senile psychoses and their psychobiologic significance. Am. J. Psychiatr. 93: 757, 1937.
21. Simon, A.: Myxedematous hypothyroidism and psychosis. Ill. Med. Journal, 75: 66-9, Jan., 1939.

DISCUSSION.

Dr. C. MacFie Campbell (Boston, Mass.): The two patients who have been discussed here are obviously very interesting cases that present many questions. Dr. Crowley has presented several questions to us but has refrained from answering them, and I shall not be daring enough to offer any explanation. The problems that have been brought up cover an extremely wide field. It would be rather difficult to formulate these questions in a precise way so that a discussion might be productive.

With regard to the occurrence of these psychoses in patients with myxedema, obviously they are very rare in a mental hospital and one might wonder why that is so. Perhaps it is because patients with hypothyroidism and with marked myxedematous conditions are of interest to the internist. They can be treated in a general hospital or at home, they seldom require admission to a mental hospital; as a rule your myxedematous patient has good insight into his or her condition.

The condition of the myxedematous patient often appears to be one of considerable depression, but when inquired into one often finds that it is more a slowness, a difficulty recognized by patients. There is a certain stickiness of thought and of action which the patient feels somewhat discouraged about. The discouragement I think is a mild reaction to this difficulty recognized by the patient as such, and that corresponds to what we find in cases of hyperthyroidism, where, with the over-activity and the over-productivity, the pathological nature of the condition is not infrequently recognized by the patient himself or herself. The patient may complain of not being able to sit still or of not being able to stop talking. They realize their own mental instability.

I should think, therefore, that the type of disorder that we see most frequently in myxedema is a disturbance of *mood* and of activity as a rule, recognized by the patient himself or herself, and for a study of mental changes in the myxedematous, I think one would go to a general hospital where one could get a series of such patients and find out what are the fundamental reactions that accompany disordered thyroid conditions.

If we take patients who are brought into mental hospitals, they are brought in for complications, and as to the relationship of these complications to the basal physical disorder interpretation may be difficult. If a myxedematous patient were somewhat discouraged and tried to commit suicide with resultant admission to a mental hospital the whole series of events would appear to be sufficiently intelligible.

As to the cases presented to us, we have in the first case, I judge, an extremely unstable individual, if one can take an elopement as an early indication of at least an unusual method of dealing with problems, and throughout her life there had been a difficulty of social adaptation, many distressing emotional factors and emotional tensions, and we find that in her psychosis she developed a hallucinatory delusional condition. The question might come up in our formulation of such a case how far the symptoms are to be correlated with hypothyroidism, and how far they represent special characteristics of the individual personality.

The patient whose general mental instability seemed to vary with the functional efficiency of the thyroid, or at least with the administration of thyroid extract, may be compared to other rather unstable individuals who react with transitory hallucinatory delusional states to minor infectious disorders, or to alcoholic indulgence.

In the second case I got the impression, perhaps erroneously, that at first during a period of several months the patient was treated with thyroid extract.

Dr. Crowley.—Yes.

Dr. Campbell.—And then later had a relapse with grip but got no further thyroid extract and for the following ten or eleven years got along admirably. It is not easy to interpret that, and this would be an unusual course in a case of myxedema.

Dr. Ralph M. Crowley (Washington, D. C.).—Dr. Campbell has commented upon the question why psychosis is so rare. That may fit into our ordinary observation that organic illnesses often clear up psychoses, so that one might expect them to be rather rare in organic illnesses like myxedema.

He has also commented upon the reaction to the myxedema itself as not constituting psychosis. I think this is very true and one cannot diagnose psychosis just on the basis of the depressive reaction which occurs very frequently. Studying such patients in general hospitals, I think would be very valuable and calls attention to the need for union between psychiatrists and internists.

I thank Dr. Campbell for his discussion.

BEHAVIOR DIFFERENCES IN MENTALLY RETARDED CHILDREN MEASURED BY A NEW BEHAVIOR RATING SCALE.*

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In previous studies we have differentiated two clinical types among children whose intelligence quotients fall within the range of the high grade moron and borderline classifications. These two types, the exogenous and endogenous, are defined by us as follows. The exogenous type includes those children in whose histories there is evidence of some physical damage (destruction through injury or inflammatory process) to the central nervous system which might account, in part at least, for the existing deficiency. Also, in these cases, history of mental deficiency is not found in the immediate family. The endogenous type comprises individuals whose histories reveal no evidence of such damage and in whose immediate family history of mental deficiency is determinable. Furthermore, clinical examination of the children in this second class shows no neurological signs of brain damage.

In previous studies, differences between these two types have been shown in the relationship between physical and mental growth,[9] in the rate of mental growth under constant environmental conditions,[10] and in changes in rate of mental growth under changing environmental conditions.[7] Our observations lead us to believe that there is a similar difference in behavior.

The present paper is designed to determine whether or not measurable differences in behavior exist between these two types.

In the usual type of rating scale, the author of the scale forms a subjective judgment of the exact nature of a given item. In using the scale, it is necessary that the rater form a similar subjective judgment as to the exact nature of the item. Subsequently, the rater must compare his observation of the occurrence of the item with his

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

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subjective norm for a general population. It is necessary to assume in such a scale that the subjective judgment of the author and the rater are identical and that the subjective picture of the norm for such behavior is the same for both author and rater.

To minimize these difficulties, the present scale attempts to have the rater describe behavior rather than to judge behavior. It is, in effect, a check list by which there can be elicited from the rater a controlled description of the child's behavior. No question is raised regarding the relative extent of such behavior.

Behavior aberrations resulting from brain damage of varying etiology have been observed and reported in the literature (Hohmann,[5] Gerstmann and Kauders,[3] Schroeder,[8] Kasanin,[6] Kahn [11]). The behavior pattern associated with such damage has been described as unstable, uninhibited, uncontrolled, etc. It was felt that words describing such general behavior trends paired with their opposites as items in a rating scale would provide the best method for the descriptive differentiation of behavior required by this study.

The combining of such a series of paired words into a rating scale is based upon the method of Gruhle.* [4]

In selecting the items for such a scale, trait names must be found which are descriptive rather than judgmental. The problem of classifying trait names as to their attributes has been undertaken by Allport and Odbert.[1] They classify all the trait names of the English language under four headings, the second of which is defined as "terms primarily descriptive of temporary moods or activities." From the words classified under this heading, all those which were thought to be descriptive of the organic behavior pattern were selected. By this method words describing behavior in the three fields of mood, social relations and activity were chosen. Since behavior disturbances are also reflected in expressive movements, words chosen from a similar psycho-lexical study by Allport and Vernon [2] were added. All words thus chosen were paired as nearly as possible with the word denoting the opposed trait. The 99 pairs thus developed formed the preliminary scale.

This preliminary scale was applied to a group of 80 children. Forty of these children were diagnosed as endogenous and 40 as

* Gruhle himself based his method upon the characterological system of the philosopher Sigwart (1881).

exogenous. For the latter, the diagnoses were as follows: 22 birth injury, 6 prematurely born, 4 inflammatory process, 4 trauma after birth, 3 neurological findings the etiology of which was not known, 1 prenatal disease. No child showing gross motor defect was included.

The endogenous and exogenous groups were matched to chronological age, I. Q., and sex. The mean chronological age in each group was 16.9 years. The mean I. Q. was 59 in the exogenous group and 60 in the endogenous group. Each group included 27 boys and 13 girls. The exogenous group had been resident in the institution an average of four years and three months at the time the scale was administered, whereas the endogenous group had been resident three years and seven months. Exogenous children, being sooner recognized as social problems in the community, tend to be committed to institutions at an earlier age. This fact probably accounts for the difference of eight months in the length of institutional residence of our group. In view of the fact that the mean period of residence for the total group is so long in all the present cases, it is felt that this difference does not affect the results of the study.

Teachers in the institutional school system served as raters. The following instructions were found printed on the face sheets of the scale. This printed material constituted the only advice given to the raters.

DIAGNOSTIC BEHAVIOR RATING SCALE OF PERSONALITY IN MENTALLY DEFICIENT CHILDREN

Instructions: On the following pages you will find a list of paired words. These pairs are considered to be opposite in meaning. Each word describes a type of behavior in children. By marking certain of these words you can give a description of the behavior of this child. This group of marked words will then describe his behavior in the same way that you would describe it were you asked directly. Make no attempt to evaluate the behavior—merely describe what the child actually does.

Possibly none of the specific words listed will completely fit the behavior of this child. There are three possibilities of marking: (1) If one word in a pair adequately describes the behavior of this child mark only that member of the pair; (2) if both members of a pair adequately describe the behavior of this child, underline both members of that pair; (3) if neither member of a pair describes adequately the behavior of this child, do not underline either member of that pair. This scale is so constructed that the second and third type of marking will occur very rarely. Describe the most outstanding behavior.

The scale is divided into two parts. The first part describes expressive movements. These are motor behavior in general and bodily and facial movements in emotional states. The second part describes the general personality make-up of the child.

Please consider each pair carefully.

SCALE.

PART I.—EXPRESSIVE MOVEMENTS.

1 graceful—clumsy
2 smooth—jerky
3 normal—peculiar
4 energetic—inactive
5 purposive—aimless
6 impulsive—cautious
7 certain—hesitant
8 mature—childish
9 restless—placid
10 shifting—steady (eyes)
11 chattering—silent (talking)
12 clear—confused (speech)
13 hearty—flat (laughter)
14 explosive—soft (laughter)
15 noisy—quiet (laughter)

PART II.—PERSONALITY.

16 fluctuating—constant
17 excessive—moderate
18 daydreaming—present
19 patient—impatient
20 temper outbursts—self-controlled
21 fearing—intrepid
22 furious—restrained
23 flighty—steady
24 confused—clear
25 erratic—stable
26 temperamental—stolid
27 excitable—not excitable
28 lonesome—sociable
29 cooperative—uncooperative
30 misfit—belonging
31 acceptable—bystanding
32 popular—unpopular
33 leader—follower
34 annoying—undisturbing
35 attractive—repellant
36 critical—acceptable
37 clear-headed—muddled
38 hit-or-miss—planning
39 accurate—inexact
40 neat—slovenly
41 distractible—concentrating
42 careful—careless
43 dependable—undependable
44 energetic—lazy
45 reliable—unreliable
46 restless—settled
47 awkward—skillful
48 reckless—cautious
49 fidgety—balanced
50 decisive—drifting
51 aimful—aimless
52 dreamy—alive

As data for preliminary scoring, only those items in which one member of a pair was marked and the other was unmarked were used. The number of responses to the first member and the number of responses to the second member of each pair of words were computed separately for each of the experimental groups. Using

the exogenous-endogenous classification as a twofold variable, coefficients of mean square contingency were computed for each of the 99 items. By means of these coefficients of contingency those items which were diagnostic were selected. Fifty-two items were thus obtained.

In developing a method of scoring, we identified the member of each pair which was underlined more frequently in the exogenous group and less frequently in the endogenous group. Numerical weights were assigned to each of these members according to the extent to which it differentiated between the two experimental groups as revealed by the coefficient of contingency. The weights thus obtained varied from one to three. The total possible score became 88. Each individual blank was then scored by totaling the weights of the exogenous items underlined. By this method of scoring, the total score indicates the relative amount of behavior typical of exogenous children noted by the rater.

The question of reliability of this scale was investigated by the split-half method. The scores on the odd numbered items and the scores on the even numbered items in each blank were computed separately. This was done for each of the eighty children rated. These two partial scores were correlated. The resulting coefficient was corrected for the decreased length of the test by the use of the Spearman-Brown prophecy formula. This procedure resulted in a corrected reliability coefficient of .918. This coefficient would indicate a relatively high reliability for this scale.

The extent to which this scale differentiates between the two experimental groups is shown graphically by the two frequency distributions presented in Fig. 1.

The amount of differentiation is indicated first by a comparison of the mean scores. The mean score of the exogenous group was 45.56, the standard deviation of the distribution being 14.17. The mean score of the endogenous group was 25.94, the standard deviation of the distribution being 13.87. This results in a difference between the means of 19.62. The probable error of the difference is 2.11 and the resulting critical ratio, 9.30. Since a difference between the mean of four times its probable error is considered to indicate a statistically significant difference, the critical ratio obtained here indicates that this scale has reliably differentiated these two groups in terms of mean score.

The separation of these two groups by this scale can be further shown through the amount of over-lapping of each distribution upon the other. The percentage of over-lapping upon the median was therefore computed. The median score for the exogenous group was 42.5. This score was reached or exceeded by 20 per cent of the endogenous group. The median score for the endogenous group was 24.5. The scores of 15 per cent of the exogenous group fell at or below this point. These percentages would indicate a

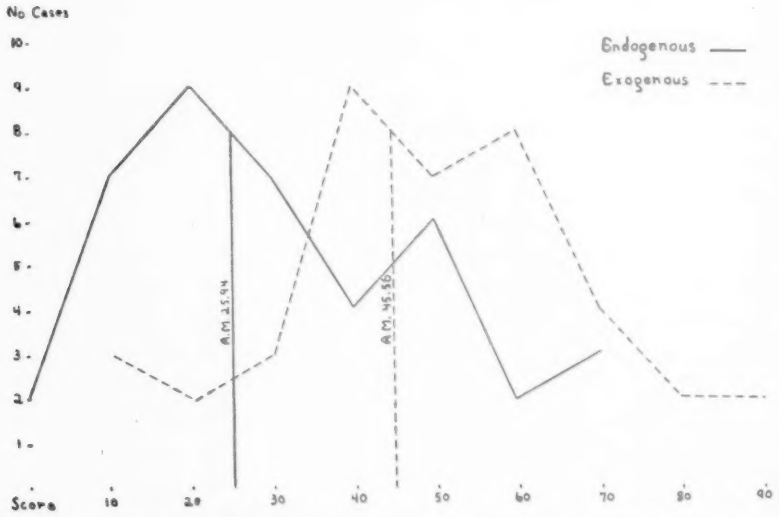


FIG. 1.—Distributions of Behavior Rating Scores for the Two Clinical Types.

rather small amount of over-lapping on the median. It must be borne in mind that the clinical diagnoses also involve a certain unreliability. This is particularly true in the endogenous group which is partly a diagnosis by exclusion. The fact that no brain damage is indicated in the case history does not exclude the possibility that such an organic factor may be present but not noted.

CONCLUSIONS.

The standardization of this scale is not considered to be adequate. The number of cases is too small and the sampling is limited

to the high grade moron and borderline levels of intelligence and to children in an institutional environment.

It was possible, however, to differentiate two groups of mentally retarded children, the one of which showed evidence of brain damage and the other of which showed no evidence of such damage, by means of this scale.

The scale shows the exogenous group to be erratic, uncoordinated, uncontrolled, uninhibited and socially unaccepted.

This study suggests that behavior problems occurring in endogenous retarded children are completely apart from the commonly accepted picture of organic behavior.

The question of the further differentiation of psychopathic behavior in children is a question for future research.

BIBLIOGRAPHY.

1. Allport, G. W., and Odbert, H. S.: Trait-names: A psycho-lexical study. Psychol. Monog., 211, 1936.
2. Allport, G. W., and Vernon, P. E.: Studies in expressive movements. New York, Macmillan, 1933.
3. Gerstmann, J., and Kauders, O.: Ueber den Mechanismus der postencephalitischen "psychopathieähnlichen" Zustandsbilder bei Jugendlichen. Arch. f. Psych. u. Nerv., 71: 165, 1924.
4. Gruhle, H. W.: Anregungen zur Charakterforschung. Z. f. Kinderforsch., 28: 305, 1924.
5. Hohmann, L. B.: Postencephalitic behavior disorders in children. Johns Hopkins Hosp. Bull., 33: 372, 1922.
6. Kasanin, J.: Personality changes in children following cerebral trauma. J. Nerv. and Ment. Dis., 69: 385, 1929.
7. Kephart, Newell C., and Strauss, Alfred A.: A clinical factor influencing variations in I. Q. Accepted for publication, Amer. J. Orthopsychiat., 1939.
8. Schroeder, P. L.: Behavior difficulties in children associated with the results of birth trauma. J. Am. Med. Assoc., 92: 100, 1929.
9. Strauss, Alfred A.: Effects of exogenous and endogenous factors in the organism as a whole in mentally deficient children. Paper read before the Society for Research in Child Development, 1938.
10. Strauss, Alfred A., and Kephart, Newell C.: Rate of mental growth in a constant environment among higher grade moron and borderline children. Accepted for publication, Proc. and Addr. Amer. Assoc. Ment. Def., 1939.
11. Kahn, Eugen and Cohn, L. C.: Organic drivenness, a brain-stem syndrome and an experience. New England J. Med., 210: 748, 1934.

DISCUSSION.

DR. JACOB KASANIN (Chicago, Ill.).—What these authors have done is something which I think is quite remarkable. They judge the intelligence of the child, his personality and his behavior largely by the types of reactions which he presents in his ordinary life situations. The child can be thus evaluated and a quality scale can be produced. Undoubtedly this scale can be used on other children, normal children and not only institutional cases, and if the authors adopt the current American technique of using a large number of children and so standardize the test, perhaps we may have criteria for estimating the child's intelligence on the basis of his behavior, without resorting to the use of artificial situations such as are created by the orthodox intelligence tests.

It is also very interesting that the authors try to differentiate between the exogenous and the endogenous cases of deficiency, something which is not particularly common in American practice in the study of mental deficiency.

I realize—and I think that the authors also probably agree with me—that this study is extremely provisional, that it is only the beginning; but it seems to me very promising and is certainly one to continue.

THE CONTROL OF NORMAL AND "CONVULSIVE" BRAIN POTENTIALS.*

By R. W. GERARD and B. LIBET.

From the Department of Physiology, University of Chicago.

The existence of a potential variation associated with the activity of such cells as nerve and muscle has been known for almost a century. Further, it is thoroughly established that such potentials are inevitable concomitants of activity and are acceptable as a measure of function. The adequate recording of voltage fluctuations from the brain or intact head is hardly over five years old—thanks to the improvement of amplifier and oscillograph technics, which permit the recording of potentials of microvolts lasting for milliseconds or less. Since, however, fairly continuous electrical rhythms are obtained from the brain, not overtly related to measurable action, it might be questioned whether in this case, as for nerve and muscle, the potentials can be used to document physiological function.

Various excitant drugs have been shown to produce repeated high voltage waves in the brain which are matched by simultaneous bursts of motor activity, and a similar correlation of brain potentials and motor discharges in epilepsy (Gibbs, Lennox, and Gibbs, 1936) is well established. Conversely, a depression of cortical function, as in sleep or under anesthesia, is associated with a characteristic shift of brain potentials towards slower rhythms; and it is possible rather accurately to gauge the depth of anesthesia and of sleep, even the state of dreaming (Blake, Gerard, and Kleitman, 1939), by the character of the potential pattern. It seems likely, therefore, that for brain also the electrical activity may be used fairly generally as a measure of neurone activity; and its study becomes of much wider significance than would be the case if it were an unattached phenomenon. Even if the electrical variations were an

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, May 8-12, 1939.

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accidental product of activity, like the heart sounds, they would still serve as a useful index of function. Actually, as is becoming ever more apparent, the potentials are more than such a superficial manifestation—they are an integral part of the machinery itself.

The actual potential record obtained on leading from the exposed brain or, less definitively, from the intact scalp is quite comparable throughout the vertebrates. Human, cat, pigeon, or frog manifests a reasonably continuous electrical beat which is sometimes regular and which tends to adhere under standard conditions to a fairly characteristic frequency for each brain part or species. The shape and sequence of individual waves, however, does not necessarily reproduce the potential profile of the individual neurone, for the recorded pattern is the envelope of the separate patterns of large numbers of cells.

Indeed, it is only when a considerable group of these units is acting in reasonable unison that any record is obtained. When many persons sing in harmony, the total sound is essentially a greatly amplified record of that of any individual and the words or melody are well recognizable. When the individuals enter the chorus at their separate whims a continuous, almost constant, noise results, which is unanalyzable. In the same way, the action potential recorded from a nerve trunk is large and smooth when all the fibers have been thrown into simultaneous activity by a single electrical stimulation, while that accompanying a moderately asynchronous reflex discharge is feeble and irregular and sometimes quite undetectable.

Clearly, a preparation containing a fairly small group of neurones, as homogeneous in type as possible, would offer the greatest promise for obtaining a total potential record built of unified rhythms of like character. If, further, the preparation could be removed from the body and, while retaining its activity, be subjected to rather extensive variation of single factors in its environment, it would be still more advantageous. The frog brain, and particularly its olfactory bulbs, weighing only 2 to 3 milligrams, admirably satisfies these conditions; and the experiments to be reported have been carried out mainly on the isolated frog brain. There is no reason to believe, however, that these findings lack general application to other brains; indeed, they have been extensively duplicated on the intact brain of the anesthetized cat.

Two major problems demand solution: first, how is the beat of the single neurone controlled; and second, what are the mechanisms which synchronize the beats of large numbers of independently active neurones?

Control of the Single Neurone Beat.

The frog brain *in situ*, exposed under ether anesthesia and studied after the drug has been eliminated, shows a fairly regular rhythm at some 6 beats per sec. and about 30 microvolts in amplitude. The same brain subsequently removed from the animal and kept moist in Ringer's solution at room temperature manifests a rhythm of the same frequency but of greater amplitude, about 100 microvolts, and much improved regularity. Since it is well established that afferent impulses reaching the brain in non-homogeneous groupings tend to engage part of the cerebral neurones and so disrupt the unified beat of the group—the well known abolition of the human occipital alpha rhythm by visual stimulation is an example of this—the improved amplitude and regularity following isolation may be attributed in part to the removal of a continuous play of afferent impulses upon the brain. A change in injury and other electrical fields may also contribute.

This regular rhythm persists for hours in the olfactory bulb of the isolated brain though the hemispheres behind it and the olfactory nerves rostral to it become completely inactive electrically in a much briefer time. This is strong evidence that the maintained beat of the olfactory neurones is spontaneous, in the sense that it does not depend upon a continuous or rhythmic stimulation of these cells by incurrent nerve impulses. Further evidence of this "intrinsic" control is afforded by the persistence of regular beats under the action of nicotine, to be discussed later. It is possible, moreover, by varying the cell milieu in various ways, to modify almost at will the frequency, regularity, amplitude, and wave shape of the potential rhythm of the bulb or even of a small teased-out bit of it weighing less than 0.1 mm. Characteristic rhythms, all regular, of frequencies from one to 50 per sec. and amplitudes of 5 microvolts to a millivolt have been recorded.

Most significant is the almost machinelike reproduction of some particular wave, which may vary from a regular sine curve to very skewed, notched, and otherwise asymmetrical patterns (Libet and

Gerard, 1939). The exact repetition of such bizarre potential profiles from a small group of cells, probably only a few hundred, could never occur if the individuals were discharging in disorderly sequence. Such unique, yet constant, profiles could only result if essentially all the cells are acting in unison, in which case the recorded potential is that of the individual unit merely magnified in amplitude by the many units acting in parallel; or if the single cells discharged in some recurrent sequence—as the light bulbs flash in order when a pattern moves across an electric sign—in which case the recorded potential documents the sequence of firing of the many cells rather than the potential form of the one. This latter possibility is *a priori* quite improbable and is, indeed, possible at all only as a consequence of trapped nerve impulses running a repeated course around anatomically set neurone circuits. But such a neurone mechanism, involving repeated synaptic transmission, would be eliminated by nicotine which blocks synapses (Libet and Gerard, 1938; Schweitzer and Wright, 1938); yet this drug actually enhances the appearance of regular skewed waves.

It may be concluded, then, that the recorded potential of the isolated bulb is, under a wide variety of conditions, essentially that of the individual neurone; and inferences as to the control of the beat of the single cell may be drawn from the record of the many. This will be true, of course, only so long as good synchronization persists, but this factor can be independently studied.

The maintained potential rhythm of the neurone depends upon a steady flow of energy supplied ultimately by the metabolism of the cell. Since this is normally oxidative, deprivation of oxygen should lead to failure of the potentials. This is particularly well demonstrated in the cat by complete obstruction of the blood supply, for cortical potentials are abolished in about 15 seconds of anoxia. It has even proved possible, on the assumption that the more rapidly respiring brain regions suffer earliest from oxygen lack and so become electricaliy inactive in the shortest time, to place many neural structures in a quantitative sequence of respiratory intensity (Sugar and Gerard, 1938). So far as other data on brain respiration go, they are in agreement with this sequence. (It is irrelevant to the present discussion, but of considerable importance in the interpretation of the various therapies which show some success in the treatment of schizophrenia, that early during anoxia there

is interposed a period of excessive activity between the normal and the depressed states; and that following the restoration of oxygen neurone activity does not merely return to the initial level but again shows a phase of exaggeration, sometimes very marked and prolonged (Gerard, 1938).)

The metabolism and available energy are increased by a rise in temperature. There is a corresponding increase in the frequency, amplitude, and regularity of the potentials. The temperature coefficients for oxygen consumption and rhythm frequency are alike, which further argues the metabolic control of the potentials.

It would be fallacious, however, to conclude, as has sometimes been done, that the rhythm frequency is always a true index of metabolic intensity; for another important factor is involved. A regular rhythm depends on the existence of a constant driving force and of a reliable interrupting or trip mechanism. When a violin string is bowed with constant strength, its frequency of vibration can still be altered by a change in the tautness of the string. The beat of a windshield wiper is speeded up by an increase in the air pressure that drives it, but no less by an adjustment of the valves that control its sweep. In the latter case, an acceleration in frequency is attended by a diminution in amplitude; in the former, frequency can increase while amplitude is maintained or augmented.

In the brain rhythm, likewise, increased frequency with constant or increased amplitude documents a greater driving energy and so greater metabolism. This is clearly the case for a temperature increase, which gives larger and faster waves. On the other hand, an increase in frequency associated with a proportionate decrease in amplitude is most probably the consequence of resetting the trip mechanism of the cell. What this is, is not established, although there are many reasons for believing that it is the surface membrane which plays this rôle and that changes in its permeability, irritability, polarization, and the like determine the time of tripping. (Still another factor modifying amplitude and rate is the degree of synchrony or asynchrony of the separate cells, to be considered later. It seems probable that the progressive building up of potential waves in grand mal epilepsy and their eventual clumping into discrete bursts is largely a matter of improving unison, only partly one of increased metabolism.)

It is desirable, therefore, in studying the influence of various factors controlling brain potentials, to analyze their separate effects on neurone metabolism and neurone membranes. Unfortunately it is rarely possible to make a sharp separation, since the membrane state is itself dependent on metabolism and most, if not all, agents which act upon nerve cells modify both the total energy and the trip mechanism. Still, certain ones exert their effects primarily on the metabolism, others on the membrane.

Metabolism Control.

Factors in the first category include: an absence of substrate or of catalyst and the various inhibitors which prevent the normal

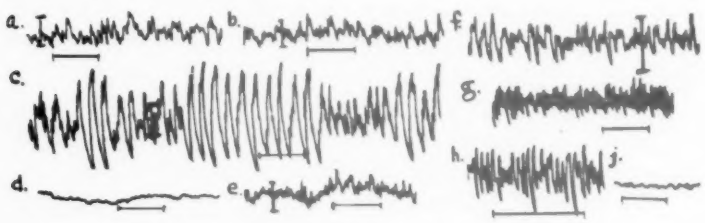


Fig. 1.—Chloroform.

a. Normal; b. in chloroform saturated Ringer's solution for 1 min.; c. 2 min. after b; d. 2 min. after c; e. 3 min. after return to Ringer's solution; f. another brain normal; g. in chloroform-Ringer 3 min.; h. same, faster speed; j. 2 min. after g.
Vertical line 30μV for a through e, 100μV for f through j.

interaction of these substances. Thus, the lack of oxygen or the addition of sufficient narcotic (ether, chloroform, Fig. 1, cocaine, etc.) to interfere with oxidations eventually abolishes brain potentials. Interference with the respiratory catalysts by cyanide has a similar action in the mammal but has led to somewhat anomalous results in the frog, perhaps because frog brain oxidations are only partially inhibited by this substance. Fig. 2 illustrates the action of various concentrations of cyanide on the isolated frog bulb.

Thousandth molar sodium cyanide, a distinctly toxic concentration to mammals, has no effect; 0.003 to 0.005 M cyanide produces smaller and more rapid waves (frequency 30 per sec., regular or irregular) corresponding to the excitation phase of decreased oxygen supply in the cat or, in general, to the overactivity shown by all kinds of protoplasm during the early stages of action of

depressants. (Compare chloroform, Fig. 1.) Further increase in cyanide concentration, however, does not lead to the expected abolition of potentials; indeed, these may continue for an hour with the brain bathed in isotonic sodium cyanide. If such a strong solution is added at once to a fresh brain, potentials are stopped but they may return. When a brain, active in cyanide, is replaced in Ringer's solution, the potentials are promptly abolished, to be restored again by isotonic cyanide. We have at present no satisfactory explanation for this peculiar behavior.

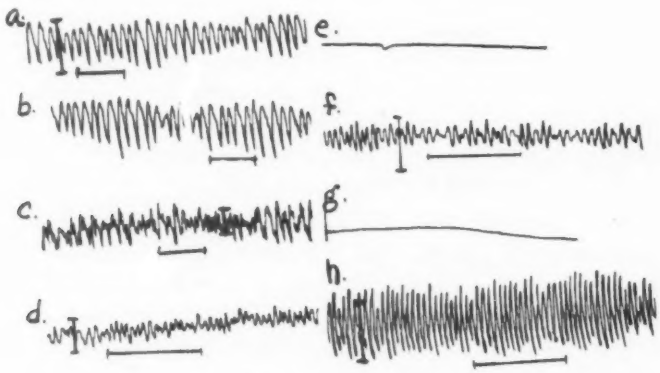


FIG. 2.—Cyanide.

a. Normal in Ringer's solution; b. 0.002 M NaCN for 3 min.; c. 0.005 M. NaCN for 3 min.; d. same, 10 min.; e. Ringer's solution for 3 min.; f. 0.1 M NaCN; g. same, cerebral hemisphere; h. single bulb in 0.1 M NaCN.
Vertical line in a, f, represents 100μV; in others, 30μV.

The normal substrate of brain is glucose. (The RQ reported for frog central nervous system, *e. g.*, Winterstein, 1929, has been below 1.0, usually about 0.9. The RQ for mammalian brain is always 1.0 when technical errors are excluded and perhaps the same would be true for the frog.) When the supply of this sugar is lowered by the action of insulin, the mammalian brain alters its potentials. As in anoxia, narcosis, and cyanide, the waves ultimately become large and slow (Himwich, etc., 1939) but they pass through a transient phase of increase, at least under certain conditions (Dubner and Gerard, 1939).

The isolated frog brain, on the contrary, is completely indifferent to the presence or absence of glucose in the surrounding salt solution. This is perhaps not surprising in view of the normal very

low glucose content of frog's blood (0.012 milligram per cent., Olmsted, 1924) and the considerable carbohydrate store in neural tissues (Holmes, 1929). It is possible, but difficult, to obtain insulin convulsions in the frog. The brains from these animals in insulin convulsions show practically no electrical activity—even the powerful waves induced by caffeine fail to appear. Addition of glucose to the Ringer's solution under these conditions does restore much of the expected potentials.

Sugar metabolism can also be interfered with, so that pyruvate accumulates, by the loss of co-carboxylase which results from B_1 avitaminosis. Potentials of the pigeon brain *in situ* are increased in amplitude and regularity in well developed beri-beri and are rapidly restored to normal by administration of the missing vitamin (Tokaji and Gerard, 1939).

Glycolysis and oxidation of sugar are interfered with by a sufficient concentration of sodium iodoacetate, and this drug applied to the frog brain *in vitro,* although it does not cause cessation of activity, markedly changes the potential pattern (Fig. 3, A). The six a second rhythm is abruptly increased to a regular one at 15 a second, while its amplitude is cut down by two-thirds. Towering above this feeble steady rhythm, there occur extremely regularly, at intervals of 6 to 12 seconds in different cases, large excursions of two hundred or more microvolts and of a polyphasic irregular profile which is repeated accurately in wave after wave. The explanation of these large potentials is lacking, although they call to mind the deep gasps which occasionally punctuate a shallow rapid respiration and which indicate similar large bursts of activity in feebly responding neurones. There is also some reason to suspect that these large potentials, like those to be discussed later, may spread spatially along the brain.

Sodium lactate, which is known partially to neutralize the action of this drug (Holmes, 1933; Chang and Gerard, 1933) has no influence upon the potentials of untreated brain but does shift those of the iodoacetate poisoned one towards normal.

Nicotine blocks lactic acid oxidation by brain and stimulates aerobic glycolysis (Baker and Himwich, 1938), and strikingly alters the electrical rhythm. In 0.5 per cent nicotine the six a second waves are slowed to four a second after some three minutes, and then discontinuously to two or one a second after five to ten

minutes. The amplitude is increased to 300 microvolts or more and the wave form is altered from a roughly sinusoidal one to some of the bizarre skewed forms previously mentioned (Fig. 4). Since this drug blocks synaptic conduction and yet produces extremely regular asymmetrical waves, its action affords especially strong evidence for the earlier conclusion that the recorded potential is that of a single unit magnified by the synchronous activity of many such units.

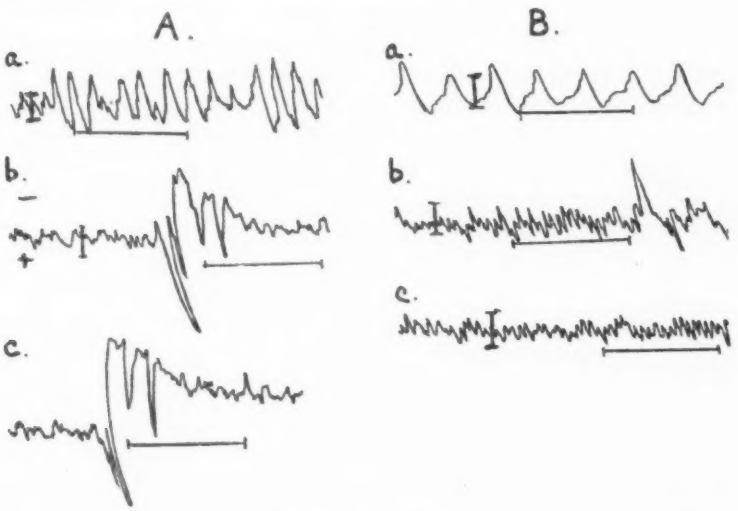


FIG. 3.—Iodoacetate.

A. a. Normal; b. 0.004 M Na iodoacetate for 5 min. (large wave starts with surface positive swing); c. same, showing large complex wave recurring at 12 sec. intervals.

B. a. Freshly isolated brain in 0.5 per cent nicotine for 3 min.; b. 0.004 M Na iodoacetate, 3 min. (large IAA wave recurs every 6 sec. in this case, slow nicotine waves are abolished); c. 0.004 M Na iodoacetate + 0.5 per cent nicotine for 5 min. Note slow nicotine waves do not appear.

Vertical line in B a. and b. represents 100μV; others 30μV.

Nicotine acts very powerfully; we have never failed to obtain the usual potentials even when it was applied to a stale brain whose spontaneous waves had largely disappeared. When combined with iodoacetate (0.004 M), however, the nicotine action on brain waves is completely eliminated and the feeble rapid potentials of the iodoacetate alone appear (Fig. 3, B). It is suggestive that the action on cell potentials of nicotine, which increases lactic acid production, is abolished by iodoacetate, which prevents glycolysis.

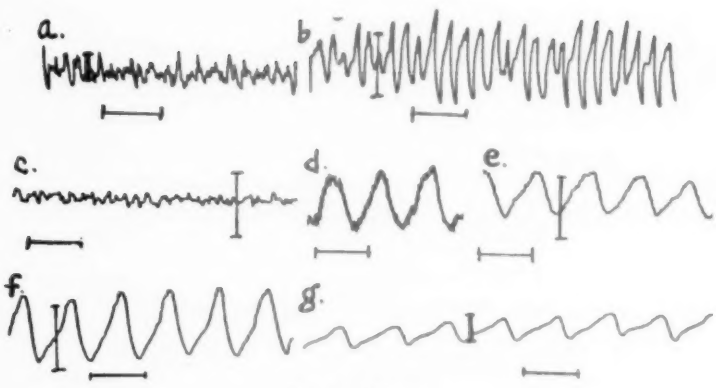


FIG. 4.—Nicotine.

a. Normal; b. 0.5 per cent nicotine for 3 min.; c. back in Ringer's solution for 3 min.; d. 0.5 per cent nicotine for 5 min.; e. same, electrodes moistened; f. nicotine for 5 min., other olf. bulb; g. 3 min. after f.
Vertical line in a. represents 30μV; others 100μV.

TRIP MECHANISM CONTROL.

Turning next to the group of agents which presumably act mainly upon the trip mechanism, the findings may be stated in summary fashion, since they are published elsewhere in sufficient detail (Libet and Gerard, 1939).

Doubling the osmotic pressure with sucrose causes the appearance of large slow waves. Replacing the normal intercellular salts with an isotonic solution of this inactive molecule has a similar effect, and produces also groups of large spike-like potentials which perhaps spread over the brain. Substituting sodium, while retaining normal potassium and calcium concentrations, has a similar action to total salt replacement but the effects are slower and less pronounced. In both cases the waves become more regular.

An increase in calcium or magnesium, with no change in the concentration of other salts, also shifts the potentials in the direction of large slow waves; while an increase of potassium or decrease of calcium, by addition of citrate, leads to small rapid ones. With a shift to slow rhythms there is also an improved regularity; with a shift in the other direction the waves become progressively disrupted. A considerable change in hydrogen ion concentration, either up or down, also produces larger and faster potentials. Over a smaller range, an increase in pH, but not a decrease, seems to have

a stimulant action. These various ions are known to act directly on cell membranes; and, further, the frequency changes they induce are commonly sudden and discontinuous—as for a change in the trip mechanism—rather than simply progressive. Increased sodium and, particularly, potassium tend to make membranes more permeable and so to decrease polarization and increase irritability; calcium and magnesium do the reverse. These substances, however, also produce equivalent changes in metabolic rate, so that it cannot be stated that they modify only the trip mechanism.

Stimulation of the olfactory nerves or direct faradization of the bulb increases the amplitude of the regular waves, for as long as 15 minutes after a few seconds tetanization, and also develops a regular high frequency ripple on the slow waves. Stimulation, of course, produces both surface and metabolic changes. The increased activity of the brain rhythm follows after a fairly uniform barrage of impulses upon the cells, and the resulting increase in state of excitation; in contrast to the disruption of the rhythm produced during the inflow of nonhomogeneous volley patterns.

Perhaps the most certain way of producing a change in the cell surface, as the primary effect, is by applying a constant current. It was previously shown for the cat's brain that steady polarization rendered the spontaneous waves of the lateral geniculate body much larger and more regular. Even more striking effects are produced in the isolated frog brain when a current of as little as 60 microamperes is passed through its length via non-polarizable wick electrodes. With the cathode on the bulbs and the anode on the occipital pole of the hemispheres, that is with an ascending current, the spontaneous potentials of the bulbs are depressed or abolished during current flow. Immediately following interruption of the current, and continuing for some minutes before settling down to the normal rhythm, the bulb shows a greatly exaggerated rebound over-activity (Fig. 5). With a descending current the picture is the exact reverse, potentials being greatly increased in amplitude and frequency while the current flows and disappearing entirely when it is terminated.

Such constant currents alter membrane polarization and threshold and may, of course, directly excite. It is striking that the cathodal region is depressed during current flow and hyperactive following (with reverse effects at the anode), which is the exact opposite of

a current's action on nerve. Further, it is significant that in the frog brain there is this marked directional quality of polarization, for it proves the pre-existence of electrical asymmetries. This will be considered later in more detail. Polarization along other than the longitudinal axis has not yet been fully explored. The effects are less with the electrodes in symmetrical lateral positions and will

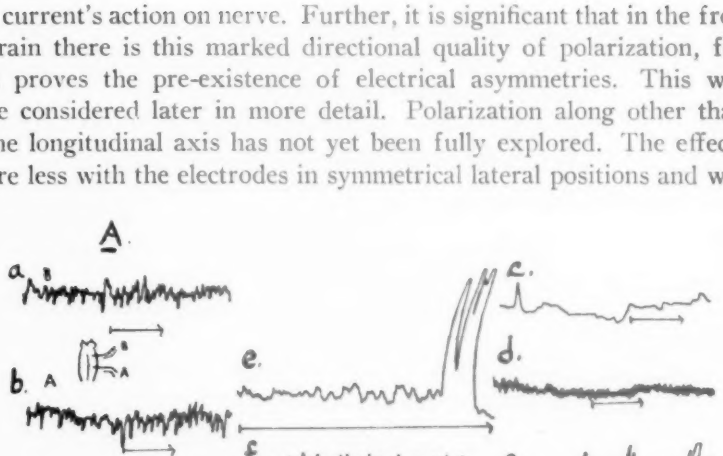


FIG. 5.—Polarization of cerebral hemisphere.

A. a. Before polarization; b. ascending current of 0.06 mA flowing for 3 min.; c. just after polarizing current stopped; d. 30 sec. later; e. polarization with descending current; f. just after current stopped. (Read down left side for A.)
B. Another brain; a. before polarization; b. descending current on; c. current off; d. ascending current on; (most of the low voltage high frequency component here is artefact); e. current off; f. 1 min. later after e.

perhaps be greater with currents led rigorously perpendicular to the hemisphere, as between the ventricular and pial surfaces.

MECHANISM OF CELL SYNCHRONIZATION.

The findings and analysis presented have led to the second large problem, of the unification of the beat of many cells, which must now be considered in further detail. When many individual units are active in a regular time relationship, either all simultaneously or in rapid succession, some integrating influence must be at work. Such synchronization is present at all levels of organization and

the general phenomenon has been discussed elsewhere in relation to neurone beats (Gerard, 1937; see also Gerard, Marshall, and Saul, 1936). More specifically, in the case of the brain, one must assume some pace-making cell or cell group which initiates each beat and whose activity sweeps into action the other units, either very rapidly or in slower succession. The pace-maker, of course, need not be anatomically fixed, but could shift about as local excitation and other conditions bring one or another cell group into physiological dominance.

The possible correlating mechanisms are few. The spread of substances from active to inactive regions cannot serve as a means of integration, certainly in the isolated brain, for diffusion is far too slow and circulation is non-existent; even with blood flow intact, the blood does not move any distance in the plane of the cortical or other cell layers. Stimulating radiation would at present be a pure *ad hoc* assumption for even the mitogenetic rays claimed by some workers to accompany neural activity have not been accused of being able in turn to stimulate inactive units. There remain two mechanisms: the transmission of nerve impulses along anatomically available fiber pathways which carry excitation from cell to cell, or group to group; and the electrical fields and currents spreading from active cells though the brain mass, mainly in between nerve processes, with the intensity of current flow determined by the electrical characteristics of the mass.

There is abundance of evidence that nerve impulses arising in an active region of the brain can serve to set up activity elsewhere, close or far, so that this means of cell integration can be taken for granted. Little attention has been paid, however, to the electrical mechanism; and the remainder of this paper is concerned with evidence that this also, even when propagated impulses are eliminated, is able to bring about coordinated cell beats.

Effective cell integration depends on: the intensity of the integrating force at its point of origin; the distance and intensity of its spread through the neural mass; and the sensitiveness to it of the units upon which it acts. In the case of an electrical mechanism these factors become: the potential field or change in this field of the pace-making units; the pre-existing potentials and impedance encountered along possible circuits through the tissue mass; and the threshold of other units, cell bodies or their processes,

for the resulting currents. An improvement in integration, leading to more perfect synchrony, would result from a favorable shift in any or all of these factors.

It is necessary to have at least semi-quantitative criteria of the effectiveness of the synchronizing mechanism. Two possible measures are obvious. If the integrating mechanism acts more powerfully, cells will become more perfectly synchronized and the recorded potential waves will be more regular and more constantly repeated. Further, with more powerful synchronization it should be more difficult to disrupt the waves by desynchronizing agents. Actually, the two criteria show good parallelism. A rhythm whose waves vary in amplitude over a range of 50 per cent, for example, is disrupted by addition of potassium ions at 0.003 M concentration. A more constant rhythm, showing a variability of only 10 per cent, is not disturbed by such a potassium concentration, but requires over three times as great a one to break it up.

On the basis of such evidence, the following conditions have been found to augment the effectiveness of cell integration: increased temperature, increased calcium, decreased potassium, decreased sodium, decreased inflow of nerve impulses (if non-homogeneous), increased constant currents (whether produced by external polarization or by injury potentials in the tissue itself), and finally the drugs nicotine and caffeine. It is perhaps premature to analyze the action of these various conditions in terms of the separate parts of an electrically integrated mechanism, but it is at least worth noting that probably every one of them increases membrane polarization and so the driving potential. Others also enhance the irritability of inactive neurones to applied potentials; and some, for example decreased intercellular ions or applied directional electrical fields, may also influence the spread of currents through the tissue mass. Since caffeine has a particularly striking influence on brain potentials, it has proved possible to continue the analysis with its aid.

When a solution of this drug (up to 0.5 per cent) is applied to the isolated brain, the usual six a second rhythm is increased in amplitude and regularity and slowed 10 to 20 per cent in the course of two or three minutes. (Fig. 6. In the figure shown, a very regular high-frequency rhythm already present before caffeine was also increased by it.) In another minute or two, characteristic caffeine

waves appear and they may continue to develop for several minutes more while the six a second rhythm decreases and finally disappears. The caffeine waves are very strong, usually 0.5 to 1.0 millivolt in amplitude and each lasting some 0.2 second, and they are repeated with great regularity—in the early stages at intervals of several seconds, later as frequently as two a second, and towards the end of the drug action perhaps only two a minute. The individual wave is most commonly diphasic but is often much more complex, and a particular profile will be repeated for some time but may

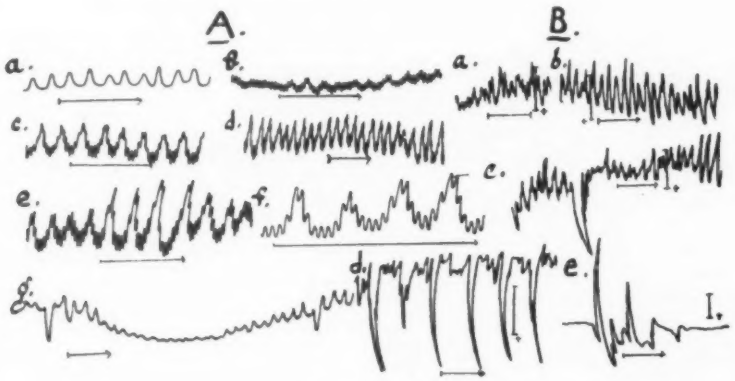


FIG. 6.—Caffeine.

A. Olfactory bulb; a. normal; b. 1 min. later, showing an aberrant spontaneous shift in frequency; c. 0.5 per cent caffeine for 1 min.; d. same, slower speed; e. 2 min. after c.; f. same, faster speed; g. 1 min. after e.
B. a. Normal; b. 0.5 per cent caffeine for 1 min.; c. 1 min. after b.; d. 2 min. after c.; e. 2 min. after d. + indicates direction of surface positivity.

then gradually shift in the course of successive waves into a very different one (Fig. 7).

The majority of these primary waves are diphasic because they spread along the brain surface. By shifting the positions of one pair, or better of two pairs, of pick-up electrodes, the point of origin and direction and speed of spread can be determined. The waves originate most commonly near the junction of the olfactory bulb and the cerebral hemisphere proper and spread caudally over the convexity of the hemisphere. There is little or no travel directly from side to side and the waves in the two hemispheres are usually not simultaneous (Fig. 8*f*, *g*).

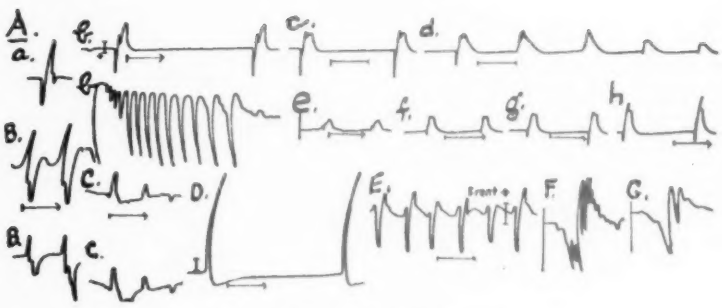


FIG. 7.—Types of caffeine waves.

A. Cerebral hemisphere; a. 5 min. in 0.5 per cent caffeine; b. 45 sec. (double record from front and back—lower line—of hemisphere); c. 75 sec.; d. 85 sec.; e. 95 sec.; f. 100 sec.; g. 105 sec.; h. 115 sec. after a.

B, C, D, E, F, G. Other brains in 0.5 per cent caffeine for 3 min. Double leads in B and C.

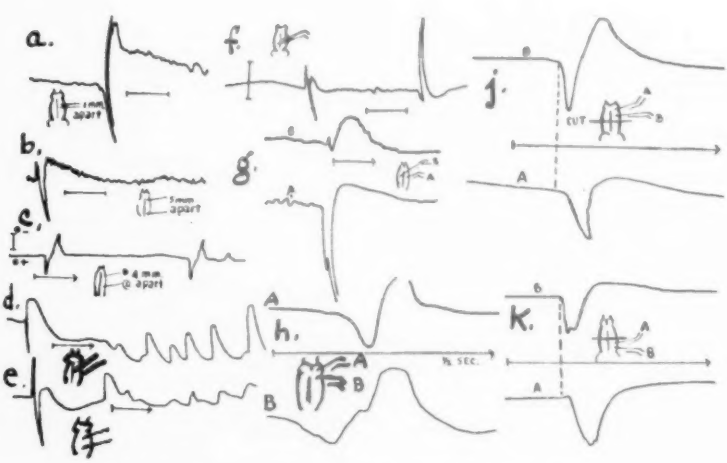


FIG. 8.—Origin and direction of spread of caffeine waves.

a. Electrodes 1 mm. apart on hemisphere; b. 5 mm. apart; c. 4 mm. apart (another brain), note the temporal separation of the phases of the diphasic wave as compared to a; d. 3 mm. apart on hemisphere in a lateral direction, note sharp spike, indicating that wave arrives at both points almost simultaneously; e. 3 mm. apart in longitudinal direction; f. one electrode on each hemisphere; g. simultaneous recording from both hemispheres, two channels; h. electrodes as diagrammed, wave starts in anterior end of hemisphere before olfactory bulb; j. double record from front piece of cut brain; k. same from hind piece. Note in j and k wave travels from occipital pole to cut and from cut to bulb.

The large potential arises from a flat base line and is initially surface positive. Commonly it is followed by a train of satellite potentials, as many as 20, which start at lower amplitude and higher rate, as high as 10 a second, and recur in any one train at progressively longer intervals and with greater voltage (Fig. 9). Even when the initial wave travels caudally, the following ones may appear simultaneously (at least within two milliseconds) over the whole hemisphere or may even travel rostrally from the occipital pole. The interaction of these various waves sometimes gives a potential pattern with a dome and spike strongly reminiscent of that in petit mal epilepsy and it is of interest that these separate components in an epileptic potential have also been reported to move across the brain independently of one another (Case and

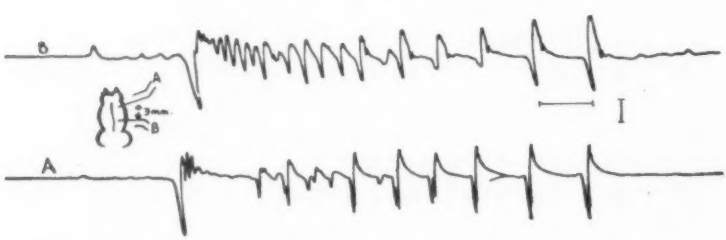


FIG. 9.—Typical caffeine train recorded from two points simultaneously, showing initial (surface positive) wave spreading from front to back of hemisphere.

Bucy, 1938).* The initial waves usually travel but sometimes even these appear simultaneously over the brain. They then begin with a surface negative potential instead of a surface positive one.

The waves spread normally at the rate of 6 cm. a second at 21° C. (Fig. 10), but their velocity readily changes with temperature. Thus, at 15° they are slowed to 4 cm. a second; at 26°, velocity may increase discontinuously to 22 cm. a second. Since the point of origin can shift relatively to the two pairs of lead-off electrodes, apparent velocities are not entirely reliable. A third channel would be necessary to eliminate this source of error. Increase of temperature also

* The common picture of epilepsy is of successive cortical waves spreading further and further from a center of origin until the motor region is reached and motor discharges begin. If the character of the aura indicates the epileptogenic focus, the interval between aura and fit might show some relation to the spatial separation of the corresponding cortical areas.

increases the amplitude, approximately doubling it for 10 degrees, and shortens the duration, from 0.3 second at 15° to 0.1 at 26°.

When polarizing currents are applied to the caffeinized brain, they produce effects entirely analogous to those in the untreated one. A descending current increases the amplitude, frequency, and rate of travel of the caffeine waves while it is flowing, inhibits them subsequently; and an ascending current has the reverse action (Fig. 11). Nicotine does not prevent these effects of the polarizing current, which cannot, therefore, depend upon redirected nerve impulses.

The slowly spreading large waves are strongly reminiscent of those set up in the mammalian cortex by locally driving one

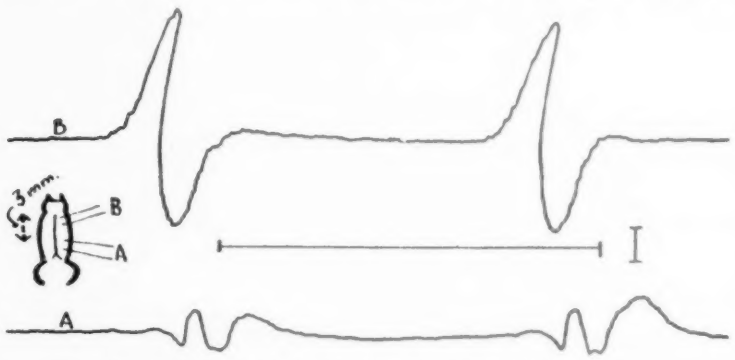


FIG. 10.—Spreading caffeine waves recorded at higher speed. Velocity 3 cm. a sec. in this case.

region (as with electrical stimuli, Adrian, 1936; convulsant drugs; or in Jacksonian epilepsy), and which presumably depend upon propagation along nerve pathways. It has been possible to show, however, that these caffeine waves in the isolated frog brain can continue to spread when neural transmission is excluded.

In the first place, they continue in the usual fashion after the application of sufficient nicotine to block synaptic transmission. Caffeine does not prevent this synaptic lock, for potentials evoked in the optic lobe by visual stimulation are promptly abolished by nicotine even when caffeine is present (Fig. 12). Not all synapses in the brain can be tested directly, however, so the objection can be raised that the particular neurone chains needed for the spread have not been blocked by nicotine. This assumption is gratuitous

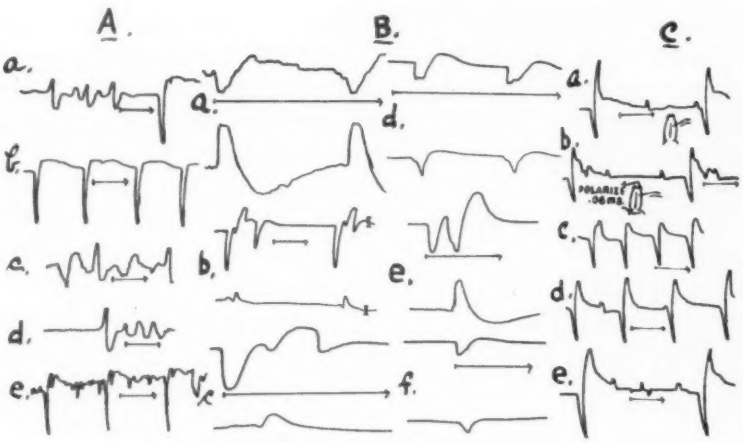


FIG. 11.—Polarization on caffeine waves.

A. Cerebral hemisphere; a. in 0.5 per cent caffeine 3 min.; b. during descending current of 0.06 mA; c. current off; d. during ascending current; e. current off.

B. Another brain; a. caffeine waves, simultaneously recorded from two points; b. same during descending current; c. same, faster speed, note the decreased velocity of spread; d. current off, waves practically simultaneous; e. during descending current again; f. current off.

C. Another brain; a. usual caffeine waves; b. during descending current; c. current just off; d. 15 sec. after c; e. 40 sec. after c.

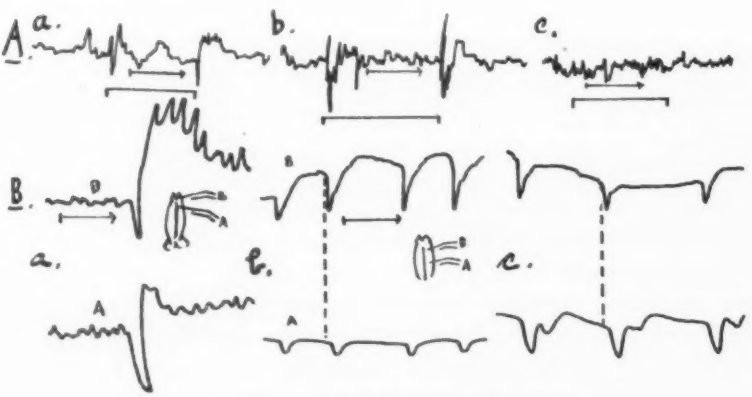


FIG. 12.—Nicotine and caffeine.

A. Optic lobe *in situ*, light flashed into contralateral eye, "on" and "off" as indicated. a. Normal on and off responses; b. in 1 per cent caffeine for 5 min.; c. 1 per cent caffeine and 0.5 per cent nicotine for 3 min. Note nicotine abolishes the optic responses.

B. Two channel records, isolated brain. a. In 0.5 per cent caffeine for 3 min.; b. in 0.5 per cent caffeine and 0.5 per cent nicotine for 5 min.; c. 1 min. after b. Note nicotine does not abolish caffeine waves.

and unlikely, since all types of synapses tested were blocked by the drug, but it makes further evidence desirable.

The most unequivocal method of stopping neural transmission is that of anatomically interrupting the pathways. If the caffeine waves should be able to pass across a cut, neural transmission could not be responsible. Fortunately this simple experiment was successful. When the entire brain is transected with a rapid stroke of a very sharp razor blade and the two halves allowed to reappose, caffeine waves often continue to travel longitudinally across the cut (Fig. 13). The rate of spread is unaffected, and it is simple to show, by the appropriate placing of both sets of electrodes on either half, that the waves are not arising at the injured surfaces but come to the cut in one half and go from it in the other (Fig. 8*j*, *k*). Further, when the two halves are separated by a

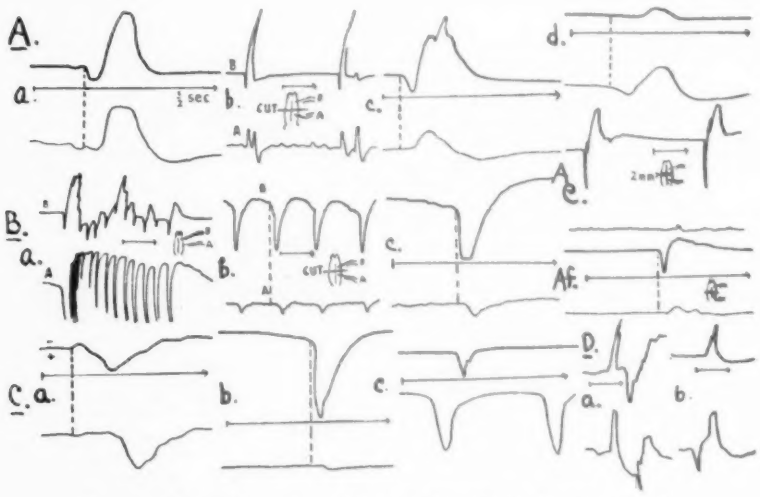


FIG. 13.—Interaction across cut. Two channel simultaneous records.

A. a. Caffeine waves before cut; b. after complete transection across hemispheres, recording from each piece as shown; c. same later, showing front piece originating wave; d. still later, showing hind piece originating wave; e. pieces separated by 2 mm. (connected by Ringer's), note lack of interaction; f. pieces reapposed.

B. Another brain; a. caffeine waves before cut; b. after complete transection; c. same, faster speed, note that wave clearly spreads with finite velocity across cut.

C. Another brain; a. caffeine waves before cut; b. after complete transection between the two sets of electrodes; c. later, independent action of the two halves, showing non-interaction of recording systems.

D. Another brain; a. caffeine waves before cut; b. after cut.

millimeter or less, even though the gap is filled with Ringer's solution, travel is completely blocked, to be restored by reapproximation.

Many types of control have convinced us that we are dealing with a true spread of electrical activity from cell to cell across a cut and that artefactual interaction between leads and recording channels is excluded. Fig. 14 illustrates, for example, records from the two halves which show certain waves which are simultaneous in both (marked by arrows) while other waves are completely independent in the two portions. Any electrical interaction in the system would produce simultaneous potentials in all cases, and the fact that some are independent proves that the coincident ones are really present together in both halves. Further, no appa-

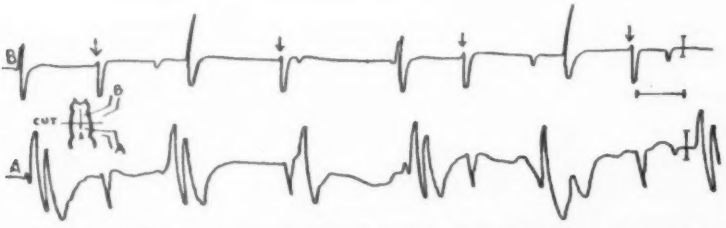


FIG. 14.—Caffeine waves, two channel records, on either side of a complete transection. Note that certain waves (arrows) are clearly interdependent in both halves, while others are independent.

ratus interaction could account for the time lag in the responses of one of the halves, with sometimes the front and sometimes the hind one preceding the other by a time interval appropriate to the known velocity of spread and the distance separating the electrodes.

A final experiment to demonstrate the distance action of the electrical fields eliminates even the local injury produced by a cut. One isolated brain is caffeinized and placed in Ringer's solution with a second uncaffeinized brain near it. Electrical activity in the hemisphere of the untreated brain is practically nil when the two are not in contact. On moving the untreated specimen so that its olfactory bulbs are in contact with the caffeinized hemisphere, however, the previously inactive brain shows a strong potential rhythm. This promptly disappears when the two brains are again separated (Fig. 15).

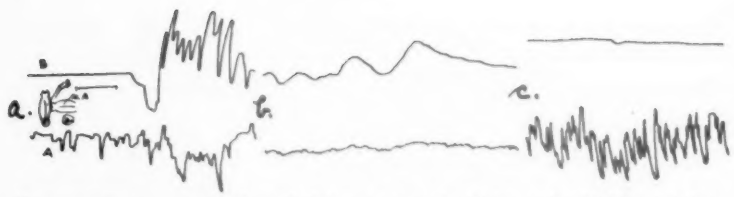


FIG. 15.—Brain 1 previously soaked in 0.5 per cent caffeine and exhibiting caffeine waves; Brain 2, untreated. Simultaneous recording as in diagram.

a. Brain 2 touched to Brain 1, as shown; b. brains separated by 1 mm.; c. as in a. Note activity in hemisphere of Brain 2 only when in contact with 1.

DISCUSSION.

The evidence thus seems conclusive that electrical potentials and currents generated by some neural units are able to spread through brain tissue, independently of the anatomical conducting pathways, and to engage and modify the activity of other neurones. Many observations have recently entered the literature indicating analogous actions of currents, of which only three instances will be mentioned. Hill has reported (1939) that Nitella cells lying, without contact with one another, in a solution which sets up spontaneous electrical beats, soon come to discharge in unison. Erlanger (1939) has demonstrated that the nerve impulse in a single fibre can jump a region of block at least two internodes long, the potential which reached the proximal end of the block being able to excite the fibre beyond it. Eccles has reviewed (1939) several papers dealing with electrotonic potentials in the spinal cord and their influence on reflex behavior.

It seems probable, indeed, that here is a mechanism of widespread importance which has gone largely unrecognized. Actually, it is possible to account in considerable detail for the phenomena observed in the frog's brain, in terms of a concrete picture of electrical spread which has much in common with the orthodox schema accepted for transmission of the nerve impulse itself. This will be more fully elaborated elsewhere, but may be outlined briefly in concluding this paper. (Fig. 16.)

The neurones in the frog hemisphere are arranged as a fairly uniform sheet of cells. If each unit has, in addition to an internal-external polarization across its surface membrane, another directed along its axis from the outer dendritic pole to the inner axonic one, such a cell sheet would behave much like a single polarized surface. (A polarization gradient along the neurone was suggested

some years ago, Gerard, 1932, p. 547, and has recently been developed independently by Gesell, 1939.) We may assume further that the dendritic end is negative to the axone and that increased negativity of the latter is associated with action and the discharge of nerve impulses. This is in harmony with the fact that the large initial potential swing of a spreading caffeine wave is surface positive and with the directional effects of polarization. Referred to the outside, these effects are in the reverse direction to those in nerve, just as the polarization assumed for the cell sheet is the reverse of that of the membrane in nerve.

The sheet of cell batteries oriented in parallel serves to maintain polarization across this layer despite the continuous leakage of

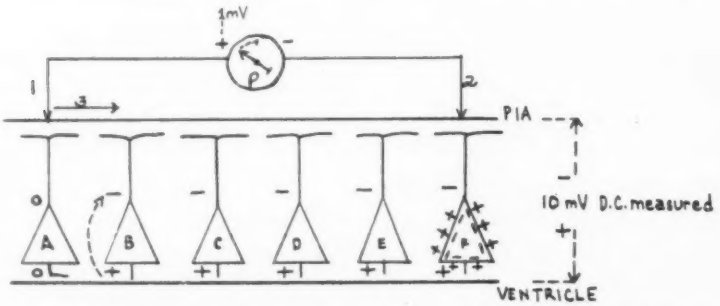


FIG. 16.—Schema of cell layer in frog's cerebral hemisphere. See text. A caffeine wave is spreading as indicated by arrow 3 and has just discharged the neuraxial polarization of cell A. Cell B is discharging. The amplifier shows a pial surface positive wave of 1 mV. The usual interior-exterior polarization of the neurone surface is shown for cell F.

current through the intercellular conducting fluid between cells. When, however, one or several of these cell batteries in a small region lose their oriented polarization—that is, when the axone hillock becomes less positive and so approaches the potential of the dendritic end—then a considerable increase of leakage between the two surfaces of the sheet can occur in this vicinity. Adjacent cells are thus allowed to depolarize and the effect can spread.

Though the morphological substratum is entirely different from that of the nerve fibre and the propagation of nerve impulses as such is in no way involved, it will be recognized that this picture of interneuronal spread is fundamentally identical with that of the nerve impulse. The rate and effectiveness of such field propagation are dependent on the factors previously considered, again analogous to those controlling impulse velocity in the nerve fibre.

When the units are highly irritable and are manifesting a spontaneous potential rhythm, the slightest field set up by a pace-maker group suffices to catch other units at essentially the same potential phase and so bring about synchronous beats. When irritability is less or spontaneous potentials minimum, potential changes move more slowly, for each unit requires time to respond to the change produced by activity of its predecessor, and the potential wave spreads gradually over the whole cell plate.

The spreading caffeine waves fall into this second class. It remains to account for the fact that they do not spread symmetrically but rather in a longitudinal orientation along the hemisphere. This would necessarily occur if still another potential gradient on a grosser scale existed between different portions of the brain, for the waves would naturally tend to travel along pre-existing potential gradients. That such potentials do exist between olfactory bulb and occipital pole has been established by direct measurement with a potentiometer and unpolarizable electrode system. The olfactory bulbs are two millivolts or more negative to the occipital pole. Further, the above hypothesis calls for a maintained potential between the pial and the ventricular surfaces of the hemisphere with the latter positive. When tested, a potential was found with the predicted orientation and 8 to 10 millivolts in magnitude. This voltage gradient perpendicular to the cell plate is increased by caffeine, and abolished by cocaine or ether, the latter reversibly.

These voltage studies will be reported in detail elsewhere but it is now established that there exist in the frog's cerebrum three levels of polarization: from inside to outside across the cell membrane; from dendrite to axone hillock of the single neurone, perpendicular to the brain surface; and from rostral to caudal pole of the entire hemisphere, along large numbers of separate neurones. These considerable and regularly occurring potential fields seem to be of great significance in the normal and pathological functioning of the brain. Perhaps the spread of epileptic activity about the brain depends in part on such electric currents; and the unexplained phenomena of electronarcosis (Silver, 1939) begin to take on meaning in terms of polarization gradients. We hope to elucidate further the mechanisms through which these potentials control action, and the metabolic gradients which must be responsible for their maintenance.

Summary.

Brain potentials can be used within limits as an index of the action of cerebral neurones. The recorded waves are the envelope of the beats of many single neurones and their profile depends on the shape of the unit potential and the temporal relation of the many units.

The isolated frog brain, with relatively few and similar neurones which can be subjected to a wide variety of experimental conditions, is especially favorable for studying the control of potentials. Mammalian brain *in situ,* so far as similar experiments have been made, shows like behavior.

The olfactory bulb of the isolated brain gives an electrical beat, at six per second, larger and more regular than before removal from the frog. Under varied conditions, the frequency, still regular, may be shifted from one to 50 per second and the single wave from a sine to many highly skewed forms constantly repeated. Such patterns indicate strong unison of the many cells and so reproduce the potential profile of the unit.

The unit beat is controlled by the metabolism of the cell, oxidative or glycolytic energy supplying the driving force. It is also controlled by a trip mechanism, presumably the cell membrane, which determines the frequency of oscillation at constant drive. Amplitude and frequency vary inversely as the trip mechanism is altered, together as metabolism is changed.

Control of cell potentials mainly through metabolism is considered in the cases of: oxygen lack, narcosis, cyanide, glucose lack, insulin, B_1 avitaminosis, iodoacetate, and nicotine poisoning. The action and interaction of these and subsequent agents is presented.

Control mainly through the trip mechanism is considered for: altered osmotic pressure, sodium, potassium, calcium, magnesium, and hydrogen ions; for neural stimulation and, in more detail, for polarization by constant currents.

Unification of the beats of the many units into synchronous or simultaneous patterns must depend on the propagation of nerve impulses or on electric fields and currents. The mechanism of unification is analyzed and tests for its effectiveness are described. The following conditions favor unison: high temperature and calcium, low potassium and sodium, diminution in afferent nerve im-

pulses (in patterns), increased polarization (or injury potentials), the drugs nicotine and caffeine.

Further analysis is made with caffeine, which leads to large repeated waves which may travel along the hemisphere. These are described in detail; and also the control of their size, shape, frequency, speed, and direction of travel by temperature and polarization.

The spreading caffeine waves are not stopped by nicotine, which blocks synaptic conduction; they cross a complete transection of the hemisphere when the two halves are in good apposition and so in electrical but not neural continuity; and a caffeinized brain can augment the activity of an abutting normal one.

It is concluded that, besides neural coordination of separate neurons, a strictly electrical mechanism can regulate nerve cell beats and cause a spreading activation. This may prove of considerable importance in the interpretation of normal activity, electronarcosis, and of the massed "convulsive" action of the cortex in epilepsy.

A theoretical explanation of the mechanism of spread is presented. It demands the existence in brain of maintained polarization gradients in certain directions. These have been found present as predicted.

BIBLIOGRAPHY.

Adrian, E. D.: The spread of activity in the cerebral cortex. J. Physiol., 88: 127-161, 1936.

Baker, Z., and Himwich, H. E.: Effect of nicotine on the oxidation of glucose. Am. J. Physiol., 123: 6, 1938.

Blake, H., Gerard, R. W., and Kleitman, N.: Factors influencing brain potentials during sleep. J. Neurophysiol., 2: 48-60, 1939.

Case, J. T., and Bucy, P. C.: Localization of cerebral lesions by electroencephalography. J. Neurophysiol., 1: 245-261, 1938.

Chang, T. H., and Gerard, R. W.: Studies on nerve metabolism. VII. The rôle of lactic acid, Am. J. Physiol., 104: 291-297, 1933.

Dubner, H., and Gerard, R. W.: Factors controlling brain potentials in the cat. J. Neurophysiol., 2: 142-152, 1939.

Eccles, J. C.: The spinal cord and reflex action. Annual Rev. Physiol., 1: 363-384, 1939.

Erlanger, J.: The initiation of impulses in axons. J. Neurophysiol., 2: 370-379, 1939.

Gerard, R. W.: Nerve metabolism. Physiol. Rev., 1932, 12: 469-592, 1932.

Gerard, R. W.: Brain waves. Scientific Monthly, 44: 48-56, 1937.

Gerard, R. W., Marshall, W. H., and Saul, L. J.: Electrical activity of the cat's brain. Arch. Neurol. Psychiatr., 34: 675-738, Chicago, 1936.

Gesell, R.: Unpublished manuscript (in press), 1939.

Gibbs, F. A., Lennox, W. G., and Gibbs, E. L.: The electroencephalogram in diagnosis and in localization of epileptic seizures. Arch. Neurol. Psychiat., 36: 1225-1250, Chicago, 1936.

Hill, S. E.: Contribution to local circuit theory. Am. J. Physiol., 127: 118, 1939.

Himwich, H. E., Hadidian, Z., Fazekas, J. F., and Hoagland, H.: Cerebral metabolism and electrical activity during insulin hypoglycemia in man. Am. J. Physiol., 125: 578-585, 1939.

Holmes, E. G.: Carbohydrates of crab nerve. Biochem. J., 23: 1182-1186, 1929.

Holmes, E. G.: The relation between carbohydrate metabolism and the function of the grey matter of the central nervous system. Biochem. J., 27: 523-535, 1933.

Libet, B., and Gerard, R. W.: Automaticity of central neurones after nicotine block of synapses. Proc. Soc. Exp. Biol., N. Y., 38: 886-888, 1938.

Libet, B., and Gerard, R. W.: Control of the potential rhythm of the isolated frog brain. J. Neurophysiol., 2: 153-169, 1939.

Olmsted, J. M. D.: Effect of insulin on cold blooded vertebrates kept at different temperatures, Am. J. Physiol., 69: 137-141, 1924.

Schweitzer, A., and Wright, S.: Action of nicotine on the spinal cord. J. Physiol., 94: 136-147, 1938.

Silver, M. L.: Electrical anesthesia in rats. Proc. Soc. Exp. Biol., N. Y., 41: 650-651, 1939.

Sugar, O., and Gerard, R. W.: Anoxia and brain potentials. J. Neurophysiol., 1: 558-572, 1938.

Tokaji, E., and Gerard, R. W.: Avitaminosis B_1 and pigeon brain potentials. Proc. Soc. Exp. Biol., N. Y., 41: 653-658, 1939.

Winterstein, H.: Der Stoffwechsel des Zentralnervensystems. Handb. d. norm. u. path. Physiol., 9: 514-611, 1929.

DISCUSSION.

DR. WILDER PENFIELD (Montreal, Canada).—Dr. Gerard showed a very interesting fact that in the isolated brain the waves which he records electroencephalographically were communicated to another adjacent brain which was simply abutted to the first brain. As I understood, he assumed that this might indicate that an epileptic discharge occurs not along neural pathways but diffusely through the brain also. I should like to ask whether this isolated brain can be made to produce by electrical stimulation anything that resembles an epileptic seizure, as determined electrically.

Also it seems to me he is assuming what Dr. Lennox also assumed, that the essential characteristic of epileptic seizure is the electrical wave, which seems to me is open to much discussion.

DR. RALPH W. GERARD (Chicago, Ill.).—I am grateful to Dr. Penfield for raising the question, because I failed to make perfectly clear some of those relationships in my hurry. The experiment of abutting a second brain against a first is just the reverse of the other experiment of cutting an active brain in two, and in both cases the spread does occur.

Now can one directly, by stimulation with electrical agents, produce potentials which are equivalent to the epileptic ones? If you recall one of the slides in which the brain was polarized descendingly, there was present in that record exactly the same sort of convulsive potentials as obtained with some of the chemical agents. I think the answer is "yes" in so far as one dare equate potential changes in an isolated frog brain with epileptic seizures that attack human brains. As far as the potential indices go, they parallel one another.

That brings me to the last part of your question, namely, to what extent dare one use the electrical index as a measure of what is happening? It certainly is not the whole story, but I think it is more than the noise that goes with the running of the engine. If the sort of evidence I have presented turns out to apply in the human case, then these potentials are more than just an index of the activity of the cells but are themselves part of the mechanism of excitation and integration and therefore of spread. Indeed had there been opportunity, it would have been possible to show rather a close parallelism between the spread of activity from one cell to another along an oriented mass of cells in the brain cortex and the spread of activity from one active region in a nerve fiber to another inactive one. In both cases, electrical circuits are the means of transmission of excitation from the active to the inactive element. And in both cases spread is not by conduction along a nerve fiber, because a nerve impulse is developed by electrical means within a nerve fiber and itself constitutes a propagated disturbance.

The changes that underlie these effects, the things that initiate the increased activity, the increased spread, the increased electrical potentials, are not immediately electrical but of the chemical character that has been emphasized by all the speakers so far, of which carbon dioxide at the moment looks particularly impressive. It is true that all the agents which produce these convulsive potentials do likewise increase the metabolic rate of these cells. Caffeine, for example, greatly increases respiration, proper types of polarization increase respiration, potassium increases respiration. There is a discrepancy with carbon dioxide, which in large amounts tends to decrease the metabolism, but that is carrying us beyond your question.

INSULIN SHOCK TREATMENT OF SCHIZOPHRENIA.

By FANNY G. HALPERN, M. D., SHANGHAI.*

The differences of opinion about the insulin shock treatment of schizophrenia and its results are such that any new contribution to this issue appears justified, the more so as observations herein recorded were made on patients who differ from those observed in western countries in respect to their race and who live under different climatic and social conditions.

Since other observations in the sphere of neuro-psychiatry point to the fact that there exist some differences between the Chinese and western people with regard to the symptoms of the disease and reactions to therapy, it is of interest to determine if any differences also exist with regard to the insulin shock therapy.

We are fully aware of the deficiencies and errors attached to statistics calculated from a comparatively small number of patients and we realize that the judgment of the results of every method of treatment in psychiatry as well as the classification of cases is bound to meet with considerable difficulties. Nevertheless we give the results of our observations by means of statistical tables for convenience in discussion though we do not lay claim to their mathematical value.

As to the technic of the treatment, we followed Sakel's method in general.

We begin the treatment by introducing a preparatory phase (Sakel's phase I) in which gradually increasing doses of insulin are given. The dose which is followed by an apparent physical reaction and mental shock is maintained. The shock phase (phase II) consists of production of series of hypoglycemic shocks. The rest phase (phase III) is the insulin free interval between series of severe shocks. We finish the treatment with the "polarization phase" (phase IV) during which smaller doses of insulin are given sufficient to produce hypoglycemia without coma in which the patient is left for five hours daily.

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At the beginning of the shock, we consider the state in which the patient's contact with the outside world ceases and his response to tactile and acoustic stimuli disappears. The optimal dose proved to be that amount of insulin which causes somnolence and occasionally signs of motor irritation within the second hour after the injection and the beginning of the shock in about three hours. We try to obtain the deep shock with loss of corneal reflexes and positive Babinski's sign in the fourth till the fifth hour after the injection. According to the individual case, we leave the patient 20 minutes to 1 hour in deep coma thus extending the total hypoglycemia from 4½ to 5 hours. During the hypoglycemia we carefully watch the physical condition of the patient—regularly registering the pulse rate, blood pressure and the appearance of various neurological symptoms.

We must emphasize that we frequently deviate from the above routine method in individual cases for each of which the optimal procedure must be found.

In general, we terminate the hypoglycemia in stuporous or severely inhibited cases during the stage of the hypoglycemic excitement but observed that though many patients reacted favorably to this procedure, others respond only to deep comas. Psychomotor excitement could be removed by slight shocks or even by under-coma doses of insulin in quite a number of patients. In other cases, however, though less numerous, hypoglycemia without coma had an obviously exciting effect on the patient and the excitement could be removed by deep comas in the course of further treatment. In paranoiac types as well as in schizophrenics with depressive-hypochondriac features we usually use comas of long duration, while in cases of "acute schizophrenia" less deep and long shocks proved to be sufficient.

The physical symptoms of hypoglycemia in the Chinese are similar to those observed in occidental people.

The mental condition varied in individual patients throughout the treatment. In a few cases, improvement was obvious during the preparatory phase, in some others a sudden improvement occurred after a few shocks, but mostly the remission only very gradually developed in the course of the shock therapy. In most cases the fading of the secondary schizophrenic symptoms such as hallucinations, delusions and disturbances of psychomotility occurred much sooner than the improvement of the schizophrenic changes of personality which usually could not be removed at all.

For the great majority of cases we could not confirm Sakel's observation—*i. e.*, that at the beginning of treatment the patient

first becomes lucid during the hypoglycemia while in later stages of the therapy the periods of lucidity occur after termination of coma. Although in single patients, mostly catatonias, a sudden brightening of the psychosis set in during the hypoglycemia, in the overwhelming majority of patients improvement began so gradually that neither the time of its beginning nor the manner of its development could be indicated. The clinical picture of the psychosis was mostly changing without any regularity throughout the treatment so that hours or days of lucidity were frequently followed by psychotic phases of variable duration.

We also had no opportunity to observe among our patients Sakel's so-called "activated psychosis" or "reversal of the reaction." According to Sakel, improved patients who already are non-psychotic after the termination of the shock in the afternoon, become psychotic again during the hypoglycemia. This so-called "activated psychosis" is supposed to reproduce the picture and course of the individual psychosis in an image-like and concise form. The treatment should be continued until the activated psychosis during the hypoglycemia disappears. We could observe, of course, that some of the patients who were already clear in the afternoon, regularly became confused during the hypoglycemia and the confusion was frequently connected with wild psychomotor excitement, some mannerisms and grimacing. This "hypoglycemic psychosis," however, was not a reproduction of the original schizophrenia and happened to occur throughout the whole treatment, disregarding the duration of the therapy and the mental condition of the patient when not in hypoglycemia. We incline to consider this hypoglycemic confusion as a sign of intoxication by high insulin doses, not being able to find out if this type of reaction to overdoses of insulin has any relation to the schizophrenia at all.

Among the patients with remissions in whom the shock therapy was terminated in spite of the occurrence of the hypoglycemic psychosis, no more relapses of schizophrenia occurred than among others who did not react in such a manner to insulin.

From November, 1936, until March 1, 1939, there were treated at our division 76 patients by the insulin shock method, 11 of whom were foreigners, the others Chinese. Six of these patients are still under treatment at the present writing while in 13 cases the treatment was prematurely discontinued. In eight patients the treatment

was terminated on account of their physical unfitness for shocks such as repeated cardio-vascular collapses, persistent vomiting, laryngospasms, etc., while in the other five patients the therapy was discontinued for non-medical reasons. One patient died of pneumonia and circulatory disturbances a few days after the last coma.

All the patients who proved to be unfit for the shock therapy as well as the patient who died happened to be Chinese. Consequently, we come to the figure of 17 per cent of Chinese schizophrenics who are unfit for the insulin shock therapy. As all our patients had a thorough physical examination before starting treatment and cases with any contra-indication were excluded beforehand, the percentage of physically unfit Chinese for severe hypoglycemia seems to be very high. It is striking, however, that most of the complications which rendered shock therapy impossible, occurred during the hot and humid climatic period in Shanghai. In five of the eight apparently "unfit" patients, the shock therapy had to be discontinued during this climatic phase and the fatality occurred during an especially hot time in August. Other patients, also, Chinese as well as foreigners, who otherwise could stand shocks without complications, frequently reacted with severe circulatory disturbances to hypoglycemia during the season in question, and in a few cases the shock therapy had to be postponed until more favorable weather.

In the face of the comparatively scarce material, no definite conclusion can be drawn as to the dependence of physical reactions to hypoglycemia on climatic or racial factors but besides questionable racial influences, the climate also seems to have some influence upon the hypoglycemic shock.

The results of the shock treatment are tabulated as follows: *

* The following statistics are calculated from those 56 patients only who were able to have the full course of shock therapy. This figure includes 10 foreigners, while the remaining 46 patients were Chinese.

+++ = full remission, *i. e.*, mental restitutio ad integrum.

++ = good remission, *i. e.*, insight for the disease, social usefulness, abolition of delusions and hallucinations but with schizophrenic changes of personality.

+ = poor remission, *i. e.*, obvious but incomplete improvement with a remainder of schizophrenic symptoms and restriction of working power.

− = unchanged or very slightly improved.

TABLE II.*

RESULTS OF SHOCK THERAPY.

	Results.								
	+++		++		+		−		
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	No. of cases.
Total cases	20	35.75	7	12.41	11	19.48	18	32.14	56
Duration of disease under one year.	18	75.0	4	20.0	0.	0.	3	5.0	25
Duration of disease over one year...	2	6.45	3	9.68	11	35.48	15	48.39	31

From Table II it becomes evident that the prognosis is to a great extent dependent on duration of the disease. Patients with duration of disease under a year show remarkably more full remissions than chronic cases while the number of negative results is greater in the group of chronic schizophrenia.

TABLE III.

RESULTS OF TREATMENT IN ACCORDANCE TO THE DURATION OF THE DISEASE WITHIN THE GROUP OF CHRONIC CASES.

	Results.								
	+++		++		+		−		
Duration of disease.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Total cases.
1-2 years	2	18.18	2	18.18	3	27.27	4	36.37	11
2-3 years	..	....	1	11.11	4	44.44	4	44.44	9
Over 3 years....	..	....	..	....	4	36.37	7	63.63	11
Total	2		3		11		15		31

Table III shows that with increasing duration of disease in the chronic cases, the chances for remission decrease markedly.

With regard to the ratio between the duration of the treatment and the duration of the disease, the opinion given in the first report

* Table I, containing details of individual cases, is not printed for the sake of brevity. The table contains personal data about every patient as to sex, age, type of schizophrenia, duration of the disease, duration of therapy, result of treatment, shock dose at the beginning and in the later stages of treatment as well as remarks concerning individual cases. Any information about a particular case will be gladly given on inquiry.

A preliminary report on the insulin shock treatment at our division was given to the Biennial Conference of the Chinese Medical Association, Shanghai, April 1937 (Halpern, Chinese Medical Journal, 52: 541-548, Nov. 1937). Some differences between the first and the present report are due to a longer period of observation and a greater number of patients.

must be revised. It first appeared as if in patients with a shorter duration of the disease, the remission occurred sooner than in chronic cases. From a greater number of patients we learned that the average duration of the treatment is not very markedly different in both groups of patients. The shock therapy lasted in cases with duration of disease below one year from 8 to 18 weeks with the average of 9 weeks, while in cases with duration of disease above one year from 6 to 19 weeks with an average of 11 weeks. Taking into consideration only those cases who had a remission, we have an average duration of treatment of 8 weeks in acute cases and an average of 9½ weeks in chronic cases.

These figures, however, have a limited value only because the duration of treatment expressed in terms of weeks is also dependent on the duration of the preparatory phase which varies in individual cases as well as on the duration of the polarization phase which depends on the subjective judgment of the physician as to when the therapy has to be terminated. The way of expressing the duration of treatment by means of the number of shocks, as suggested by Frostig, has the advantage of not being dependent on phases I, III and IV. We have to consider, however, that hypoglycemias without shocks are also a part of the therapy. Consequently, the statement of the duration of treatment by both methods used together, appears to be most satisfactory.

According to Frostig, shock therapy which does not cause improvement after 50 shocks should be terminated as being without prospect for remission. From our observations we must agree with this as most of our patients who had no remission after about 45 to 50 shocks did not improve with the continuation of the shock therapy. The time required for 50 shocks is approximately 12 to 15 weeks and with a few exceptions we usually do not exceed this number of shocks. The longer periods of treatment were given in a few special cases who had a comparatively small number of comas.

In some patients full remission occurred a few weeks to months after the termination of the shock therapy. As all these patients (cases 5, 9, 27) were schizophrenics with a short duration of disease at the beginning of treatment, the question arises whether the late remission has any relation to the therapy at all. The past treatment might, of course, have activated certain tendencies for remission which otherwise would have stayed latent but it is also possible that

the late recovery is virtually a spontaneous remission which would have occurred without the shock therapy.

Similar considerations seem to be justified in several other acute cases in which the shock therapy which lasted a few months finally led to a remission. This consideration seems all the more justified in Chinese patients, as according to observations among Chinese more spontaneous remissions occur than among Westerners.*

As example may be cited case 9, female, age 23, first attack of paranoiac schizophrenia, duration of disease 3 months. The patient did not respond to the shock therapy for 2½ months at all. With continuation of treatment for 4 months a slight improvement appeared which turned into full remission 6 months after completion of the shock therapy. Consequently, the patient finally recovered 13 months after the onset of her psychosis. In this and similar cases which were under observation it is very questionable whether the remission is due to the insulin shock therapy at all.

However, there are other cases in which the remission was obviously connected with the treatment, *e. g.*, in patients in whom symptoms suddenly disappeared after a few shocks only. In chronic cases, however, with changes of personality we cannot consider such sudden improvement as real recovery for only the striking secondary symptoms of the disease disappeared while the essential change of character remained.

Discussing the dependence of the remission on the therapy, one case must be mentioned in whom each improvement of the mental condition was connected with the shock therapy while each interruption of treatment was followed by a regression of the psychosis.

Case 4, female, age 28, paranoiac schizophrenia with acute origin, duration of the disease four months, final result of the shock therapy +++. After 14 days of treatment (only a few comas) full remission occurred. On interruption of the treatment for physical unfitness, the mental condition gradually became worse. Mild shocks were induced again and led to full remission after six weeks of treatment. During phase IV psychotic symptoms appeared again and could finally be removed by a few more deep shocks.

* In some patients cardiazol shock therapy was introduced after an unsuccessful insulin shock treatment or vice versa. In a few cases improvement occurred thereafter. In two of these patients (case 37 and 51) the improvement was evidently due to the supplementary therapy. In the others however, who suffered from the first attack of schizophrenia of short duration, it appears doubtful that the final remission is a result of the subsequent shock therapy since both treatments together covered such a long period that the probability of remission during the later treatment was greater than at the time of the first shock therapy.

From this case we learn that the treatment should not be interrupted too early and that the shocks should be sufficiently deep and of sufficient duration.

The results of treatment in the various types of schizophrenia are indicated in Table IV.

The patients are divided in five groups. Contrary to the previous publication the group of "acute schizophrenia" is omitted in the present statistics for the sake of better comparison with other statistics which do not gather such cases into a special group. In Table IV such cases are tabulated according to the dominating symptoms into the particular group of paranoiac schizophrenia or catatonia.

TABLE IV.

RESULTS ACCORDING TO THE TYPE OF SCHIZOPHRENIA.

	Results.								
	+++		++		+		−		
Diagnosis.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Total cases.
Paranoiac type ..	13	43.33	6	20.0	4	13.33	7	23.33	30
Catatonia	6	35.29	2	11.75	4	23.50	5	29.47	17
Hebephremia ...	..	....	..	....	3	60.0	2	40.0	5
Schizophrenia simplex	..	....	..	....	..	....	3	100.0	3
Circular type ...	..	....	..	....	..	....	1	100.0	1
Total	19		8		11		18		56

Paranoiac types seem to react better to shock therapy than catatonia, the former group showing more full and good remissions and fewer unimproved cases than the latter. Consequently, that type of schizophrenia has best prospects for improvement which otherwise presents least inclination to spontaneous remissions and can be little influenced by other forms of treatment.

The number of our patients is too small for further subdivisions within the particular groups, *viz.*, in regard to the duration of schizophrenia, the form of its onset (acute or gradual) and course as well as according to the clinical symptoms such as productivity, spontaneity, stupor, excitement, etc. No reliable statistics can be given for each form from these points of view but it must be emphasized that the above mentioned qualities play a considerable part in the effects of the shock treatment upon the individual patient. Thus, for example, productive forms with numerous delusions and

hallucinations usually respond better to the therapy than the unproductive and inhibited autistic types; and excited catatonias better than stuporous cases.

Table V demonstrates the results of therapy in patients with duration of disease under one year with regard to the manner of origin of the disease.

It is evident that chances for remission are better in cases of schizophrenia which begin in an acute way than in patients with a slowly developing psychosis. Although acute schizophrenics also show most inclination to spontaneous remissions, yet the percentage of full remissions in treated patients is higher than in the non-treated ones.

TABLE V.

RESULTS ACCORDING TO THE ONSET OF THE DISEASE IN THE CASES WITH DURATION OF THE DISEASE UNDER ONE YEAR.

	Results.								
	+++		++		+		−		
Origin of the disease.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Total cases.
Acute	12	85.71	1	7.14	..	....	1	7.14	14
Gradual	6	54.55	3	27.18	..	....	2	18.09	11

In patients with a long duration of disease the results of shock therapy are not evidently dependent on the form of the onset and therefore such cases are not included in Table V.

The number of patients with schizophrenia simplex, hebephrenia as well as circular types of dementia præcox who were given insulin shock therapy is too small to justify conclusions. At any rate, it seems that in hebephrenia and dementia simplex the prospect for remission is rather poor.

In the former publication it was stated as an impression that schizophrenics with depressive-hypochondriac features do not respond well to shock therapy, similarly to the greater resistance of hypochondriac-melancholic types of general paralysis towards malaria therapy. With a greater number of patients and longer observation, the previously given opinion must be revised as quite a number of such cases (cases 9, 14, 27, 40) had a full or good remission.

The maximal-minimal and average doses necessary to produce coma are evident from Table VI.

There are, of course, great individual variations of the shock dose, but remarkable is the fact that, on the average, in advanced cases larger doses are needed to produce coma than in recent cases and that with the progress of treatment, patients respond with shocks to smaller insulin doses than at the beginning.

Of the 56 patients the shock dose decreased during the therapy in 46 patients, *i. e.*, in 82 per cent of the cases.* In one patient (case 22), no coma could be obtained by as much as 560 insulin units, in four patients (cases 15, 26, 38, 41) the dose remained unchanged throughout the treatment while in five others it was raised (cases 32, 34, 50, 59, 62).

From these observations it is evident that the organism becomes more sensitive to insulin throughout the shock therapy. The interpretation of this phenomenon is difficult. This may be due to an

TABLE VI.

MAXIMAL AND MINIMAL AND AVERAGE VALUES OF THE SHOCK DOSE EXPRESSED IN INSULIN UNITS.

Duration of the disease.	Initial.	Advanced stage of the treatment.
Under one year	30-260	20-240
	100	70
Over one year	40-300	5-300
	125	77

inhibition of the regulatory adrenalin secretion or it may be that the schizophrenic process itself, being connected with endocrine-vegetative disturbances, makes the nerve cell less irritable to insulin. H. O. Pfister and others consider schizophrenia as a systemic disease of the regulatory vegetative nervous apparatus which affects the whole organism including the cerebral cortex and they prove various disturbances of balance in the vegetative system which correspond to the duration of schizophrenia.

From this point of view we may conclude that the schizophrenic process itself is able to change the irritability of the ganglia cells towards insulin by decreasing their sensitivity. Consequently, initial cases react, on the average, to a smaller dose than chronic cases with a more advanced schizophrenic process. As far as some accidental observations permit any statement, it appears that non-

* The fact that at the beginning of the therapy larger doses are usually needed in order to produce shock than in later stages was already pointed out by us at the C. M. Conference, April 1937.

schizophrenic people, even diabetics, react more intensely to insulin than schizophrenics, lower doses than those used in schizophrenics presenting for them serious physical danger.

Proceeding along this line of thought we may imagine that with the improvement of the balance of the regulatory vegetative apparatus during shock therapy, the irritability of the nerve cell towards insulin becomes greater. Also in cases with no obvious clinical improvement, the influence of the shock therapy upon the neuro-vegetative system cannot be excluded.

It is interesting to state that of the nine patients in whom the dose was increased or remained unchanged during the therapy, only two had a good remission while the remaining patients did not show any essential improvement. A remarkable attitude with regard to the shock dose is illustrated in Table VII concerning case 4,

TABLE VII.

Case 4.	Insulin shock dose.
First treatment of 2 weeks' duration, remission (+++)	50
First relapse—3 weeks after interruption of the shock therapy	..
Second treatment of six weeks' duration:	
Beginning	95
Remission (+++)	45
Second relapse (at the end of phase IV).	
Third treatment of six weeks' duration:	
Beginning	65
Remission (+++)	50

mentioned previously in another connection, in whom every improvement was accompanied by decrease of the shock dose while every time the condition became worse the coma dose became higher.

In this particular case we have the impression that the coma dose would be an indicator for the mental condition of the patient. However, the observation on this patient was not made in other cases who presented similar fluctuations of the shock dose without corresponding changes of the mental condition. In some cases it was found that the shock dose must be raised with resumption of treatment after an occasional interruption but without regression of the mental condition during the rest phase.

At the time of writing, *i. e.*, after two years' observation, relapses have occurred in five of our patients who showed full remission, 25 per cent of all full remissions. The occurrence of the relapse varied from 4 to 12 months after termination of the therapy. In

the majority of these patients the repetition of shock therapy did not give as good results as the first treatment and only one, a catatonia which relapsed after four months, became well again.

SUMMARY.

The method used and general observations made during insulin shock treatment of schizophrenia are discussed.

From November, 1936, to March, 1939, 76 cases of schizophrenia were treated; 56 of them finished the complete course of therapy, the remaining 20 are either still taking the treatment or the treatment was interrupted for various reasons.

A higher percentage of patients in China compared with those in western countries proved to be unfit for insulin shock treatment. Observations point more to climatic than to racial differences.

From this study, the prospect for remission appears to be dependent on the duration of the disease, the type and course of schizophrenia as well as the form of the onset, cases with an acute onset showing more inclination for remission than those with a gradually progressive course.

The duration of the treatment until remission is not dependent on the duration of the disease.

Remissions in certain cases with protracted treatment or late remissions after shock therapy cannot be positively considered as the result of the shock therapy.

The shock dose shows lower average values in cases with shorter duration of the disease than in chronic cases. The dose required for a shock usually decreases during the course of treatment.

In one particular case, the shock dose fluctuated in accordance with the mental condition of the patient.

Relapses occurred in 5 of the 20 cases with full remission after the shock therapy.

I wish to express my thanks to Drs. John C. Tseng and Richard Wang for their valuable assistance.

BIBLIOGRAPHY.

Berglas und Susic: Psych.-neur. Wschr. No. 48, 1936.
Bersot, H.: Ann. Méd.-psychol., 94, II, 1936.
Dussik, K. T.: Allg. Zeitschr. Psych., 107, 1938.

Dussik, K., und Sakel, M.: Ztschr. ges. Neur. und Psych., 155, 1936.
Ewald, G.: Med. Welt, 1937, 899-902.
Frostig, T. P.: Schweiz. Arch. Neur. 39, erg. H., 1937, 153-157.
Hager: Psych. Neur. Wschr. 1937, No. 39.
Harris, M. M., Blalock, J. R., and Horwitz, W. A.: Psych. Quart., 12, 1938.
Heilbrunn, G.: Schweiz. Med. Wschr., 1936, II.
Hesse, H.: Fortschr. Ther., H. 5, 1937.
Horwitz, W. A., Blalock, J. R., Harris M. M.: Psych. Quart., 12, 1938.
James, G. W. B.: The Lancet, 1101-1104, 1937.
Katzenellenbogen, S., Harms, H. E., and Clark, D. A.: Arch. of Neur., 39, 1938.
Kuppers: Dsch. Med. Wschr., I, 1937.
Meduna, L. v.: Psych. Neur. Wschr., No. 27, 1935.
Meduna, L. v.: Die Konvulsionstherapie der Schizophrenie, Halle A. S., 1937.
Meduna, L. v.: J. Nerv. Dis., 87, 1938.
Müller: Schweiz Med. Wschr., 36, No. 39.
Müller: Schweiz Arch. Neur., 39, Erg. Heft, 9-21, 1937.
Pfister, H. O.: Schweiz Arch. Neur. 39, Erg. H., 77-83, 1937.
Pullar, Strecker: Schweiz Arch. Neur., 39, Erg. H., 178-179, 1937.
Russell, L. W.: J. Ment. Sci., 89, 1938.
Sakel, M.: Neue Behandlungsmethode der Schizophrenie, Vienna, 1935.
Sakel, M.: Zeit. Neur., 149, 1930; 152, 1933.
Sakel, M.: Wien. Med. Wschr. No. 45 1934 to No. 7, 1935.
Salm, H.: Allgem. Zschr. Psychiatr., 109, 1938.
Savitt, R. A.: Psychiatr. Quart. 12, 1938.
Vander Veer, A. H., and H. H. Reese: Am. J. Psychiatr., 95, 1938.
Winkelman, N. W.: Americ. J. Psych., 95, 1938.
Wortis, J.: J. Nerv. Dis. 85, 1936.
Wortis, J., Bowman, K., Orenstein, L., Rosenbaum, J.: Am. J. Psychiat., 94, 1937.

CLINICAL OBSERVATIONS IN THE INSULIN TREATMENT OF SCHIZOPHRENIA.

Preliminary Report.*

PART I.

The Symptomatology and Therapeutic Factors of the Insulin Effect.

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The treatment of schizophrenia by the use of insulin was introduced in Vienna by Manfred Sakel. Experiments with large amounts of insulin had been made earlier but the investigators failed to comprehend fully the importance of their observations and therefore did not draw adequate conclusions. It is greatly to the credit of Sakel to have clearly understood the meaning of psychic improvements which accidentally occurred while he was treating drug addicts with insulin and, in spite of many difficulties, to have courageously persisted in his experiments and the application of his findings to the treatment of schizophrenia.

Early surveys of the results of this treatment have shown that it is as a rule very effective when applied in cases of relatively short duration but that the rates of improvement and recovery vary greatly in different treatment centers. For example, several careful investigators report complete recovery in approximately 70 per cent of their cases while others report no more than 20 per cent to 30 per cent of improvement. The writer had the opportunity to observe the administration of the insulin treatment in many hospitals in Europe and has attempted to analyze a number of communications reporting low rates of improvement. Both personal observation and critical analyses of results led him to conclude that the wide variations in the effectiveness of this therapy are due for

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Detailed results will be published later. Co-workers in testing and recording were: J. Kister, A. Manasson, W. Matecki, Ch. S. Muller, I. Persyko.

the most part to differences in its application. These differences may have been chiefly due to the fact that the directions of the Vienna Clinic were not as clear as they might have been. No clear directions for accurate insulin dosage were formulated, the depth of shock was not defined and no information was given concerning the optimum duration of the insulin effect. Whatever the reasons, the following factors were poorly understood at the time the writer began his investigations:

1. The quantity of insulin required to produce the desired effect.
2. The depth of the insulin effect.
3. The duration of the insulin effect.

Because it is impossible to investigate three unknown factors simultaneously, it was decided to set the duration of the insulin effect arbitrarily at five hours. The time factor was chosen because it was the only one of the three which could be set as "constant" in advance. The premises upon which the writer based his investigations were therefore:

(a) The dose and the depth as variable factors.

(b) The time as a constant factor.

(c) Improvement to be used as measure of the efficacy of the method. No changes in method to be made in those cases in which improvement did not occur.

Having set the duration of the insulin effect as the constant, the writer had the task of determining the relationship between the size of the dose and the depth of the insulin effect. Inasmuch as the depth is a progressive function it was necessary to evolve a system of recording by which not only the quality of the symptoms but also their time relationship could be recorded.

The investigation was made in the Psychiatric Institute "Zofiowka," Warsaw, Poland, and repeated at the Harlem Valley State Hospital, Wingdale, New York. The daily course of every treatment was recorded on special charts, complete examinations being made every 15 minutes. The results are based upon the observation of 195 cases representing a total of 6587 insulin days. Part I deals with the general results and conclusions only.

Symptomatology of the Insulin Effect.

The symptomatology of the insulin effect is undoubtedly related to the functions of the central and autonomic nervous systems.

The dose necessary to produce cerebral manifestations varies widely, from a minimum of 17 units to a maximum of 520 units in the cases here reported. The following description of the symptoms is based upon statistical evaluation of 2000 treatment records. It is possible that analysis of the entire material will result in slightly different findings, but the difference will not be great.

The symptoms may be divided into five clinical groups:

(a) Disturbances of consciousness.
(b) Disturbances of exteroceptive and interoceptive sensitivity.
(c) Disturbances of motor functions.
(d) Disturbances of the autonomic nervous system.
(e) Psychotic disturbances.

A. DISTURBANCES OF CONSCIOUSNESS.

The first evidence of disturbed consciousness is usually somnolence, which appears in 97.2 per cent of all shocks. Speech is slow, perception and thinking become more difficult, orientation is poor and the threshold of psychic reactions is heightened. The patient complains of being hot or cold and there are disturbances of the body scheme. Macropsia, micropsia, metamorphopsia, dizziness, and sensations of floating may occur. Spatial changes have been described (Benedek). Apraxia, agnosia or aphasia may be observed. The clinical picture of this phase varies greatly. Angyal believes that its variations depend chiefly upon individual cortical predilection for the effect of insulin; that effects may originate either in the frontal, parietal or occipital regions. Clouding develops, as a rule, slowly and progresses wave-like as its depth increases. Speech becomes slurred, sometimes bradylallic; perseveration, echolalia, and echopraxia are often noted. In some cases inarticulate screaming and groaning continue throughout the stuporous state. Often stimulation by noise or light, addressing, or shaking the patient causes a brief clearing of consciousness. The patient becomes oriented and speech is more distinct but with the next wave of clouding falls asleep again or becomes restless. Finally no contact can be established.

Loss of consciousness is recorded when intentional acts either of speech or voluntary motion are no longer observed. The patient can be aroused from this state neither by touch nor by call, although

he may react to pin-prick or shaking and may even murmur a few words.

This lighter degree of unconsciousness will be called *sopor* (stupor). It is characterized by the following:

(a) Stronger stimuli evoke defensive reactions such as screaming, flight reflexes, and defensive movements. Very strong stimuli may awaken the patient. Sometimes myoclonoid twitchings tonic spasms and torsion spasms cause awakening also.

(b) Either the sympathetic syndrome prevails or the autonomic system remains in balance.

(c) Motor symptoms are usually marked.

The deeper stage of unconsciousness, *the coma*, is characterized by the following:

(a) The patient cannot be awakened by external or internal stimuli.

(b) The balance of the autonomic nervous system shifts distinctly toward parasympathetic preponderance.

(c) The muscles are hypotonic. If motor spasms occur they are tonic and small in amplitude.

(d) The lid and corneal reflexes become sluggish or disappear (Cline and Rossman).

B. DISTURBANCES OF EXTEROCEPTIVE AND INTEROCEPTIVE SENSITIVITY.

The changes in the state of consciousness are associated with distinct alteration of sensitivity to internal and external stimuli. During the period which immediately follows the loss of consciousness the exteroceptive sensitivity increases remarkably. A flash of light, the stroke of a hammer; every external stimulus evokes a quick response. In the early phase the responses assume the character of flight reactions. Later they are in the nature of myoclonoid twitchings and athetoid movements. With increasing depth the sensitivity to light and noise decreases, while simultaneous increase in the sensitivity of the muscles is observed. Passive movements of the extremities or stroking of the muscles instantly increases the tonus or produces a tonic spasm. Finally when the coma becomes very deep no responses can be elicited. The disappearance of the corneal reflex indicates that the biologic border of reversible changes has been reached.

C. DISTURBANCES OF MOTOR FUNCTIONS.

Motor symptoms begin with *hypotonia* as early as the end of the first hour after injection of insulin. Simultaneously weakness of

muscles is observed both subjectively and objectively. Hypotonia was noted in 97.4 per cent of all shocks. Hypotonic episodes occur in every phase of the insulin effect. Early hypotonia is sometimes accompanied by a fine though intense tremor.

Almost simultaneously with sopor *motor restlessness* sets in. It may begin independently or develop from the drowsy semi-purposeful movements of the presoporous somnolence. Sometimes psychotic excitement states precede the development of sopor. Motor restlessness consists of fragments of motions resembling parts of a purposeful action. They may be identified with ideomotor movements (Kretschmer) or pseudospontaneous movements (A. Flach). The patient attempts to get up, beats the air with his hands, carries out complicated movements of body and limbs. The movements are often hypometric or hypermetric, sometimes slow, often quick. A pin-prick causes first flight reflexes then movements of wiping in the region of the stimulus. Sometimes there is an uncoordinated storm of movements with pronounced motives of flight or defense, and erotic fragments are also observed.

Primitive movements crystallize out of motor restlessness most often but they may appear independently as the first sign of motor disturbance. They are observed in 71.5 per cent of shocks. We have seen "schnauzkrampf," biting of the lips, kissing and sucking movements, protruding of the tongue, licking of the lips, scratching, thumb sucking, rubbing the face and trotting and climbing motions. Primitive movements are always accompanied by forced graspings, which may occur earlier and persist longer than do the primitive movements themselves.

Approximately at the time when consciousness is lost *clonic twitchings* appear. They are observed in 79.2 per cent of shocks. Almost always unilateral at their inception they begin in the muscles of the face, especially at the angles of the mouth and in the muscles of the chin, spreading to the muscles of the eyebrows, lids and lips. Only later are clonic twitchings of the upper and lower extremities observed.

The above described group of symptoms is followed by another more definite and distinct syndrome which can easily be identified as the hyperkinetic-hypotonic syndrome (Lothmar and others). Along with clonic twitchings electric-like symmetrical twitchings appear. They are seen in the face, in the muscles of the neck and

extremities, sometimes developing gradually from the clonic twitchings. Occasionally they spread gradually over the entire body and become increasingly violent. The effect of these movements on the joints is very distinct and the amplitude is comparatively large. They resemble the paramyoclonus multiplex (Friedreich). The writer prefers to designate them as *myoclonoid twitchings.* Sometimes they occur *en masse,* their rapidity and intensity increasing until a generalized attack seizes the entire body. Genuine myoclonias (not producing motion) are also observed, but rarely.

Choreiform movements appear quite frequently while *athetoid movements* are less often seen. The latter slow down the rapidity of the primitive movements and change them to grotesque postures by causing hyperflexion of the various joints. During the athetoid spasms the foot and great toe are dorsally flexed. The stimulus for the Babinski sign elicits atonic dorsal flexion of the toe. The hemiballisms involve the upper extremities and show their typical character, as if something were thrown.

Early torsion spasms start with sympathetic spasms of the lids and one-sided movements of the extended or flexed trunk which begin mostly in the shoulder girdle. The twisting effect is a very considerable one, the movements being executed on a vertical or a horizontal axis. *Rigor* can sometimes be detected during the myoclonoid twitchings. Later the muscles are wax-like and plastic.

Individual elements of the hyperkinetic-hypotonic syndrome occur almost always but they dominate the picture only in 38 per cent of all shocks. As the shock proceeds to a greater depth the hyperkinetic phenomena become less frequent. Spontaneous movements become rare and their amplitude decreases. The muscles are still hypotonic but striking the skin or flexing the joints may produce *increased tonus,* which occurs either rapidly or slowly. Rapid increase of the tonus is often accompanied by a *rough cloniform tremor* caused by rapid relaxation of agonists and antagonists alternately. Sometimes these shaking movements seize the entire body. The muscles are tense and the body assumes various positions which depend upon the different static reflexes released by suppression of the higher centers.

As time passes the increased tonus takes on the character of *tonic spasms.* During the tonic spasms the arms are frequently

pressed close to the torso and flexed at the elbow, and the hands are clenched in tetanic position, sometimes in a fist with thumbs turned inward. The lower extremities are usually extended, often with pes varus and the toes in claw-like contraction. The spasm may last some seconds or several minutes and is followed by an interval of hypotonia during which there is a continuous tendency toward spastic reactions. Any attempt to flex the limbs is likely to cause repetition of the spasm. Tonic spasms were observed in 82.6 per cent of all shocks.

The tonic spasms are followed by *late torsion spasms,* the leading symptoms of which are conjugate deviation of the eyes with slow tense turning movements. In *crossed torsion spasms* the head and eyes move in one direction while the torso and extremities turn oppositely. Torsion spasms were observed in 25 per cent of all shocks.

Tonic spasms have the effect of lessening the depth of the unconsciousness, sometimes to the extent of complete awakening. These spasms are always accompanied by disturbances of the autonomic nervous system.

As time passes the motor picture undergoes further changes. The tonic spasms still remain dominant but they now exhibit mechanisms pointing to lower phylogenetic levels. The distribution of the tonus becomes quite different. The extremities are fully extended, the extensor muscles being strongly contracted while the flexors are soft and hypotonic. The arms show internal rotation, the forearms are stretched out, with hands in pronation and fists clenched. There is opisthotonus and the lower extremities and feet are fully extended. These *tonic extensor spasms* are to be identified with the extensor spasms of animals made decerebrate by cutting the brain stem caudal to the nucleus ruber. These spasms are at first sporadic but later they appear more frequently. A single spasm may last from several seconds to two minutes. As in the case of other tonic spasms they are accompanied by definite disturbances of the autonomic nervous system. Tonic-extensor spasms were observed in 34.1 per cent of all shocks.

The posture reflexes (Magnus and De Kleyn) were frequently seen. Upon turning the head passively to one side we observe a decrease of tension on the other side, while the limbs toward which the head has been turned show an increased tonus.

The tonic extensor spasms are the final form of the motor symptoms which may be immediately reversed when sugar is administered. They indicate the biologic border of the motor group, just as the disappearance of the corneal reflex does for the decreasing sensitivity.

Clinically demonstrable pareses were sporadically observed. Where the facial nerve was involved only the oral branches were affected. The oculomotor and abducens nerves were quite frequently weakened, occasionally during the presoporous period, causing disagreeable diplopia. The pareses usually disappeared with the awakening but in some cases they lasted some hours longer.

The *reflex signs of pyramidal tract involvement* appear comparatively late, usually at about the time when increasing tonus is noted. First abdominal reflexes disappear. The sign which we observed most frequently was that of Babinski, which was noted in 89.4 per cent of all shocks. The intensity of the pyramidal signs varies widely from time to time. At first they are markedly tonic and easily obtained. The reflex zone is extended and slight stimulation of any part of it produces instantly a pathological reflex action. As the depth increases the intensity of pyramidal signs diminishes. At first the Oppenheim disappears, then the Rossolimo. The Babinski, however, persists much longer. During the tonic and the tonic-extensor spasms no pyramidal signs can be elicited but all, or many of them, return during the hypotonic interval between spasms.

Tendon and periosteal reflexes are at first strongly active, later weaker. Toward the end of the shock the many reflexes tend to disappear. Partial absence of reflexes was observed in 3.2 per cent of all shocks. *Defensive movements,* which must be considered as closely related to changes in exteroceptive sensitivity, disappear with increasing depth of unconsciousness. Their absence was observed in 84.9 per cent of all shocks. *Lid and corneal reflexes* are the last of the reflex signs to disappear.

D. DISTURBANCES OF THE AUTONOMIC SYSTEM.

The first and most constant sign of autonomic disturbance, noted in 99.1 per cent of all shocks, is *perspiration.* It is observed to occur intermittently and to be thick and viscid during the early stages of shock, and thin and watery later. The maximum rate of

excretion occurs during the coma, while its intermittent character is best observed during the period of tonic spasms. *Salivary secretion* was increased in 90.1 per cent of all shocks.

Respiration is deep and regular during the first two hours of the insulin effect, usually becoming irregular with the beginning of the hyperkinetic syndrome. During the tonic spasms expiratory dyspnoea is often observed and is probably the result of bronchial and glottal spasm. This is accompanied by violent compensatory action of the accessory muscles of respiration. In contrast with the effect of motor spasms, respiratory difficulties cause a marked deepening of the shock. During the deep coma respiration is depressed, shallow and slow.

The *temperature* decreases throughout the shock. In 2.7 per cent of our shocks it was found to be as low at 86 degrees Fahrenheit when taken at the axilla. The decrease of body temperature is not closely related to changes in the clinical picture.

The cardio-vascular system is more profoundly affected than any other and appears to be influenced by a number of factors, some of which are not related directly to changes in the brain. The *pulse* varies widely in both rate and volume and few consistencies are discernible. However some of the variations are found to occur regularly and are understandable when related to the following syndromes of the autonomic nervous system:

(a) Immediately after consciousness is lost and simultaneously with the beginning of the hyperkinetic syndrome the face becomes flushed, the lids open widely and exophthalmia is observed. The pupils are dilated but react fairly well to light, the pulse varies between 120 and 140, and exteroceptive sensitivity is at its climax. This *sympathetic syndrome* occurs for the most part during the period in which hyperkinetic phenomena are observed. When tonus increases the pulse rate becomes slower and the balance between the sympathetic and parasympathetic branches of the autonomic nervous system is reestablished.

(b) With every tonic spasm or tonic extensor spasm the pupils dilate and do not react to light, the pulse rate increases to 120 to 180, and respiratory difficulties may cause cyanosis. In the interval between spasms the pupils become smaller, light response returns, and the pulse rate decreases to 60 or less.

(c) As the insulin effect reaches its deepest level the tonic extensor spasm recedes to the background of the clinical picture, the hypotonic intervals becoming longer and the spasms weaker. The face is now pale and sunken, the expression death-like. The pupils gradually become smaller, finally pin-point, and light reaction is lost. The pulse decreases to 60 or less and keeps constantly at this level while respiration is depressed and irregular. Lid and corneal reflexes weaken and finally disappear. This *parasympathetic syndrome* indicates that the greatest depth of the insulin effect consistent with safety has been reached. Beyond this point the changes are likely to be irreversible, and protracted coma with central pulse and respiratory difficulties may be expected.

The parasympathetic syndrome and the changes of interoceptive sensitivity were studied at the Harlem Valley State Hospital together with I. M. Rossman, W. B. Cline, Jr., O. Schwoerer, and G. Fuchs, the details of which will appear in a later publication.

E. PSYCHOTIC DISTURBANCES.

Psychotic disturbances occurred in only 20 per cent of our shocks, and were, as a rule, practically identical with the symptoms of the patients' particular psychoses. The most frequent ones were states of catatonic excitement and hebephrenic or paranoid syndromes. Sometimes anxiety, depressive or euphoric states were observed, and rarely compulsory laughing and crying occurred. Psychotic disturbances appear in the presoporous phase and sometimes during the spontaneous awakening. The "hunger excitement" (Sakel) must be counted among these. It occurs usually later than others, shortly before consciousness is lost. Regaining consciousness in the spontaneous awakening sometimes begins with hunger excitement.

Abnormal Occurrences During the Insulin Effect.

Abnormal occurrences during the insulin effect may be divided roughly into two groups:

(a) There are occurrences which are due to disturbances of various organs of the body, heart failure, vasomotor collapse, spasmus glottidis, choking caused by saliva and mucus, vomiting, etc. These complications are not directly dependent upon the effect of insulin on the brain.

(b) Occurrences which are specific for the insulin effect and are due to a changed response of the brain, caused by the administration of insulin. These are convulsions and protracted shock.

CONVULSIONS.

Seizures occurred only in 2.8 per cent of all shocks. The writer was able to describe three groups of seizure-like occurrences in the course of treatment.

1. *The Myoclonoid Vascular Collapse* (Frostig).—These seizures occur always in the hyperkinetic phase whether this phase appears early or late. The hyperkinetic and the sympathetic syndromes are always strongly marked before the fits develop. These seizures are characterized by the following symptoms:

(a) They are often initiated by forceful, electric-like myoclonoid twitchings, which precede the main fit sometimes by one-half to one hour.

(b) The beginning is mostly marked by conjugate deviation of the eyes and the following torsion of the extremities.

(c) Then the myoclonoid twitchings become more rapid and generalized as the intensity and amplitude of the movements are increased.

(d) Simultaneously with the motor attack the face turns pale, almost ashen gray, mucous membranes are livid, the pulse is slow, frequent and thready.

(e) The seizure can be provoked by exteroceptive irritation. If during the seizure stimuli are used, the violence of the twitchings increases.

(f) The seizure ends sometimes with a tonic spasm and the tetanic posture of the hands.

(g) Biting the tongue is rarely observed. Passing of urine does not occur.

2. *The Epileptoid Seizure.*—These seizures occur while tonic spasms appear.

(a) The start is marked by nystagmic movements of the eyes, conjugate deviation of the eyes. No twitchings precede the fit.

(b) Then a tonic spasm appears. It is often accompanied by tetanic postures of the hands. Opisthotonus is also seen.

(c) After a few seconds the tonic spasm relaxes and is replaced by violent clonic twitchings.

(d) The face is at first bluish-red, the vessels are well filled, but after clonic twitchings cease the face turns gray. The facies show a peculiar, sharp expression. The pupils are dilated and show no reaction.

(e) Biting the tongue is frequent, passing of urine rare, but relatively more frequent than in the type described above.

(f) Exteroceptive stimuli do not provoke the fit and have no influence on the intensity of the symptoms.

3. *The Precipitate Shock.*—Ch. S. Müller observed some seizures which resemble a condensed shock. The cerebral symptoms appear as early as thirty minutes after the injection. In a few minutes the effect may run the whole course of the symptoms as described above and may reach the latest phases of the shock. Such shock, unless it is terminated early may end in a myoclonoid-vascular collapse, in epileptoid seizure or in a protracted shock.

The Protracted Shock.—After the tonic extensor spasms and the parasympathetic syndrome have occurred and the corneal reflexes have disappeared, but no sugar is administered, the protracted shock is to be expected. In this phase of insulin effect the administration of sugar may not immediately reverse the symptoms in which case the patient remains unconscious and new motor symptoms appear while changes in the autonomic nervous system persist. The protracted shock may last a few hours, but often it takes several days and sometimes even several weeks until all symptoms disappear. Although the duration of the different symptoms of the protracted shock varies, the sequence of the symptoms remains unchanged, no matter how long the shock lasts.

After termination the patient remains motionless and comatose. The pulse is slow. No improvement of respiration is seen. Then the previously pin-point pupils become extremely dilated but no light reaction returns. Kussmaul and Cheyne-Stokes type of respiration is observed. Extensor spasms return and they are much more violent than before. The face is bluish and pale, dyspnoea is so severe that it seems as if the patient may die of suffocation. Then the extensor spasms decrease and they are replaced by tonic and torsion spasms. The pulse increases markedly in frequency, respiration becomes gasping, the face is ashen, the mucous membranes are deeply livid. During the intervals the patient is completely exhausted. Respiration becomes shallow and inaudible, the pulse is thready and sometimes imperceptible.

If the patient passes through this phase satisfactorily the tonic torsion spasms become more rare. The symptoms of exhaustion are less marked during the intervals. Choreiform athetoid and myoclonoid twitchings appear. Primitive movements, forced grasping last many hours or days. Then motor restlessness reappears and sometimes a psychotic outburst marks the final stage. A slight disturbance of consciousness may be accompanied by aphasic or

apractic syndromes and sometimes a barely observable paresis of the facial nerve.

The picture is closed by the signs of the amnestic syndromes (Korsakoff), which are always observed, although their intensity may vary.

The Awakening.—Insulin effect is abolished by giving sugar. Sugar solution may be given in different ways. It can be administered orally, if the patient is able to swallow; normally it is administered by nasal tube. When dangerous situations arise glucose may be injected intravenously.

The manner of awakening depends entirely upon the method of sugar administration. If sugar is given by tube the awakening is a slow one and it takes place within a time-period of from ten to thirty minutes. If glucose is injected intravenously, the patient awakens rapidly, often during the time of injection.

The manner of the regression of the symptoms depends primarily on the depth which is seen at the time of termination. For example, if beginning of unconsciousness and fully developed primitive movements are present at this time, these symptoms continue for a little while longer; then apractic and aphasic phenomena may reappear briefly. Gradually, within a few minutes the patient returns to his habitual state. The later the termination, the greater is the sector of regression and the more the scale of retrogressive symptoms is extended. If termination takes place during the deep coma, awakening must run back through the whole gamut of symptoms which were previously developed during the shock. Extensor spasms reappear briefly and are forthwith replaced by tonic spasms. The coordination of the trunk and the extremities is split after a few torsions. Some myoclonoid twitchings follow, and fragments of athetoid and choreiform movements may be detected. Then the picture is dominated for a few minutes by primitive movements. A wave of motor restlessness may close the sequence. During the reappearance of the hyperkinetic symptoms the Babinski reflex usually disappears, but it may sometimes be elicited even in the phase of complete restoration.

The symptoms of the autonomic nervous system regress rapidly. The change in the color of the skin is very impressive. Pallor changes to a healthy flush, the sunken and pallid features become firm and more coordinated. The pulse becomes fuller and respira-

tion is deep and regular. The temperature remains low but returns to normal within one hour. Consciousness returns early, often as early as the hyperkinetic phase, but attempts to speak or to act are disturbed by apractic and aphasic difficulties. Sometimes after termination a short period of deepening shock lasting a few minutes is observed.

The restoration of psychic functions runs its peculiar course. The patients are usually euphoric, rarely depressed or frightened, but always *sthenic*. They impulsively demand food and a dry bed. A surprising syntonic accessibility and an effective openness can be perceived. There is a relationship which is otherwise not to be had with such patients. They answer questions put to them at this time sensibly and are able to take a critical attitude in regard to their delusions and hallucinations. They are soft and pliable, they seek support and help. This phase lasts but a few minutes, at most half an hour. It may end suddenly in a psychotic state or a slow withdrawal into the shell of the autistic attitude. Psychotic symptoms increase in strength and in stability and the patients return to their habitual daytime behavior. The awakening process is ended. A new phase of the insulin day begins, which lasts until the next shock. The writer calls this the "daily constant."

The significance of the *sthenic-syntonic* phase in relationship to the insulin improvement has not been clarified as yet. It is possible that precisely at this point the closed circle of autism is broken. We had the impression as if on each new insulin day a part of these syntonic-sthenic waves flowed into the daily constant, as if these waves would furnish the energetic spring for the new "libidinous occupation of the external world." However, this is merely an impression.

The Order of Symptoms.

A survey of symptoms reveals that all signs of insulin effect as observed by the bedside are due to changes in the central and autonomic nervous system. No signs of major involvement of other organs are observed. Careful recording showed conclusively that the symptoms develop in a definite sequence. Although many difficulties arise in regard to the exact localization, there cannot be any doubt that this order of symptoms is correlated to the phylogenetic structure of the brain. At first those signs appear which

are to be considered as symptoms of suppression of the cortex. They are followed by signs of release of basal ganglia (hyperkinetic syndrome). As time proceeds these signs in their turn become suppressed and give way to symptoms indicating the release of the midbrain (tonic-tetanic syndrome). Finally, the midbrain is suppressed and symptoms due to release of the medulla oblongata (tonic extensor spasms, parasympathetic syndrome) come to the fore of the clinical picture. In the protracted shock suppression of the functions of medullary centers is seen.

In both protracted shock and awakening, the order of symptoms is reversed. At first medullary signs disappear, then successively the function of the midbrain and the basal ganglia is reestablished, finally the cortex regains control of the entire nervous system. In protracted shock every phase is prolonged and may persist for hours or days, while in the awakening the phases are telescoped and last only a few minutes. Therefore, it must be concluded that insulin successively suppresses different levels of the brain in an inverse order to their phylogenetic age; functions are restored in the exact opposite order in which they were previously suppressed.

Physiologic investigations may furnish a suitable explanation for the facts clinically observed. There are experiments which show that the brain is the only organ which does burn carbohydrates almost exclusively (Himwich, Nahum, Lennox) and that the metabolism of the brain depends mostly on the sugar furnished by the blood (McQuinty, Himwich and Nahum). The rapid decrease of the blood sugar and, consequently, of the supply of carbohydrates to the brain causes a decrease of metabolism. Himwich, Bowman, Hadidian and Fazekas showed that the oxygen utilization of the brain diminishes, while insulin effect deepens. Decrease of oxygen utilization indicates decrease of the metabolic rate. Experiments on living brain tissues (Dixon and Meyer and Sugar and Gerard) make it appear likely that the oxygen requirement decreases from the cortex toward the medulla oblongata.

Himwich maintains, therefore, that insulin influences the brain only indirectly and diminishes the carbohydrate supply of the brain by lowering the blood sugar level. It is likely that at first only those brain tissues with a high metabolic requirement will be affected, *i. e.*, cortex and cerebellum. As time passes and the effect of insulin increases, sub-cortical parts of the brain will be suppressed. Finally, the supply of carbohydrates is not sufficient even to maintain the metabolism of the medullary centers.

Time and Dose.

The gradual progress of the symptoms must necessarily draw the attention to the time factor of the insulin effect which is important both biologically and therapeutically, especially in its relationship to the duration of the insulin effect and to the appearance and disappearance of each symptom. Observations of M. Sakel have already shown that the symptoms can appear quickly and develop rapidly on one day, while on another day they are displayed late and progress slowly. In the past the time between the injection of insulin and the termination varied very greatly, therefore no correlations could be revealed. In order to learn about the correlations between dose, time and symptoms, the writer, as mentioned above, arranged the time of insulin effect to be preferably five hours, no matter whether symptoms appeared early or late. Thus it was possible to study the time relations of the different syndromes, either by increasing, maintaining or decreasing the daily dose of insulin. The results indicated that the time of their appearance depends chiefly upon the size of the insulin dose. Increase of the dose speeds the appearance of the symptoms, decrease of the dose slows their progression, but changes in the dose do not influence the sequence of the symptoms.

The exact correlations require especial elaboration. The writer wishes to emphasize only that the direct correlation between dose and time of the symptoms should be considered as a general rule with many exceptions. The more important ones will be analyzed below:

1. The number of insulin units which is necessary to develop the symptoms and to carry them to the medullary phase varies in different individuals. The nature of this individual susceptibility (M. Müller) was not disclosed by the studies of the writer.

2. Regardless of the dose, some time must elapse between the injection and the appearance of the cortical symptoms. Georgi maintains that "the latent time" is due to the required decrease of the sugar level of the blood. The shortest time observed by the writer was twenty minutes, the average is approximately 45 minutes.

3. The same dose of insulin, if given day after day, shows a different power to lower the blood sugar level. The efficiency of the

same insulin dose increases from the first day of treatment until the 12th-16th day. Then decrease of efficiency is observed; sometimes another climax of sensitization occurs when the second part of the treatment is reached. The efficiency of a given insulin dose can be determined exactly by means of the blood sugar index (Frostig, Persyko and Persyko). Thus, in choosing the daily dose, it is necessary to consider not only the individual susceptibility and the depth of the preceding shock, but also the actual change of the patient's sensitization.

Consideration of the Clinical Value of the Symptoms and Their Time Relationship.

The disclosed regular progression of the symptoms makes it possible to use them as gauges of the depth of the shock. The insulin shock may be roughly divided into four groups: (1) the cortical phase, (2) the basal ganglia phase, (3) the midbrain phase, and (4) the medullary phase. Clinical experience has taught that every phase is represented either in full or at least by a few symptoms.

The symptoms possess different clinical value. They must be considered as to their frequency and their distinctness. The most frequent symptom is loss of contact. It appears in 100 per cent of all shocks. But this percentage is an artificial one, since it was assumed that only those charts should be statistically analyzed in which loss of consciousness was entered. However, since loss of consciousness occurs most regularly, it was agreed to use it as standard symptom for differentiating mere hypoglycemia from "shock," therefore, only those insulin effects which involve loss of consciousness were designated as "shock."

Loss of consciousness is recorded when, clinically, there is no reasonable response to touch and call. This is certainly an inaccurate method, but inaccuracy is in the nature of consciousness itself. Consciousness is only defined by one's ability to speak and to act intentionally. The writer does not doubt that there are inner experiences which continue even when no intentional acts are present to display them. Of course, intuition and deduction may imagine and reconstruct the subconscious thoughts and feelings, but they are of no value for the clinical description of the insulin effect. Emphasis has to be placed on the fact that from the very moment in which contact is lost no symptoms are present which adequately indicate any changes in the unconscious as careful analysis of the syndromes "sopor" and

"coma" immediately reveals. Symptoms indicating the "depth" of unconsciousness belong either to the changes of the autonomic nervous system or to the disturbances of the exteroceptive sensitivity or to the symptoms of the motor group.

Once contact is lost motor symptoms are the leading ones. Since these depend almost entirely upon the changes in the brain, they are most reliable.

The play between suppression of the inhibiting centers and release of those previously controlled, with reestablishment of functions on the successive phylogenetic levels, can nowhere be studied as well as in the insulin shock. Motor signs are leading symptoms of the release of the basal ganglia and the midbrain. Tonic extensor spasms may indicate that parts caudal to the red nucleus are already involved.

Among the reflex signs that of Babinski has some clinical value, but it is not reliable. It appears sometimes as early as the hyperkinetic phase, sometimes as late as the tonic extensor spasm. Its disappearance often precedes spontaneous awakening, but no conclusion can be drawn from this fact since it disappears during tonic spasms and tonic extensor spasms. The disappearance of the Babinski during the development of the medullary syndromes indicates an advanced depth of this phase.

The signs of the autonomic nervous system have a special clinical importance during both the basal gangliar and the medullary phases. The course of symptoms of the autonomic nervous system can be roughly divided into four phases: (1) during the cortical phase, equilibrium; (2) during the basal ganglia phase, sympathetic predominance; (3) during the tonic phase, equilibrium; and (4) during the medullary phase; parasympathetic predominance. Consideration of the general progression, as analyzed above, may suggest that sympathetic predominance indicates the release of the hypothalamus from the control of the cortex, while parasympathetic predominance may be due to suppression of the centers rostral to the medulla.

The alteration of sensitivity is a reliable guide for the progression of the insulin effect. It gains a special importance during the medullary phase, since disappearance of the corneal reflex nearly always precedes the irreversible changes. Protracted shock may occur if this symptom is overlooked.

The special value of the medullary phase should also be stressed. The medullary phase consists of three syndromes: the tonic extensor spasms, the parasympathetic syndrome, the decrease of the exteroceptive and interoceptive sensitivity. The syndromes usually appear in the order indicated above. The medullary phase should be considered as the biologic border of the therapeutic shock—at least for the time being.

Since the rapidity of the appearance of the symptoms depends mostly upon the size of the dose, the latter can be adjusted so that the syndromes of the successive phases appear at the desired time. The writer assumed the time of insulin action to be five hours, measured from the moment of the injection to the moment of termination. He attempted to choose the dose so that the medullary symptoms appear in the last half of the fifth hour. There is, of course, no theoretical objection to assume the standard of four or six hours, but the clinical experience of the writer substantiated by several thousands of treatments, led him to the conviction that the time-period of five hours is the most convenient one. The syndromes of the shock are well developed. They follow closely one after another, yet they are not too rapid. It seems to the writer that this time-period is the most appropriate one and furnishes the most uniform results.

The standard time of five hours limits the period of the insulin shock in one direction only. It means that the "shock" is not to be extended beyond this border. The shock, however, may be terminated earlier, if situations occur which threaten either the life or health of the patient. The early termination may take place if seizures or spasms of the glottis, heart failure, vascular collapse or spontaneous awakening occur. A miscalculated dose changes the rate of the appearance of the symptoms. The medullary phase may appear, for example, at the end of the third or the fourth hour. This is, of course, also an indication for an early termination.

Assuming the period of five hours and the appearance of the medullary phase in the end of the fifth hour, the evaluated averages of symptoms according to quality and time order may be presented in the table on page 1186.

Recorded only are symptoms which appear in no less than 15 per cent of all shocks.

TABLE OF SYMPTOMS.*

Hours.	Symptoms.		Localization.
First	Latent Time Somnolence (97.2%) Hypotonia (97.4%)	Perspiration (99.1%) Salivation (90.1%)	Suppression of cortical and cerebellar activity.
Second	Clouded consciousness. Fine tremor. Aphasia. Apraxia. Agnosia. Fall of temperature. Diminished exteroceptive sensitivity.	Perceptual disturbances. Motor excitement. Confusion. Psychotic syndromes.	
Third	Loss of consciousness (100%). Primitive movements (71.5%). Forced graspings (71%). Clonic twitchings (79.2%). Myoclonoid twitchings. Choreiform, Athethoid, Hemibalistic } movements (35.6%). Early torsion spasms.	Motor restlessness. Exophthalmus. Dilated pupils. Light reaction present. Pulse rate increased. Exteroceptive sensitivity increased.	Release of the basal ganglia and hypothalamus.
Fourth	Deepening stupor. Increased tonus. Course tremor. Tonic spasms (82.6%). Tetanic postures. Conjugate deviation of the eyes. Late torsion spasms.	Pupils small or large. Pulse rate increasing or decreasing. Signs of paralysis of the pyramidal functions. Decreased exteroceptive sensitivity. Increased interoceptive sensitivity.	Release of the midbrain. Suppression of pyramidal functions.
Fifth	Tonic extensor spasms (34.1%). Posture reflexes. Depression of tendon reflexes. Deep coma. Diminished sensitivity. Lid and corneal reflexes sluggish. Shallow respiration. No corneal reflexes.	Pin-point pupils. No light reaction. Pulse rate decreased.	Release of the medulla oblongata.

* An abridged and schematic Table of Symptoms of the writer was published in the AMERICAN JOURNAL OF PSYCHIATRY Vol. 96, No. 2, September 1939, pages 314 and 373.

SUMMARY.

The writer has attempted to describe the symptoms of the insulin effect and their relationship to the time and the dose. Striving to apply pathological experiences for clinical purposes, it is necessary to adapt them for pharmacological use. We are accustomed to have pharmacological advice upon the administration of a treatment in figures referring to the doses of the applied drug. But in our special case, individual susceptibility, sensitization and many other factors do not permit us to use the common approach of pharmacological research. Moreover, it is likely that it is not the insulin dose or its effect on the blood sugar level which is important, but rather the effect upon the brain has to be considered as the therapeutic agent. Hence, this effect has to be adjusted pharmacologically for practical use. Norms of treatment should be created, therapeutic factors clarified, and proper indications for the management of the shock evolved. In this way only can misuse of the large insulin doses be met and a planless administration of insulin transformed to an efficient therapeutic instrument.

A complete fulfillment of these postulates belongs for the time being to the realm of scientific dreams. There are many unknown factors: individual susceptibility, sensitization, constitutional and acquired dispositions of the nervous system, individual endocrine balance, differences of metabolic response to hypoglycemia, including the reaction of the liver and the pancreas and of the muscles. No pathology of schizophrenia is known; therefore, no way is open to explain the therapeutic effect of insulin.

Though rules can be established for the application of the treatment, these rules are entirely empirical. They can be tested only in the light of the observed results and for this reason they must be followed very carefully. Analyses of failures have shown that mistaken and apparently meaningless deviations from the rules of the Clinic in Vienna were to blame for many poor results.

In the light of this clinical investigation, the "depth" of the shock can be clearly defined and exactly determined at any stage of the progression of the insulin effect by means of neurological examination. The syndromes actually present not only indicate the degree of progression but also the magnitude of the suppressed area of the brain. While hyperkinetic syndrome is present the

functions of cortex are suppressed. The presence of medullary syndromes, however, indicates that the cortex, basal ganglia and midbrain are inhibited. The expression "greatest depth" is limited now to the presence of the fully developed parasympathetic medullary phase and to the decline of sensitivity (lack of corneal reflex). The shock must be terminated when these signs appear.

The time required for the insulin effect to reach the medullary phase depends upon the size of the dose. If (relatively) large doses are used the entire cycle can be run in three hours. Smaller doses produce the same effect in six or seven hours. Too great doses have the disadvantage of precipitating the shock while too small doses often are followed by spontaneous awakening. Practical experience showed that the most convenient time for a proper development of the shock is five hours. Therefore, it is proposed to choose the dose in such a way that the medullary symptoms appear in the last half of the fifth hour. The dose necessary to produce this effect must be determined daily by analyzing the charts of the preceding insulin day. If the medullary phase appeared too early (*e. g.*, in the fourth hour) the dose must be diminished; if the medullary phase was not reached at all, the dose should be increased. Moreover, the trend of the sensitization must be taken into consideration. If increase of the sensitization is observed, the same dose may produce a deeper shock the next day, while decrease of sensitization requires an additional amount of insulin to cover it in order to produce the same effect as the day before. It must be stressed that the exact management of insulin shock is possible only if the progression of symptoms is properly recorded.

Clinical experience teaches that a great depth should be reached in order to obtain beneficial therapeutic results. The writer had the opportunity to observe that low doses and, consequently, termination in the hyperkinetic phase were often to blame for poor results. It appears, therefore, that shocks in which only the hyperkinetic phase was developed within the five hours of duration of the insulin effect must be considered as therapeutically insufficient. The writer treated 129 cases with the above described method. The medullary phase was reached in only 34.1 per cent. Nevertheless his results in treatment of recent cases are classed among the best (74.4 per cent of recovery). It must be concluded, therefore, that

for recent cases those shocks are therapeutically sufficient in which the tonic phase persisted no less than one hour before termination provided by the standard of five hours.

Cline and Rossman claim relatively better results in old cases, when the medullary phase is carried to the border of the appearance of pin-point pupils and sluggishness or disappearance of the corneal reflex. For the time-being we have, therefore, two different rules, the first applied to recent cases, the other to the old ones. For recent cases it may be sufficient to reach the beginning of the medullary phase shortly before termination, while in old cases the shock has to be carried through the medullary phase to the border of the protracted shock. Such treatment of old cases requires, of course, a great deal of experience.

Conclusions.

1. Observations of symptoms of 6587 insulin treatments recorded on special charts with regard to quality and time of appearance showed that the symptoms succeed each other in a definite and constant order.

2. The progression of symptoms is determined phylogenetically and moves in an order inverse to the phylogenetic age of the layers. At first the activity of the cortex becomes suppressed, releasing the syndromes of the basal ganglia. As time progresses activity of the basal ganglia ceases, giving way to a release of the midbrain. Finally centers in the medulla oblongata are released.

3. If the signs of release of the medulla oblongata have lasted beyond the time in which pin-point pupils were observed and corneal reflex disappeared, a protracted shock may be expected. Release of medulla oblongata has to be, therefore, considered as the biological border of the therapeutic application of the insulin effect.

4. The time of appearance and disappearance of symptoms depends upon the size of the dose. Increase of the dose speeds up, decrease of the dose slows the rate of progression. Sensitization is another factor affecting the time of appearance of the symptoms. The dose does not influence the sequence of the symptoms.

5. In order to adapt the insulin effect on the brain to the therapeutic purposes, the writer proposed a standard time of five hours.

6. The pharmacological shock has to be managed in this way, that medullary symptoms (tonic extensor spasms, parasympathetic syndrome) are to be expected in the last half of the fifth hour.

7. The shock seems to be therapeutically sufficient, if the tonic phase lasted no less than one hour.

I desire to express my gratitude to Dr. J. R. Ross, Superintendent of the Harlem Valley State Hospital for facilitating this work and to Dr. Emmy Metzger, Stony Lodge, Ossining, N. Y., and Dr. W. B. Cline, Jr., for their aid in the preparation of this manuscript. I wish also to thank most heartily Dr. H. E. Himwich, Dr. I. M. Rossman, Dr. W. B. Cline, Dr. O. Schwörer and Dr. G. Fuchs for their cooperation in these investigations.

PSYCHIATRIC FACILITIES IN CINCINNATI AND OHIO.*

The Northwest Territory was officially opened at nearby Marietta in 1789. Cincinnati soon became the Queen City of the West and a gateway to pioneer migrations into what became adjoining states toward the West. There is little record of the care of the mentally ill during the first generation in this vicinity.

In 1821, Dr. Daniel Drake who had founded the Ohio Medical College, now the University of Cincinnati College of Medicine, prevailed upon the state legislature to authorize, "a Commercial Hospital and Lunatic Asylum for the State of Ohio," to be erected at Cincinnati. The separate division for mental patients was not completed until 1827. It did not become a state hospital until 1857. This early institution may be considered the forerunner of both The Cincinnati General Hospital and Longview State Hospital. Dr. Drake was particularly interested in having hospital facilities for the teaching of medical students, an arrangement which has come down to the present-day situation in this city.

In Cincinnati—Institutions.

Psychiatric Department, University of Cincinnati College of Medicine and Cincinnati General Hospital.—When the Ohio Medical College and the Miami Medical College consolidated to form the present medical college of the University of Cincinnati in 1909, Dr. Herman H. Hoppe was named professor in neurology, Dr. David I. Wolfstein in psychiatry, and Dr. Frank W. Langdon associate professor in neurology. These three men probably represent the greater part of the neuropsychiatric background of Cincinnati. In 1923, Dr. Wolfstein resigned and was replaced by Dr. Emerson A. North in the department of psychiatry. Dr. Hoppe continued as the neurologic chief until his death in 1929. At this

* This article was provided through the courtesy of Dr. Emerson A. North, director of the Central Clinic of the Community Chest of Cincinnati and Hamilton County. Dr. North appointed a committee consisting of Dr. A. T. Childers, Dr. Philip Piker and Dr. Jack Hertzman, all of Cincinnati, who compiled the material embodied in this report.—Editor.

time, Dr. Charles Kiely was appointed acting director of both the departments of neurology and psychiatry. Until 1932, Dr. North acted as associate professor of psychiatry and as director of the Central Clinic. In 1932, the Central Clinic was moved to the Cincinnati General Hospital and became the Child Guidance Division of the Department of Psychiatry. Coincidentally, the departments of neurology and psychiatry were separated, neurology becoming a sub-department under medicine, with Dr. Howard D. McIntyre in charge. Psychiatry then became a major department with Dr. North as acting director. In 1934, Dr. North was officially appointed director.

The psychiatric department at the Cincinnati General Hospital is divided into two parts: (1) an out-patient dispensary clinic, and (2) an in-patient department which is the psychiatric pavilion of the General Hospital. The psychiatric pavilion is on the hospital grounds and consists of four wards, two male and two female, with combined capacity of 66 beds. Two wards are equipped to handle disturbed and excited patients, and the remaining two wards are set up to care for convalescent and quiet patients. The psychiatric service is staffed by three rotating internes, each of whom spends two months on the service and three fellows in psychiatry, two of of whom act as assistant residents, and one of whom is chief resident in direct charge of ward activities in addition to psychiatric consultations for the rest of the hospital. The fellows also spend two afternoons a week in the OPD clinic. The entire pavilion is under the direct charge of a full time assistant professor of psychiatry of the University of Cincinnati Medical College, who is responsible for the teaching and instruction of the senior medical students on the wards, and also of the internes and fellows in psychiatry. The medical students, in small groups of nine or ten, spend one month of their senior year working on the psychiatric wards; and during this period they work up cases and are given lectures and clinical presentations of psychiatric problems. Preliminary lectures in psychopathology in the sophomore year, and in the neuroses and psychoses in the junior year, prepare the students for the clinical instruction on the wards. During the eight months of the school years, a member of the visiting psychiatric staff of the hospital meets with the students and house staff twice a week for

case presentations, with each staff member spending a month on the service. The students and house men also have a conference once a week with a psychoanalytic member of the visiting staff.

The unit functions primarily as an observation ward, although cases may be kept for either short or long periods, at the discretion of the house staff. Cases are admitted to the service from various sources. Many come from the Probate Court, Police Court, other hospitals, and other parts of the Cincinnati General Hospital; and an appreciable number are referred by private physicians, by various social agencies in the community, and by the out-patient clinics. During 1939, 1305 patients were admitted to the service; and of this group 408, or less than one-third, were committed to a near-by state hospital, the majority of the remaining cases being discharged to their homes. Specific types of therapy for the various psychoses are given when indicated, and tubs are available for hydrotherapy. The pavilion also contains an occupational therapy unit; and two enclosed court yards adjacent to the pavilion are used for patients for out-door activities.

The psychiatric department works in close cooperation with the other departments of the hospital, and is an integral part of the General Hospital system. A neurosyphilis clinic, under mutual direction of the psychiatric and neurological units, is utilized in the teaching of medical students. The facilities of the psychiatric wards are utilized intensively by the School of Nursing and Health of the University of Cincinnati for instruction in psychiatric nursing. Active teaching is given to the nurses on the service by the psychiatric nursing staff and the psychiatric house staff.

The neurological service at the Cincinnati General Hospital is a part of the department of internal medicine. There are available 30 beds on the medical wards for the accommodation of patients suffering from neurological diseases. The offices of neurology and neurosurgery are combined in their locale and cooperation. The activities of the department center about a neuropathological laboratory which is directed by one of the neurologists and the neurosurgeon. There is a full time neuropathological Fellow in training in the laboratory. The main investigation work under way at present is a clinical and neuropathological study of neurological diseases associated with nutritional deficiency in man.

The beginnings of Longview State Hospital can be traced to the Commercial Hospital and Lunatic Asylum of the State of Ohio which was opened in Cincinnati in 1824, largely due to the activities of Dr. Daniel Drake. In 1860, the present main building (built on the Kirkbride plan) was opened for the accommodation of some 400 mental patients. This building, at the time of its opening, was considered one of the best equipped and most commodious structures for the care of mental patients then existing in this country.

Since 1880, Longview Hospital, although owned by Hamilton County, has been maintained entirely by the State. In 1927, arrangements were made for the sale of the hospital property to the State of Ohio. At the time of this sale, the grounds of the old Hamilton County Home were included. The buildings on both tracts, which are adjacent, have accommodations for 1869 patients. The total area occupied by these buildings, which is entirely within the city limits of Cincinnati, is 300 acres, 160 acres of which is under cultivation. The hospital is located seven miles north of Fountain Square on State Highway Number 4.

The patients' census as of February 8, 1940, was as follows: Total in residence 2672. Total absent 450. Total on books 3122. Total personnel as of this date was 308. Seven ward physicians are allowed in the tables of organization. The relation of ward personnel (attendants and nurses) to patients is 1 to 17.7. The cost per capita was $93.95. The admissions for 1939 totaled 584 and were received from the four southwestern counties of Ohio; *i. e.*, Butler, Brown and Clermont, in addition to Hamilton County. The total removals from the hospital were 500, 164 of which were deaths. The re-admissions for the year totaled 62 or 10.7 per cent of the total admissions. The relation of discharges to first admissions was 64.6 per cent. The relation of deaths to the whole number was 4.5 per cent. The population of the institution exceeds the normal capacity by 42 per cent.

There has been greatly added interest in research at Longview Hospital in the last few years. In cooperation with the College of Medicine and other research laboratories in the city a number of interesting approaches to the study of mental disorders have been made. The acquisition of an electroencephalograph, the development of an active shock therapy department and additional research

assistants have aided this. Of greatest importance has been the financial assistance given by the community and by the Scottish Rite Masons. Present research centers about the changes in psychological, electroencephalographic, blood flow and blood adrenalin during shock therapy, many aspects of the postencephalitic Parkinsonism and oculo-gyric crises. A more detailed description will appear in the program of the forthcoming annual meeting.

Other Hospitals.—The Good Samaritan Hospital is a general hospital in which two wings are set aside for psychiatric patients. This facility is available to private practitioners throughout the city. The patients are under the supervision of the attending physician, who is assisted in their care by the medical resident and the medical interne. Diagnostic resources are those of the usual private general hospital. Adequate personnel and equipment are available for all forms of psychiatric treatment, other than intensive psychotherapy. Insulin and metrazol shock, hyperpyrexia, occupational therapy and hydrotherapy are in active use. Provision has been made for separating the disturbed from the quieter patients.

The Cincinnati Sanitarium, located in the suburb of College Hill and established in 1873, is a private hospital for nervous and mental diseases having a capacity of 75 beds. It is probably the oldest hospital west of the Alleghenies, specializing in psychiatry and allied problems. Members of the resident and consulting staffs have always been prominent and active in the city's psychiatric affairs.

Extra-Mural Facilities.

The Mental Hygiene Council was organized in 1917 as one of the first voluntary health agencies of this community. It is a part of the Public Health Federation, the Health Branch of the Community Chest. The Mental Hygiene Council serves as an agency for the dissemination of information in its particular field, as a coordinator of mental hygiene interests in the community, and as a medium to further state and local facilities for the care of mentally sick persons. Membership in this Council is open to all persons whose interests are directly or indirectly related to mental hygiene problems. The Council meets once a month, except during

the summer, and these meetings serve as a forum for discussion, reports of committees, the coordination of activities and an interchange of ideas. The Mental Hygiene Council is one of twelve councils embraced in the Public Health Federation, dealing with health education, tuberculosis, heart disease, diabetes, social hygiene, cancer, housing, etc. The Council is the first disseminator of public information in this field. It maintains a speakers' bureau, prepares articles for the press, and from time to time car and bus cards and factory posters which enjoy a wide distribution. The Council is recognized by the Academy of Medicine as a mediator in cases which involve technical relations between the medical profession and social agencies.

The Central Mental Hygiene Clinic was organized January 1, 1923, to serve chiefly the needs of the many social agencies of the Cincinnati Community Chest. For several years its cases consisted both of adults and children. Later, the adults were cared for through a combination of services with the psychiatric out-patient department at the General Hospital. Still later, the entire clinic moved from its down-town location in the Community Chest Building to the General Hospital and became a part of the department of psychiatry there. This has been a child guidance clinic, differing from similar clinics in other large cities only in local practices, needs and facilities. It has always been one of the teaching resources for medical students, interested physicians, nurses, social workers and other students.

The Child Guidance Home of the Jewish Hospital is located in a separate building on the grounds of the Jewish Hospital. It was among the first clinics in this country to deal with child guidance work, being established in 1920. However, it differs markedly from the usual child guidance clinic in the fact that the patients live at the Home during the period of observation, the average length of stay being four weeks. During this time, a four-fold method of approach is followed. In other words, the child receives an intensive medical, psychological, psychiatric and social analysis, which permits a true evaluation of the causative factors underlying the child's behavior difficulties. The sources of referral are public and private and both local and national. While the work of the Home is primarily diagnostic, a great deal of follow-up work is carried out in connection with the referring sources. The follow-up in

many cases has extended over a period of 17 years or more. In addition to this, the Home also functions as a research center and is an educational center, being an integral part of the psychiatric department of the Medical College of the University of Cincinnati.

The Catholic Guidance Clinic is a new mental hygiene unit, established October 1, 1938. It is located in the Fenwick Building, on East Fifth Street, which also houses the Bureau of Catholic Charities, under the directorship of Monsignor R. Marcellus Wagner. Its purpose is to render diagnostic and treatment services to the Catholic clients referred chiefly by the Catholic Charities and the parochial schools. Its major emphasis is on behavior and personality problems of children. This clinic's case-load and activities have grown enormously during the short period of its existence.

The Hamilton County Juvenile Court under the direction of Judge Charles W. Hoffman, has been one of the leading social institutions of its kind in this country for more than a quarter of a century. Its needs for psychiatric services have been mostly met through a close working arrangement with the Central Mental Hygiene Clinic. The latter clinic, in conjunction with the psychiatric out-patient department of the General Hospital, also serves in selected cases for some of the other courts, particularly, Common Pleas and Domestic Relations Courts. Many of the psychiatric cases from the city and county for commitment through the Probate Court, are first studied in the psychiatric observation department of the General Hospital.

For a number of years, the Cincinnati Public Schools with its various special departments, has been one of the chief referring agencies for the Central Mental Hygiene Clinic and other local psychiatric facilities. The Board of Education has provided not only special schools for different kinds of education, but also, special facilities for treating handicapped children, such as, crippled, deaf, and the like. In addition there are the opportunity farm schools, Glenview School for boys, and Hillcrest School for girls. These are well managed institutions for the re-training of boys and girls who have been mildly delinquent. A small proportion of these children are to be regarded as psychopathic. The Board of Education maintains an extensive psychological laboratory service which is centered in its building on East Ninth Street. Here, also, are

located the offices of the attendance, visiting teacher and vocational guidance services. The enormous amount of mental hygiene work done in the public schools can merely be mentioned here.

It is not feasible to name all the social agencies and institutions of this community, which are keenly alive to the psychiatric implications of their work and who make active preventive effort in this field. A large number of such agencies are members of the Community Chest organization and many of them have offices in the Chest's building, on West Ninth Street. The Associated Charities has as a special feature its family consultation service. The Ohio Humane Society calls frequently for psychiatric aid in its efforts to protect children from improper care. The Children's Home is the chief Protestant agency for the placement and foster-home care of children. Its extensive work and well qualified staff, are features. The Bureau of Catholic Charities, has a number of functions, such as family case work and the placement of children, when desirable, in foster homes or institutions. It is also served by its own child guidance clinic which is described a little more fully elsewhere in this article. The Children's Hospital not only has its own mental hygiene service but also calls freely upon other community resources. The Children's Convalescent Home has for a number of years been very active in preventive work related to mental hygiene. These, and many other agencies, have utilized freely the psychiatric facilities of the city. Probably in no other city can be found a finer spirit of friendliness and cooperation between all agencies, psychiatrists and psychiatric clinics and hospitals.

State-Wide View.

Historical.—Ohio has the distinction of being represented in the group of the "original 13" who founded what is now The American Psychiatric Association almost a hundred years ago. Dr. William A. Awl of Ohio was one of the most distinguished physicians in Ohio's mental hygiene history. During his life (1799-1876), he helped found the Ohio State Asylum for the Insane in Columbus, and became its first superintendent when it opened in 1838. After serving in this office for 12 years, he became physician at the Ohio Institution for the Blind, having played an important rôle in its establishment.

The Commercial Hospital and Lunatic Asylum of Cincinnati, established in 1821, was the first in Ohio. It did not become a state hospital, however, until 1857. This institution and the Ohio Lunatic Asylum in Columbus were the only institutions caring for the mentally ill until 1855. The Toledo State Hospital, which was opened in January, 1888, was the first state hospital for mental patients in the United States to be erected on the cottage plan. The Ohio Hospital for Epileptics, which was opened in Gallipolis in 1893 was the first of its kind in the world supported by public revenue.

Other data regarding Ohio's institutions are shown in the accompanying table:

Ohio was one of the first states to create an agency for the study of juvenile delinquents and problem children. The present Bureau of Juvenile Research, located in Columbus, was established in 1914.

Management.—From the time of the opening of the Lunatic Asylum of Ohio in 1838 until August, 1911, all the institutions were under the management of a Board of Trustees, appointed by the Governor. The Trustees appointed superintendents who appointed their co-workers. From August, 1911, to July, 1921, the institutions were under the control of a Board of Administration, which was composed of four members appointed by the Governor. In 1921, the Department of Public Welfare came into existence with a director in control of all state institutions.

No significant changes in legislation were made until 1937, at which time there were a number of alterations in the laws of Ohio pertaining to the care and treatment of the mentally ill, epileptic and feebleminded. This new law established within the Department of Public Welfare a Division of Mental Diseases, consisting of a Commissioner and an Advisory Council. The intent and purposes of this new law were to provide more humane medical treatment and care, preventive and curative, and to promote a study of the causes of mental illness . . . . etc. Under the present arrangement, the Director of the Department of Public Welfare is the executive and administrative head of the Division of Mental Diseases. The Commissioner of Mental Diseases has complete charge, supervision and full responsibility in all matters relating to the treatment and care of the mentally ill in the State of Ohio.

DEPARTMENT OF PUBLIC WELFARE—DIVISION OF MENTAL DISEASES—STATE OF OHIO.

Name of hospital.	Superintendent.	Date of opening.	Capacity.	No. in hospital, Jan., 1940.	No. on trial visit and escape, Jan., 1940.
Hospitals for the Insane:					
Athens State Hospital	C. H. Creed, M. D.	January 9, 1874	1,880	1,821	473
Cleveland State Hospital	R. E. Bushong, M. D.	March 5, 1855	1,750	2,278	246
Columbus State Hospital	E. F. Clouse, M. D. (acting)	November 30, 1838.	2,400	2,534	765
Dayton State Hospital	E. L. Hooper, M. D.	September 1, 1855.	1,418	1,740	328
Hawthornden State Hospital	Guy H. Williams, M. D.	July 25, 1938	685	735	55
Longview State Hospital	E. A. Baber, M. D.	April 30, 1857	1,869	2,582	531
Massillon State Hospital	Arthur G. Hyde, M. D.	September 6, 1898.	2,279	3,230	775
Toledo State Hospital	O. O. Fordyce, M. D.	January 6, 1888	2,038	2,717	833
			14,319	17,637	4,006
Hospital for Criminal Insane:					
Lima State Hospital	H. M. Turk, M. D.	July 10, 1915	1,076	1,147	18
Hospital for Epileptics:					
Ohio Hospital for Epileptics	G. G. Kineon, M. D.	November 30, 1893.	1,991	2,072	175
Institutions for Feeble-Minded:					
Institution for Feeble-Minded (Columbus, Ohio)	F. L. Keiser, M. D.	August 1, 1857	2,025	2,142	661
Institution for Feeble-Minded (Orient, Ohio)	C. C. Kirk, M. D. (established as farm colony, 1898)	July 15, 1926	2,450	2,687	427
Institution for Feeble-Minded (Apple Creek, Ohio)	M. N. Fowler, M. D.	February 14, 1931	596	578	120
			5,072	5,407	1,208
		Grand total ...	22,457	26,263	5,407

In October, 1938, Dr. J. Fremont Bateman was appointed Commissioner of Mental Diseases. The present Division of Mental Diseases has had a rather unlucky beginning in that the Division began to function at a time when one administration was leaving office and there was a strict curtailment of all funds. The platform of the next administration, which is the present one, was that of strict economy, pledging no new taxes; and, therefore, the Division of Mental Diseases has continued as an experimental baby, obtaining what nourishment might be available.

There have been, however, a few significant accomplishments which are worthy of mention. In the first place, the number of physicians has been increased from 54 to 68. While a year ago we had three social workers in our institutions, today we have nineteen. All our state hospitals now have at least one full-time dentist, and many of them have two. Corresponding increases have been made in the various units of the medical department, such as occupational therapists, recreational therapists, laboratory and x-ray technicians, and clinical pathologists; and considerable new work in research has been started in those hospitals in districts where medical schools are available. The minimum salary for assistant physicians has been increased from $1700 to $2500 a year, with full maintenance. A corresponding increase has been made in the associated departments of medicine. The various state hospitals are attempting to increase their out-patient clinical facilities in order to do more work in the field of preventive psychiatry.

In order to give the physicians in the state hospital service the advantage of a review course in the basic medical sciences, and to coordinate and unify the medical work in all the state institutions, there was organized in 1934 the Ohio Institute of Post-Graduate Training in Psychiatry and Allied Branches of Medicine. This school is conducted annually at the Columbus State Hospital and the Ohio State University. Members of the faculties of Western Reserve University and the University of Cincinnati assist in the teaching.

Because of severe overcrowding and lack of facilities in the Cleveland and Massillon State Hospital districts, it has been necessary for the state to establish a "farming out" plan of care for about 1200 patients. These so-called "detention hospitals" are run by

private individuals or by private concerns at the joint expense of the state and the county. These detention hospitals were established in 1935.

At the present time there are four buildings under construction at the Massillon State Hospital and Hawthornden State Hospital, which will accommodate the majority of the patients now in the detention units. This method of caring for the group of patients who could not be admitted to state institutions has proven to be expensive and unsatisfactory. There have been some significant changes in Ohio's building program in that the trend now is toward well equipped medical hospital units and not so much the custodial cottage plan of building. In view of the fact that Ohio rates very low in its per capita cost of maintenance and ratio of employes to patient population; and with the institutional population 17 per cent overcrowded, it is proud that it ranks among the lowest in readmissions with the highest in the number of discharges.

It is hoped the fore-going material will provide not only historical information, but also a general view of the present day psychiatric set-up in the City of Cincinnati and in the State of Ohio. While not claiming leadership in very many respects, it is believed that this section has at least kept pace with the average of the rest of the United States. Ohio's psychiatrists have been pretty well aware of the state's needs for properly caring for its citizens who have become, or may become, mentally ill. Conditions of overcrowding in the state hospitals have become serious, particularly in some institutions. A state-wide bond issue for $4,000,000 was defeated by the voters in 1932, largely because of the economic conditions at that time. Economic limitations hamper progressive plans at present.

The set-up in Cincinnati is featured by the comprehensive arrangement by which so many facilities are drawn together not only for community cooperation, but also for available teaching opportunities. As in many other cities there is great need of both public and private funds for psychiatric work, research, therapy and prevention. This great urban area of nearly a million people has an outstanding place for philanthropy in the psychiatric field.

INSULIN THERAPY OF SCHIZOPHRENIA IN THE ELGIN STATE HOSPITAL.

(With Special Reference to Relapses and Failures.)*

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A review of the recent literature concerning the insulin shock treatment of schizophrenia indicates that the enthusiastic claims of three years ago have yielded to a more conservative evaluation of the treatment. Some clinics now consider their percentage of recoveries to be not significantly different from the "spontaneous" recovery rate.[1-4]

The following report is a review of our experience based upon the treatment of 315 patients diagnosed as schizophrenics at Elgin. Up to the present time only a few authors have published the results of more than 100 cases treated in a single clinic. Mueller [5] in 1938 surveyed 495 cases, taken, however, not from a single clinic but from 22 different Swiss institutions, Ross and Malzberg [6] in 1939 compiled their statistics on 1757 cases from 19 New York state hospitals and Berezowski's [7] 508 cases in 1938 represent the results from 15 Polish hospitals. Mueller states that the remission rate of 59.1 per cent in the Swiss Clinics including full and social remissions for patients with a duration of psychosis not exceeding six months, "does not come up to the high figures that have been and are still being reported by certain clinics." Patients with a duration of disease from 7 to 18 months show a remission rate of 48 per cent. Ross and Malzberg reported 23 per cent recovered and 41 per cent much improved among very recent cases, and only 13 per cent recovered and 29 per cent improved among cases of between 7 and 18 months duration. Berezowski's figures are even less favorable.

The results obtained at Elgin in 315 schizophrenic cases treated over a period close to three years continue to approximate those previously [8-9] presented.

* From the Elgin State Hospital, Elgin, Ill. Read at a meeting of the Illinois Psychiatric Society in Chicago, December 7, 1939.

In a group of 78 cases with an apparent duration of psychosis of not over six months the recovery rate was 72 per cent. Another 10 per cent were considered socially recovered thus giving a total of 82 per cent recoveries and social remissions.

In Group II, including 90 patients ill from 7 to 18 months, 54 per cent were paroled home, 34 per cent as recovered and 20 per cent as greatly improved.

Thus, in our experience the treatment benefits 68 per cent of patients with an onset of psychosis not over 18 months prior to treatment. In 147 cases of over 18 months in duration only 2 per cent of recoveries and 8 per cent of social remissions occurred, the total figure of 10 per cent being only slightly higher than the "spontaneous" remission rate in this group.[10]

This evaluation represents the status of the patient upon leaving the hospital. From these figures, however, one has to subtract the number of patients who relapsed within a year and did not respond to a second course of treatment. Since by far the majority of the relapses occurred during the first year after discontinuation of the treatment, it is obvious that percentages should be based upon results maintained for at least a year. After these deductions had been made it appeared that 60 out of 100 patients with a duration of illness not exceeding 18 months can be expected to maintain recovery or great improvement for 12 months or more.

Among the 315 cases treated with insulin were 89 who had previously undergone a course of metrazol convulsive therapy with little or no improvement. These cases showed about the same recovery and improvement rate as those not treated with metrazol previously. In other words insulin shock is effective in recent cases after metrazol shock has failed, and in percentages approaching those obtained in similar non-metrazol treated patients.

The discrepancy between the Elgin figures and those of Ross and Malzberg and Mueller may be due to one or all of three factors, first, the selection of cases, second, the evaluation of results, and third, the technique.

Every case in the Elgin State Hospital diagnosed schizophrenia with a duration of psychosis up to 18 months was selected for insulin treatment unless subjected to some other form of therapy. In case of failure insulin was then instituted. All those patients, about whom there was even the slighest doubt as to the diagnosis

of schizophrenia, were not included in our statistics. Hence it is evident that we avoided special selection of cases which might have influenced the statistics.

The evaluation of the treatment results was strict. The degree of improvement in each case was decided by 10 to 15 staff members, including those who knew the cases upon the receiving service, those who had close contact with the patient during the treatment and some who had never seen the patient before. This procedure was chosen in order to obtain as unprejudiced a judgment of the patient's condition as possible.

The third possible explanation of the contrast between the Elgin results and those of Mueller and Ross and Malzberg is a difference in technique. Our procedure has remained essentially unchanged since the treatment was begun approximately three years ago. Statistical check-ups after one, two, and more than two and a half years' treatment revealed a variation of only 12 per cent, indicating a definite consistency in the findings.

The administration of an adequate number of treatments was considered to be of importance. Since several patients did not show improvement before the 40th or 45th shock, we continued treatment with every patient up to 50 shocks unless sufficient response was shown prior to that point. If the patient had not responded by that time it was according to our experience useless to continue. However, the slightest response made us carry on the treatment as long as the patient showed progressive signs of improvement. The average number of shocks for successfully treated patients was 35.

The safety of the treatment was thought to depend upon two important factors and the fact that thus far no fatilities or lasting serious complications have been encountered may be attributed to our insistence upon keeping the dosage as low as possible and adjusting the patient slowly to shock. The treatment was started with 10 or 15 units and increased by five units daily until the shock dose was reached. Occasionally lowering the dosage by 40 or 50 per cent during the rising phase facilitated the induction of shock. We explain this phenomenon by failure of the adrenal glands to respond to the sudden reduction of the amount of insulin.[11] As soon as the shock dosage was reached the amount of insulin was adjusted daily.

The patient was never allowed to go into shock unless he had shown no improvement with five or six hypoglycemic states. The first five shocks lasted only 10 to 15 minutes, the next five 15 minutes longer and so on unless improvement had been noted. The longest duration of shock in any case was 1½ hours. It would seem that the stage of hyperactive reflexes (Babinski frequently present) was of sufficient therapeutic intensity to preclude the necessity of a deeper coma with abolished reflexes.

The therapeutic value of epileptiform seizures during insulin treatment has been repeatedly discussed in the literature.[12-18] Of all our patients 33 per cent had one or more such seizures, but they occurred in only 24 per cent of our successfully treated patients. There was no therapeutic benefit derived from them. At the last meeting of The American Psychiatric Association verterbal fractures during insulin convulsions were reported. X-ray examination of the spinal columns of a sampling of our patients who had between 1 and 12 epileptiform attacks did not reveal any fractures.

Our opinion that seizures during insulin treatment are of no therapeutic value was also supported by the fact that metrazol convulsions produced during hypoglycemia in the precomatose state did not increase our recovery or improvement rate in 30 patients who had had both insulin and metrazol without success prior to the combination treatment.

We produced three convulsions a week, the patient also being under insulin on the interval days, and limited this treatment to 15 convulsions. The average amount of metrazol used for producing a convulsion was 3.2 c.c. compared with the average amount of 6.8 c.c. of a 10 per cent solution necessary to provoke a convulsion during the previous course of metrazol treatment. We discontinued this modification especially in view of the observation that even small amounts of insulin and metrazol when used in combination produce severe damage of the ganglion cells in the brains of rabbits, whereas, similar amounts used separately produced no demonstrable brain changes.

RELAPSES.

Seventeen patients, apparently recovered following treatment, had to be readmitted to the hospital because of recurrence of their psychotic symptoms. They represent 15 per cent of the recovered

and greatly improved patients of the first two groups. This is in accordance with the number of relapses observed in other clinics.

These patients present three special problems. First, should they be treated again? Second, are there any special criteria which might suggest a possible recurrence of symptoms? Third, are there any events in the interval periods which may be interpreted as premonitory signs?

The results of our studies of these cases show that 4 out of 17 had had a self-limited psychotic episode before the one treated with insulin, while in the group of non-relapsed recoveries only 5 per cent of the patients were found to have had a previous attack with "spontaneous" recovery. None of the four patients responded satisfactorily to a second course of therapy, suggesting a poor prognosis for patients who had had remissions prior to insulin therapy.

We have gained the same impression from 10 other cases who were treated a second time. Though eight of these showed a good remission with a second course of insulin, three had a third and one a fourth attack. The tendency to stay well diminished with each attack. This is also evidenced by the shortening of the intervals between the attacks and by the fact that of our entire group of 14 re-treated relapses only five stayed well and these are not considered out of danger yet. In spite of the fact that the prognosis becomes worse with each attack, we think that each relapsed patient should have the benefit of another course of treatment.

The second point of our study was: by what criteria a relapse might be predicted. Three features were noted common to all the relapsed patients. They all had a leptosome habitus, pre-psychotic introverted personality, and in so far as we could determine the psychosis had started very suddenly. It was suspected that environmental conflicts, especially the home situations might have driven the patient into another psychotic attack, but in only two cases could such a possibility be considered, and even in those two the evidence was more than doubtful. In consideration of the many difficulties faced by patients who have remained well since treatment was discontinued, we are very guarded in prognostications based upon bad or good home environments.

We have seen no difference in symptomatology, course and development of the schizophrenic process between these patients

individually and those who did not relapse. From the foregoing it may be inferred that a past spontaneous recovery is a presumptive warning of a future relapse.

In the third place one is almost invariably justified in predicting a new outbreak of psychotic symptoms in a very short time, if, during the interval the patient presents even the slighest evidence of instability such as nervousness, irritability, loss of sleep, appetite or weight.

We observed that the symptomatology of the first and second attacks was almost identical in all our cases except two who changed from a mute, catatonic stupor in the first attack to a rather manic-like picture during their second.

The relapses occurred usually within six months after completion of treatment, though two cases relapsed as late as 15 months after treatment had been discontinued. Thus, the greatest danger of a relapse lies within the first six months after discontinuance of treatment and then decreases rapidly with every month the patient is in remission.

FAILURES.

Of our unsuccessfully treated patients we have considered in this study only those 55 whose symptoms were of less than 18 months duration. The question naturally arose, whether these patients were in any way different from those who either recovered or improved greatly. We, therefore, compared by duration-of-psychosis groups an equal number of failures with unselected patients of the recovered group. The study included: first, the symptomatology of the psychoses; second, an evaluation of the patients' personality factors; and third, physiological differences.

The symptomatology study did not furnish an answer to our question. The onset of the psychoses was rather sudden, usually within three weeks, accompanied or preceded by loss of weight and malaise in a like number of patients in the failure and control groups. Obvious physical or mental factors possibly precipitating the psychoses were sparsely but evenly scattered throughout the groups. The types of psychosis were also uniformly distributed. Occurrence of disturbed consciousness, memory defects and untidiness, significant alterations of affect did not indicate any differentiation.

All such factors related to the patients' immediate personality as age, constitutional type, heredity, pre-psychotic behavior and environment, education and intelligence showed no significant predominance in either group.

We have no data as yet as to physiological comparisons. The work of Jellinek and his associates [19] suggests, that there is a difference between patients who later recover and those who fail to do so. "The recovered patients showed before treatment greater pulse variability than normal, but less than the not recovered patients. Before the treatment the recovered group showed a tendency toward hypotension. Initially the ratio of cholesterol ester to free cholesterol showed great inconsistency in the subsequently recovered patients, but normal consistency in the not recovered patients."

These observations are, however, of little practical help for the prognosis of one single case. We found such an aid in the patients' reaction to intravenous sodium amytal. Sodium amytal was administered to 26 patients before treatment was begun and the responses were compared with the final results of insulin treatment. We found that of a group of 15 insulin failures 86 per cent had not responded satisfactorily to the injection of sodium amytal inasmuch as they answered questions only hesitatingly and showed but little more interest in the environment. Only 14 per cent showed a response as seen in the group of 11 recovered patients, all of whom showed a very good response to the amytal injections. They answered questions readily, realized that they were sick and in need of treatment and corrected their delusional ideas. From these findings it would seem that those schizophrenic patients who show little or no response to injections of sodium amytal are apt to fail to improve under insulin, whereas a large percentage of those in whom it is possible to obtain a lucid period with sodium amytal will respond favorably to insulin therapy. These findings are confirmed by a recent publication by Harris and his associates [20] who state that 77 per cent of patients who showed an "ameliorating response to the injection of sodium amytal had a favorable therapeutic response to insulin hypoglycemic treatment."

SUMMARY.

Three hundred fifteen schizophrenic patients treated during a period of almost three years showed 72 per cent recoveries and

10 per cent social remissions in cases with a duration of psychosis less than seven months, 35 per cent and 20 per cent respectively in the group ill from 7 to 18 months and 10 per cent for cases over 18 months duration.

A similar percentage was obtained in patients who had not responded to previous metrazol therapy.

There were 15 per cent relapses in the first two groups.

The treatment with insulin and metrazol simultaneously did not benefit patients who had failed to respond previously to either therapy. For this reason and also because of the disproportionately severe toxic changes in the cerebral ganglion cells of similarly treated rabbits this modification was discontinued.

No fatalities or lasting serious complications were encountered.

Epileptiform seizures occurring in 33 per cent of the patients had no therapeutic effect. No spinal fractures were found in a sampling of patients.

The number of relapses was especially high in cases which had had previous spontaneous remissions. The chance of another recovery decreases with each relapse. No definite criteria for a future relapse could be established from the patient's history before, or the symptomatology during the psychosis. The relapses occurred most frequently within six months after discontinuance of treatment.

A group of failures was compared with a group of recovered patients as to symptomatology of the psychosis and evaluation of personality factors. This study failed to furnish any fundamental differences. A practical prognostic help was found in the patient's reaction to intravenous sodium amytal.

BIBLIOGRAPHY.

1. Stalker, Harry, Miller, W. Malcolm, and Jacobs, J. M.: Remission in schizophrenia: Insulin and convulsion therapies compared with ordinary treatment. Lancet, I:437, 1939.
2. Lehoczky, T., Eszenyi, M., Horanyi, B., and Bak, R.: Vergleichende Untersuchungen ueber aktive und nicht aktive Behandlung der Schizophrenie. Orv Hetil. 1939, 307. Ref. Zentralbl. Psych. Neur., Vol. 89, No. 6, 398, Sept. 1, 1939.
3. Langfeldt, Gabriel: Neue Gesichtspunkte zur Wuerdigung der Insulinbehandlung bei Schizophrenie. Nord. med. Tidskr., 888, 1938. Ref. Zbl. Neur. Psych., 92: 276, 1939.

4. Horanyi-Hechst, B., and Szatmari, A.: Ueber Erfahrungen mit Insulinschockbehandlung an der Budapester Psychiatrischen und Nervenklinik. Mschr. Psychiat., 98: 101, 1938.
5. Mueller, M.: The insulin therapy of schizophrenia. Am. J. Psychiat., Vol. 94, Suppl. issue, p. 5, 1937-38.
6. Ross, J. R., and Malzberg, B.: A statistical study of results of pharmacological shock therapy in the New York State hospitals. Am. J. Psychiat., 96: 2, 1939.
7. Berezowski, F.: Bemerkungen ueber die bisherigen Ergebnisse der Insulinbehandlung der Schizophrenie nach dem experimentellen Material in Polen. Roczn. psychjatr. H. 31, 99, 1938. Ref. Zbl. Neur. Psych., 90: 157, 1938.
8. Read, C. F., Heilbrunn, G., and Liebert, E.: Studies in the insulin treatment of dementia præcox. J. Nerv. Ment. Dis., Vol. 90, No. 6, 747, 1939.
9. Steinberg, L., Heilbrunn, G., and Liebert, E.: Evaluation of the newer treatments of dementia præcox. Ill. Med. J., Vol. 75, No. 5, 405, May, 1939.
10. Shapiro, L. B.: Prognosis in dementia præcox prior to introduction of shock therapy. Elgin paper, Vol. 3, 1939.
11. Heilbrunn, G., and Liebert, E.: Observations of the adrenalin level in the blood serum during insulin hypoglycemia and after metrazol convulsions. Endocrinology, 25: 354 (Sept.), 1939.
12. Ruslander, D.: Modifications in the insulin treatment of schizophrenia. Psychiatr. Quart., 13: 357, 1939.
13. Salm, H.: Erfahrungen und Erfolge mit der Insulinbehandlung bei 150 Schizophrenen. All. Z. Psych., 109: 116, 1938.
14. Dinther, B. van, et Jansen, E.: Du traitment de 45 malades, souffrant de schizophrenie, par la cure au choc par L'insuline therapie selon Sakel. Psychiatr., Bl. 42, 112, 1938.
15. Finiefs, L. A.: Significance of fits in the insulin therapy of schizophrenia. Lancet, I: 776, 1938.
16. Ruslander, D.: The epileptic seizure in the hypoglycemic treatment of schizophrenia. Psychiatr. Quart., 12: 372, 1938.
17. Schulz, H. E.: Zur Frage des epileptischen Anfalls im Insulinschock, Psch. Neur. Wschr., 307, 1938.
18. Gross-May, G.: Ueber epileptiforme Anfaelle bei der Insulintherapie der Schizophrenie. Nervenarzt, 11: 400, 1938.
19. Jellinek, E. M., Cameron, D., Randall, L. O., Looney, J. M., and Dyer, C. G.: Physiological studies in insulin treatment of acute schizophrenia. Endocrinol, 25, Nos. 1 and 2, pp. 96-110 and 278-285, July and August, 1939.
20. Harris, M. M., Horwitz, W. A., and Milch, E. A.: Regarding sodium amytal as a prognostic aid in insulin and metrazol shock therapy of mental patients (dementia præcox). Am. J. Psychiat., 96: 2, 327, 1939.

NEUROPSYCHIATRIC DISORDERS OCCURRING IN CUSHING'S SYNDROME.

(Pituitary Basophilism.)*

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Following the description by Cushing [1] in 1932 of a disease which he termed pituitary basophilism and which was immediately recognized as constituting a clinical entity, there have been reported an ever increasing number of similar cases from all parts of the world. It is not our purpose to review the extensive literature on this subject which has been done adequately by Jonas,[2] Pardee [3] and Kessel.[4] The prominent clinical features of this disease, as first mentioned by Cushing and confirmed by later observers, are: a rapidly acquired, painful adiposity having an axial distribution with appendicular sparing; sexual dystrophy manifesting itself as early amenorrhea in females and impotence in males; plethoric skin; purplish striæ; hypertrichosis in females; vascular hypertension; tendency to develop kyphosis; lumbospinal pains; fatigability and ultimately extreme weakness. Other symptoms that may be present in some cases are: purpura-like ecchymoses; dryness of skin; polyphagia, polydipsia and polyuria; aching pains in the eyes; insomnia. Laboratory studies will frequently reveal: osteoporosis; glycosuria; hyperglycemia with diminished glucose tolerance; hypercholesterolemia; erythrocythemia. All the clinical features that have been enumerated are not to be found in every case and, indeed, it has been suggested that there are many relatively benign examples of this condition which may properly be considered "forme fruste." In fully developed cases the average duration of illness is five years and death often is attributable to

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the unusual susceptibility to pyogenic infections which these patients show.

Recently, Maclay and Stokes [5] have recorded a case of Cushing's syndrome in which a psychosis developed. We wish to describe a case in whom the development of a similar psychosis was an outstanding feature. This patient also showed evidence of an extrapyramidal neurologic disorder which, so far as we have been able to determine, has hitherto not been described in Cushing's syndrome. Three anatomically unverified cases have been reported in which were observed such neurologic disorders as flaccid quadriplegia,[6] spastic paraplegia [7] and motility impairment of the extremities.[8]

CASE REPORT.

L. F. G., female, 27 years old, admitted to the New York State Psychiatric Institute and Hospital in September, 1936; history of major tics since early childhood; early in 1934 gradually developed change in body contour; in September, 1934, sudden onset of psychosis characterized by marked depression and self-condemnatory ideas and associated with amenorrhea; persistence and progression of all symptoms; on admission showed physical signs characteristic of Cushing's syndrome plus evidence of extrapyramidal disease of the central nervous system; mental picture characterized chiefly by depression and psychomotor retardation; condition remained unchanged until death in February, 1937, following left suprarenalectomy. Post-mortem showed: External appearance characteristic of Cushing's syndrome; small and atrophic right adrenal; histologically benign neoplasm of the left adrenal; marked hyaline changes (Crooke) in the basophil cells of the adenohypophysis; marked congestion and edema of the brain parenchyma with focal areas of encephalomalacia in the frontal lobes.

Family History.—On the paternal side, patient was of Russian-German ancestry and on the maternal side of Polish-German ancestry. Father was born in New York City and died there at 66 years of age of Bright's disease after a chronic illness of one year. He is described as having been a stern, exacting, miserly man, whose marital life was marked by frequent quarreling with his wife concerning finances. Mother was born in Cincinnati and is living and well at 59 years of age. She has always been on friendly terms with her children but may possibly have been partial to the patient who was the youngest of six siblings. A sister of the patient died at eight years of age as a result of ptomaine poisoning and one brother, who was affected by the same illness, subsequently had tics for many years. Paternal grandmother died at 72 years of age after having been ill for ten years with paralysis agitans. A paternal aunt died in a psychiatric hospital where she was diagnosed as having dementia præcox, paranoid type. The remainder of the antecedent and collateral lines reveals no history of nervous or mental disease.

Personal History.—Patient was born in New York City January 31, 1909. She was not a wanted child by the mother and a mild attempt to induce abortion by medication was made early in the pregnancy. Birth and early development were normal except for nocturnal enuresis which persisted until six years of age. Patient is said to have been a talkative infant who early began to use big words. Throughout infancy, she was small in stature. At three years of age she had an acute illness referred to as "ptomaine poisoning" and was seriously ill for ten days, convalescence lasting about two months. Patient started school at six years of age and made satisfactory progress throughout. At about six years of age she gradually began to develop a tic involving one shoulder and arm and accompanied by rotation of the head to the homolateral side. This tic persisted and was frequently so marked that she was forced to stay out of school for days at a time. Patient is said not to have been particularly self-conscious about it and when scolded by her mother, would reply "I don't know I am doing it" or "I can't help it." She also had a tic-like grunt. Treatment was received at various institutions during childhood without any noticeable beneficial effect. Somnambulism was an occasional occurrence between 8 and 11 years of age. Patient is described as having been a vivacious and sociable child who played freely with other children but who preferred to be in the company of adults. She was always considered the baby by the remainder of the family. In 1918, when she was nine years of age, she had influenza and was seriously ill for one week; delirium and generalized muscle contractions were a transient phenomenon in this illness. Measles, varicella and pertussis were unaccompanied by complications or sequelae.

Menarche occurred at 14 years of age and menses showed no irregularity. At about this time interest was manifested in members of the opposite sex. During her adolescent years, patient is described as having been a laughing, vivacious girl but inclined to be opinionated. After her graduation from public school, she went to business school and then worked as a stenographer. At this time she developed an increasing dislike for her father because of his strictness regarding her social activities and financial expenditures. This dislike became so marked that they practically ignored each other and were barely on speaking terms. At the time of his death, she showed little evidence of grief. For a period of one year at the age of 19, against the opposition of her family, she maintained a companionship with a man much older than herself.

Patient was always a slender girl, her weight averaging between 100 and 105 lbs. For many years she was sensitive about the shape of her nose and at 20 years a plastic operation was performed. During the period of convalescence after this operation, she was very nervous. Such a degree of nervousness had only been manifested on one previous occasion when she was panic stricken after swallowing a bone. At about this time patient moved to Rochester where she lived with her sister and while living there was active socially and financially independent. After a short courtship, she was married November 21, 1931. She knew her husband had heart disease and

seemed to worry considerably about this. Marital life was happy although she is said to have had little desire for sexual intercourse. The tics present since childhood persisted but were possibly somewhat less noticeable after marriage.

History of the Present Illness.—The exact date of onset is indefinite. It is known that following marriage patient gained weight gradually and early in 1934 a change in body contour became noticeable. In August, 1934, while vacationing in the mountains, a slight change in behavior was noted in the form of a more subdued attitude. At this time an episode occurred in which the patient became greatly excited and thought she was going blind when lights flickered during a thunder storm. In September, 1934, a sudden marked change in behavior occurred immediately after an incident when the patient's husband fainted at the unveiling of the tombstone of his father. At the time she was shocked by the thought that he might be dead. She continued to be emotionally disturbed the remainder of that day and on the following morning urinary incontinence was discovered. Patient appeared to be dazed and resistive. Following this she gradually became disinterested in her person and environment. She frequently expressed a desire to die and threatened frequently to stop everything, even her bowel habits. Self-condemnatory ideas were also expressed. Amenorrhea first appeared in October, 1934, and persisted. Pregnancy was disproved by laboratory examinations. A vacation resulted in no change in the patient's condition. Fecal incontinence was noted in the spring of 1935 and subsequently recurred on one other occasion. Patient's speech gradually changed, becoming quieter and slower until finally it was almost inaudible. A speech defect in the form of stuttering appeared and persisted. A change in gait was noted, consisting of slowness and small steps. Flushing of the face also appeared. Early in 1935, there was a rapid increase in weight which reached 130 lbs. She was seen by various physicians and a gynecological operation (D. and C.) was performed in the spring of 1935. Admission to another psychiatric hospital occurred in July, 1935, and upon her discharge in December, her condition was diagnosed as "manic depressive psychosis, depressive type." Subsequently her condition became progressively worse. She received various forms of medication, including injections of theelin, but therapy was ineffective. Childish speech was noticed at times and shortly before admission when asked about the reason for her illness, patient stated "I wanted to stop, oh God, when will this end, I won't go on." She was also heard to moan and shout whenever a noise occurred and no one was in the room with her. After the onset of her illness, her major tic involving the head and shoulders gradually subsided and finally ceased entirely but facial tics persisted.

Physical Examination.—Patient was a short, round shouldered, obese, young white female with a mask-like facies. Obesity was characterized by a distribution involving the trunk, neck and face, with sparing of the extremities. There were definite, localized, palpable collections of adipose tissue in the supra-clavicular regions, giving the face a "moon-like" appearance (Fig. 1). She had a ruddy complexion, with moderate cyanosis of the face and mucous membranes. There was a definite tendency towards hirsutism,

the degree of which was not completely in evidence because of the artificial removal of hair on face and legs. There was a masculine distribution of pubic hair. In the erect posture, a protuberant abdomen was noted and in the recumbent position there was observed slight abdominal distention with blue striæ on the antero-lateral aspects of the abdomen (Fig. 2). The skin, except for hyperhidrosis of the palms and soles, was dry and showed slight scaling. Examination of the chest revealed impaired resonance over the right base and axilla, accompanied by crackling rales over the same areas. Area of cardiac dullness was enlarged to the left. There was a muffling of the first sound at the apex and an accentuated P-2. Blood pressure was 148/110 and on another occasion 160/112.

Neurological Examination.—In the erect position, patient showed a tendency towards fixation of body posture. Gait was characterized by slow and abbreviated steppage when walking and by bizarre hopping when running. In assuming the erect from the sitting position and vice versa, there was noted a decomposition of body movement. In walking, there was a diminished associated swinging of the arms. In the dorsal decubitus, the posture of the patient was characterized by the right lower extremity being held in slight adduction, internal rotation and plantar flexion of the foot (striate position); left lower extremity was held normally except for an increased tendency towards dorsal flexion of the foot. The left upper extremity showed a tendency towards universal flexion, especially noted in the fingers and hands; right upper extremity showed an extra-pyramidal posture of the hand with ulnar deviation, rounding of the dorsum and adduction of the thumb. Athetoid movements were noted in the fingers and toes of all extremities, more so on the left. There was a slight intention tremor in the finger-to-nose test bilaterally. Transient cog-wheel rigidity was obtained at the right elbow and right wrist. There was an irregular inconstant blepharospasm which if prevented by the examiner was still shown in the upward deviation of the eyes. A tic-like retraction of the right angle of the mouth was most prominent when the patient attempted to speak. Speech was barely audible and was characterized by a defect in the form of stuttering. Deep reflexes were generally hyperactive; superficial reflexes were present and equal; no pathologic reflexes were elicited. There was no disturbance of sensation. The pupils were equal, regular and reacted well to light and in accommodation. The left palpebral fissure was greater than the right. Patient showed some difficulty in maintaining conjugate gaze and there was a slight dissociation of head-eye movements. Fundi showed no gross abnormalities. Corneal reflexes were active. There was no evidence of facial, palatal or lingual paresis.

Psychiatric Examination.—Patient was a quiet, cooperative, well behaved person. She showed marked psychomotor retardation and her spontaneous speech was nil. Voice was practically inaudible when answering questions but showed a remarkable change when the patient was reading, becoming at such times distinctly audible. The marked stuttering defect ordinarily noted in her speech was much less noticeable when she read. Interviews were conducted by means of written answers to questions. In expressing herself

in this manner, she showed a marked poverty of thought as indicated by the paucity of words used. Answers in most instances consisted of a single word, but were coherent and relevant at all times. In writing, she held the pencil properly but often showed marked delay in initiating necessary movements. The mood was apparently one of constant depression, with weeping occasionally noticed. No definite mental trend could be ascertained. She was unable to recall the onset and course of her illness, but described her condition as "I've been sad all the time." There were no hallucinations, delusions or ideas of reference. Patient was well oriented in all spheres. There was no impairment of memory. Retention and immediate recall were good. Fund of school and general knowledge seemed to be rather poorly retained. Difficulty in concentration was manifested during test for intelligence rating with responses frequently consisting of the phrase, "I don't know." At times she appeared perplexed. Insight was fair. Judgment was definitely impaired. Her plans for future consisted of "go home; be with mother."

Course.—Following admission, the condition of the patient, both mentally and physically, remained essentially unchanged. Her adjustment to the ward routine was satisfactory and she required no special attention. She was quiet and cooperative; caused no disturbances; ate and slept well. She took no particular interest in her surroundings and was usually observed sitting by herself. All instructions were obeyed. There was no evidence of untidiness or of sphincter incontinence. When asked how she felt, her answers uniformly consisted of such curt phrases as "I'm sad" or "I'm tired." There was no spontaneous speech. Marked psychomotor retardation persisted and she constantly appeared to be in a state of depression, with weeping noted occasionally. At times her facial expression appeared to reflect tenseness and worriment, but the presence of these feelings were difficult to confirm. After the completion of various laboratory examinations which seemed to indicate the existence of a left supra-renal neoplasm, patient was transferred to the Presbyterian Hospital where she was operated upon February 1, 1937. Immediately following the operation, she was able to talk in a normal tone of voice with her husband. Twelve hours afterwards, however, she developed a hyper-pyrexia associated with tachycardia and her condition rapidly became worse with exitus next day.

Laboratory Data.—Urinalysis showed specific gravity 1.030 and a slight trace of albumin. There was no glycosuria and no Bence-Jones protein. Blood count: R. B. C. 5,750,000; W. B. C. 11,700; Hb. 99 per cent. C. I. .87, Polys. 78 per cent, Lymph. 18 per cent, Monos. 4 per cent. Blood chemistry studies revealed: urea nitrogen 11.9 mg., sugar 119 mg., serum cholesterol 294 mg., calcium 10.9 mg., serum albumin 2.0 grams and serum globulin 4.5 grams. Dextrose tolerance test after the administration of 60 grams of dextrose showed: fasting level—113, one-half hour—193, one hour—236, two hours—252, three hours—204. Blood and spinal fluid serology was negative for evidence of syphilis. Spinal fluid showed: 6 cells, ± Pandy, total protein 97 mg., colloidal gold curve 1212110000. B. M. R. was + 5. X-ray of the chest showed slight increase in the left ventricular outline of the heart consistent with hypertensive heart. X-ray of the skull, pelvis, spine and

long bones showed a slight decalcification of the cranial tables and a coarse osteoporosis of the shafts of the long bones. Encephalogram, after the introduction of 150 c.c. of air, showed the ventricular system to be normal in size, shape and position with considerable air in the subarachnoid regions normally distributed. Flat plate of the abdomen showed the left renal outline to be slightly larger than the right but both were regular in contour and normal in shape and position. Cystoscopy showed no gross abnormalities. Pyelography revealed no evidence of renal disease. Peri-renal insufflation of air showed the right kidney and supra-renal gland to be normal in size and position; the left kidney was lower than the right and appeared to be normal in size but the outline of the upper pole was obscured by a mass which was well surrounded by air and was considered to be suggestive of a supra-renal tumor.

AUTOPSY *

Body was that of a well developed, obese, young white female. There was a florid, cyanotic hue to the skin of the face, neck, shoulders and back. There was no jaundice. The fat distribution was unusual, being most prominent in face, neck, shoulders and about the pelvis. The extremities were not obese. Hirsutism was noted, especially on the upper lip where the hairs measure up to 1 cm. in length; also on the face and the skin of the extremities. Purplish striæ were present in both flanks and on the anterior surface of each thigh. The abdomen was distended and tympanitic to percussion. The clitoris was slightly enlarged and the labia minora were a little more prominent than usually seen.

Examination of the viscera revealed: a fatty liver, with a small hemangioma in the upper portion of the right lobe measuring 1.5 cm. in diameter; fibrous peritoneal adhesions; a pancreatic rest in the jejunum; hemosiderosis of the spleen; an anomalous distribution of the coronary arteries.

Endocrine Glands.—On gross study the glands of internal secretion were found to be normal except for an absent left adrenal (removed surgically) and a small right adrenal gland. Microscopically the pancreas and the thyroid, parathyroid and pineal glands showed no abnormalities. There was an absence of graafian follicles in the ovaries. Significant changes were observed in the hypophysis and in the adrenal glands.

Hypophysis.—This gland, which showed no gross abnormalities, was cut in serial sections and stained by a modified Mallory method. In nearly all the basophil cells there were: marked hyaline changes in the cytoplasm, partial to complete disappearance of the granules, vacuolizations and disfiguration of the nuclei (Fig. 3).

* We wish to express our appreciation to the Department of Pathology, College of Physicians and Surgeons, N. Y. C., for permission to quote the pathological findings in this case and to Dr. S. E. Barrera, Principal Research Psychiatrist, Psychiatric Institute and Hospital, for the microphotographs and advice in the interpretation of the neuropathological material.

Right Adrenal.—This gland was normally located; was much thinner than normal; and on section, it was seen that the cortex was reduced to about one-half its normal width. The medulla appeared normal. Microscopic examination disclosed a thin cortex which, except in small adenomatous nodules, was poor in lipoid.

Left Adrenal.—This gland was oval in shape, weighed 13.6 grams and measured 4.5 x 3.5 x 3.0 cms. It was covered by an intact fibrous tissue capsule. A cross section revealed a golden yellow surface traversed by wavy, brownish striations. The bulk of the mass was made up of orange-yellow zones, measuring about 3 mm. in width, surrounding greyish-brown areas varying in size. Nowhere could the ordinary arrangement and structure of the adrenal cortex be identified.

Microscopic examination showed that the tissue was made up of several types of cells (Fig. 4); some were very large, with foamy cytoplasm and small, dark, pyknotic nuclei; others were of normal size, with deeply eosinophilic cytoplasm and normal nuclei; no medullary tissue could be identified. In a section stained for connective tissue, there was only a scant amount of collagenous stroma. In a section stained by the Foot-Bielschowsky method, a definite reticulum continuous with that in the capsule outlines the clumps of cortical cells. In a section stained for iron, there was no prussian blue reaction visible. In a section stained for fat, about one-half of the cells were filled with sudanophilic globules. In a section stained by the Giemsa method, no specific colored granules were seen.

Brain.—On macroscopic examination, the cerebral hemispheres were symmetrical. There was no evidence of cortical atrophy. The leptomeninges were thin and translucent throughout. The vessels at the base were normal. The ventricular system appeared normal. Sections at various levels through the cerebrum, cerebellum and brain stem revealed no gross abnormalities.

Microscopic examination of the right frontal lobe showed that the cortex was edematous and congested; many of the ganglion cells contained an excess of lipoid. A large area was disrupted in the white matter (Fig. 5). Much of the structure had disappeared and there were small numbers of compound granule cells, some of which were in transition from microglia. Some of the capillaries showed endothelial hyperplasia. All were congested, and there was marked congestion at the margins of the lesion. There were a number of recent hemorrhages in this same zone. In a section of the parietal lobe, the leptomeninges and parenchyma were congested and edematous. Many of the ganglion cells contained an excess of lipoid. There was diffuse loss of neural elements in the cortex. The edema of the parenchyma was intense in some zones in the substantia nigra. There was also some congestion. Many of the ganglion cells in all of the nuclei contained an excess of lipoid. There were no other changes of note in the nuclei, tracts or wall of the third ventricle.

The parenchyma in a section of the medulla was edematous and considerably congested; in the region of the floor of the fourth ventricle there was a recent perivascular hemorrhage.

DISCUSSION.

Considered from the standpoint of its varied symptomatology, there can be little doubt that pituitary basophilism is a polyglandular disease and this was recognized by Cushing.[1] The concept originally proposed was that the pathologic lesion responsible for this syndrome is a basophilic adenoma of the hypophysis which produces many of its effects indirectly by its action upon other endocrine glands such as the gonads, supra-renal cortex, pancreatic islets and parathyroids.[9] This concept in its original form cannot be maintained in view of the fact that many cases of Cushing's syndrome have been reported in which either no basophilic hypophyseal adenoma was found or the pathologic lesion proved to be extrahypophyseal. It has long been known that a disease closely similar to if not identical with Cushing's syndrome may be caused by neoplasms of the supra-renal cortex and this has been termed the "supra-renal cortical syndrome."[10]

In the face of this confusion as to pathogenesis the observations of Crooke,[11] subsequently confirmed by Rasmussen,[12] seem to provide a possible solution to the problem and are compatible with the diagnostic classification of Cushing's syndrome as "pituitary basophilism." In a histopathologic study of cases of this syndrome, Crooke discovered a specific hyaline change in the cytoplasm of the basophil cells of the adenohypophysis and he concluded that this hyaline change "is probably an expression of altered physiologic activity." Both Crooke and Rasmussen have found this specific alteration of the cytoplasm of the basophil cells in all cases of Cushing's syndrome including those where no basophilic adenoma was demonstrable or where extrahypophyseal lesions existed. It will be noted that in our case, although adenomatous lesions existed in the suprarenal cortex, Crooke's changes were observed in the basophil cells of the adenohypophysis.

The occurrence of mental symptoms seems to have received little attention in most articles dealing with clinical descriptions of Cushing's syndrome. However, Jonas[2] in his extensive review of the subject reports the incidence of mental disturbances to be 20.5 per cent. He states that in mild cases one finds such symptoms as mental dullness, forgetfulness and depression, while in more severe cases there may also occur hallucinations and cyclic psychoses.

We wish to draw particular attention to the character of the psychiatric disorder which developed in our case and which, it would seem, not uncommonly has been noted in cases of Cushing's syndrome. Mental depression, either psychotic or borderline in type, has been described not infrequently.[13] It is difficult to judge the incidence of depression when presumably it has been milder in degree but we would surmise that it has occurred more commonly than generally supposed. Brief mention of this symptom has been made in at least seven additional cases.[14] In general the mental depression has been considered to be of a reactive type and attributed to the psychically traumatic effect of the somatic alterations which appear in the course of the disease. Exacerbations and remissions of physical signs as a result of treatment have been reported as being accompanied by corresponding changes in the mental status and this has been considered to be supportive evidence of the above hypothesis. On the other hand, Maclay and Stokes [5] have justifiably considered the possibility that there may be a more specific pathogenic relationship between the psychic alterations and the type of endocrine dysfunction that exists in Cushing's syndrome. We wish to direct attention still further to this problem and to emphasize the possibility that there may be a more or less specific form of psychiatric disorder associated with Cushing's syndrome. The mental depression observed in these cases may bear a close relationship to the depressive phase of manic-depressive psychosis and to involutional melancholia. As regards the latter psychiatric disorder, Raab [15] has already pointed out the resemblance between many of the clinical features in Cushing's syndrome and those not uncommonly seen in advancing years of adult life. This resemblance would appear to be more striking for the involutional period in woman.

From a study of our case, it is readily appreciated how difficult it may be to differentiate a psychiatric disorder associated with Cushing's syndrome from a manic depressive psychosis. This is especially true when mental symptoms appear early in the course of the disease and partially mask the physical manifestations. The latter have usually antedated the former by a relatively long period of time so that the difficulty in making a diagnosis, which was noted in our case, has been uncommon. It is important, however, to

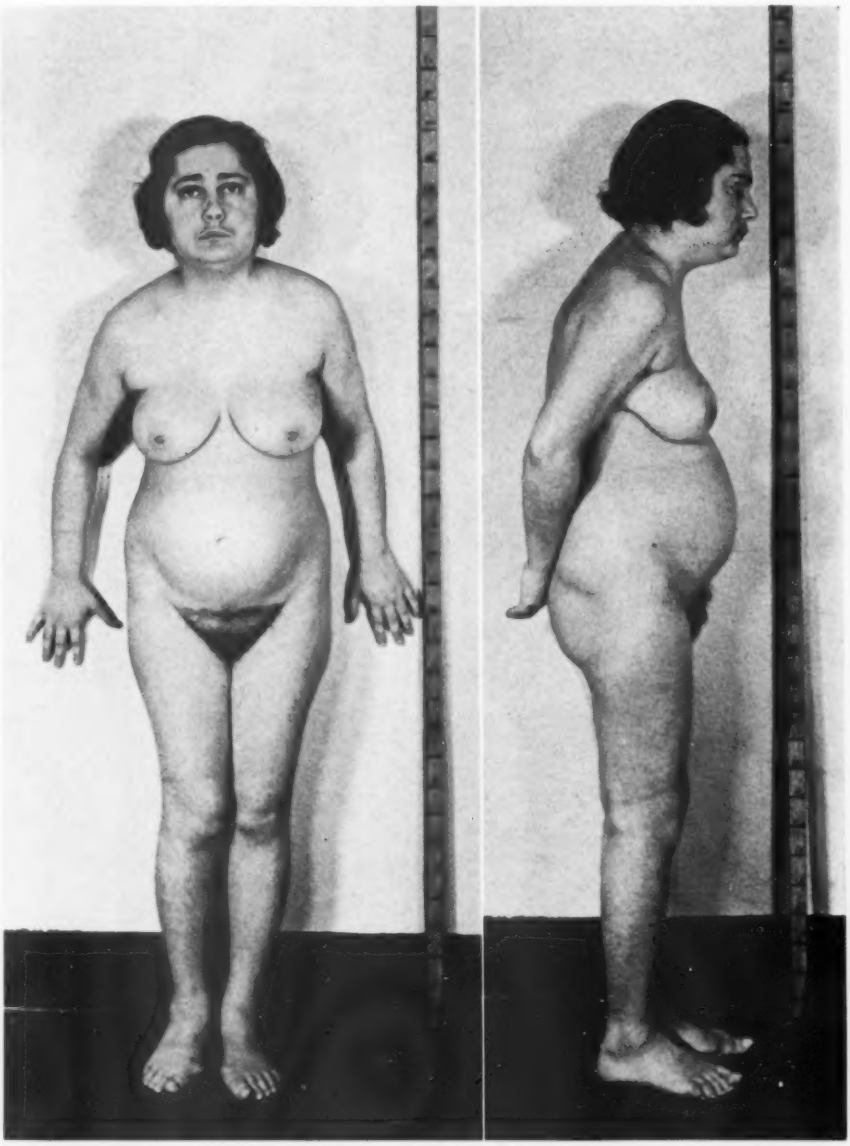


FIG. 1 A. FIG. 1 B.

FIG. 1.—Anterior and lateral views showing such characteristic features as "moon-like" facies, axial obesity, kyphosis and protuberant abdomen.

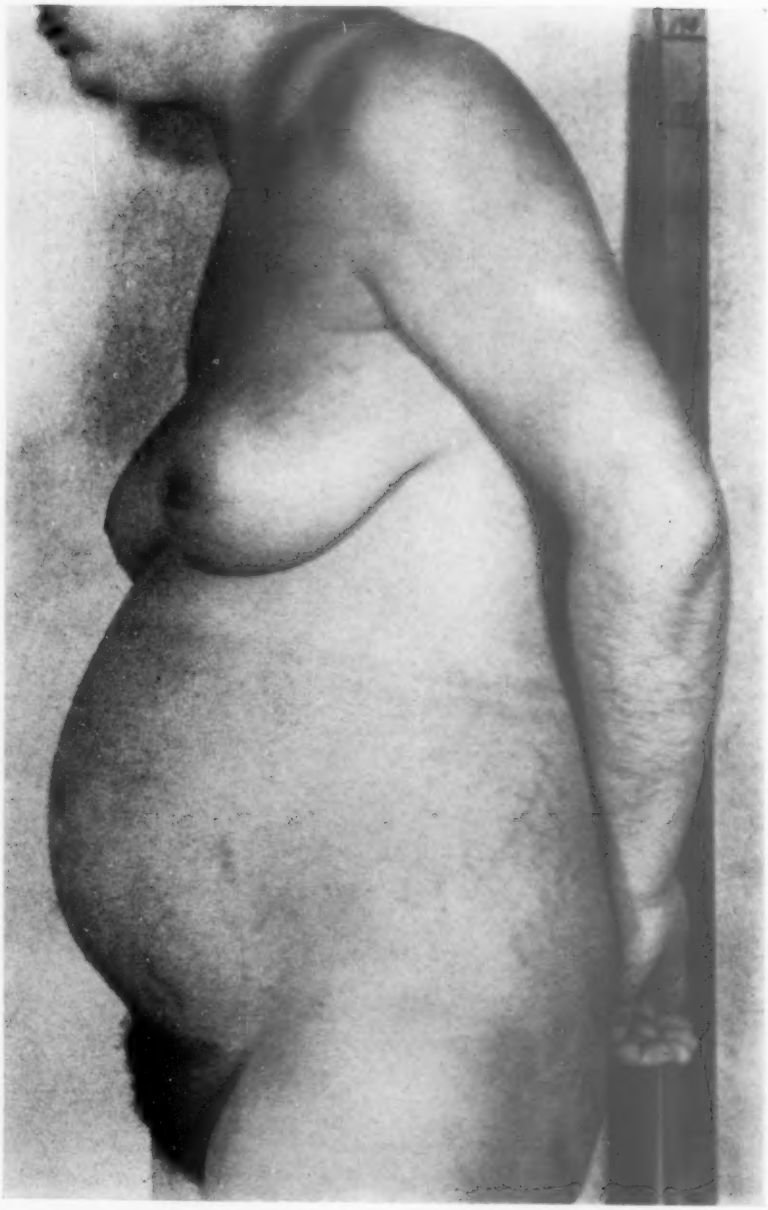


FIG. 2.—Lateral view showing blue striæ on antero-lateral surface of abdomen.

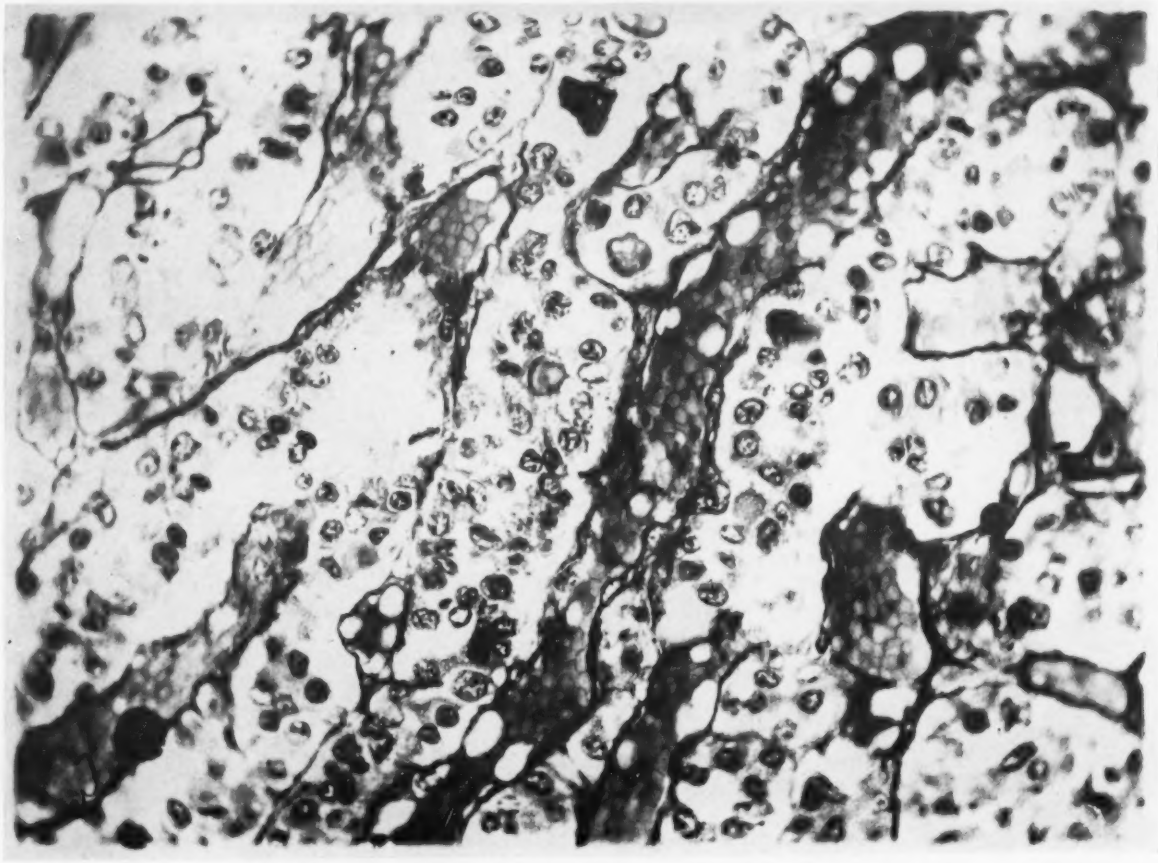


FIG. 3.—Section of adenohypophysis. Note the hyaline changes in basophil cells. Mallory stain (modified).

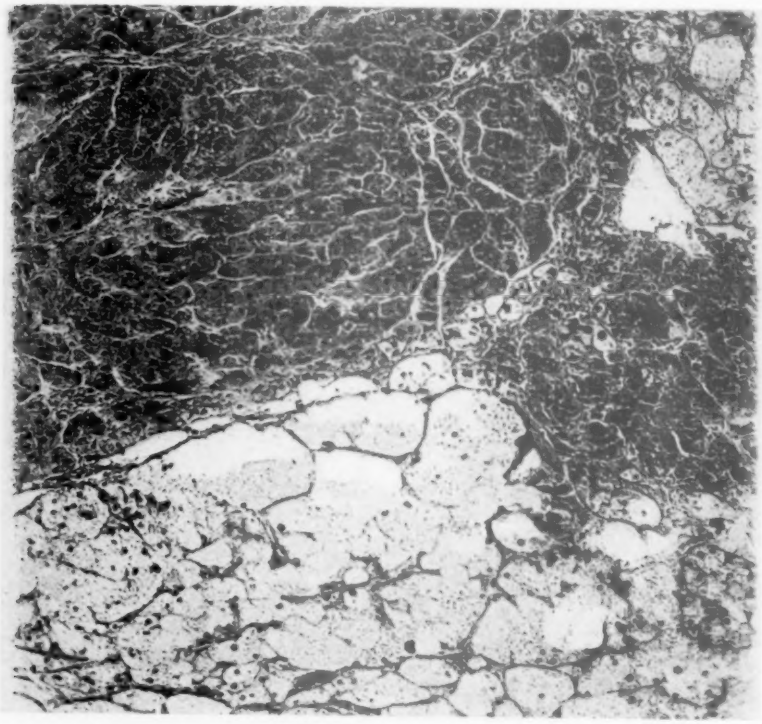


FIG. 4.—Section of left adrenal gland showing neoplastic changes. Notice the large, foamy cells. Hematoxylin and eosin stain.



FIG. 5.—Section showing encephalomalacia in white matter of right frontal lobe. Hematoxylin and eosin stain.

realize that such errors in diagnosis may conceivably be more frequent and may remain uncorrected.

It is well known that endocrine manifestations are often prominent features in psychoses and they have been the subject of considerable investigation.[16, 17] These phenomena have sometimes been considered as indicative of an underlying specific somatic disease in these psychoses. The association of mental depression with Cushing's syndrome in a more than coincidental manner lends support to such a hypothesis. In addition, one may consider the concomitant fluctuations in the endocrine and psychiatric aspects of this disease following treatment, well shown in the cases described by Dattner,[13] as not militating against such a hypothesis but rather as favoring it.

The varied symptoms in Cushing's syndrome as yet have had no adequate explanation offered for their concurrence and mechanism of production. This may be said to be even more true for the mental symptoms than for the many physical manifestations observed in this disease. Whether the former are directly attributable to hypophyseal dysfunction or may be a product of associated dysfunction of other endocrine glands cannot be definitely determined at this time. It is well to remember that similar mental symptoms have been reported in cases having the "cortico-adrenal syndrome," [18] thus again emphasizing the fact that Cushing's syndrome is a polyglandular disease. In this connection one may reassert that "it is not easy to disentangle the specific contribution of the disordered action of the gonads to a pluriglandular syndrome." [19] A striking example of the influence of the menstrual cycle upon the psyche has been observed by one of us.[20]

A precise etiology for Cushing's syndrome which will satisfactorily explain its development in all cases as yet remains undetermined. Neither basophilic adenomas of the hypophysis, nor neoplasms of the supra-renal cortex have been constantly observed. On the other hand, the apparently constant cell changes noted by Crooke as yet have not been ascribed to any specific etiologic factor in non-neoplastic cases. In elucidating the latter question, our case seems to offer a clue. It will be recalled that this patient had evidence of a neurologic disorder which developed concurrently with the endocrine disorder and, at first, passed unnoticed, as did the latter. The neurologic signs were similar to those frequently

observed in patients who have encephalitic parkinsonism. Neuropathologic study was insufficient to conclusively determine the presence or absence of evidence of chronic encephalitis. There exists the possibility that the Cushing's syndrome in our case is due to an encephalitic neuroendocrine affection. The involvement of the hypophysis in a disease process which is known to have a predilection for the hypothalamus is not a wholly unreasonable hypothesis. However, we do not believe that encephalitis is a common etiologic factor in Cushing's syndrome but merely suggest it for unusual cases in which the etiologic factors are especially obscure.

SUMMARY AND CONCLUSIONS.

1. The clinical and post-mortem findings in an unusual case of Cushing's syndrome are described. There existed in this patient a depressive psychosis and an extrapyramidal neurologic disorder, the overt development of both being associated temporally with that of the specific endocrine disorder. It is suggested that the etiology of the complex clinical picture observed in this case may be a neuroendocrine disorder on an encephalitic basis.

2. A review of the literature reveals that mental symptoms not infrequently occur in Cushing's syndrome and that mental depression of varying degree has been a prominent feature in these psychiatric disorders. The mental symptoms may be a direct manifestation of the underlying specific endocrinopathy although the mechanism of their production remains obscure. The possibility that the form of psychiatric disorder, namely depression, in these cases bears a close relationship to manic depressive and involutional psychoses is pointed out.

3. Patients hospitalized with depressive psychoses should be carefully examined for evidence of Cushing's syndrome.

BIBLIOGRAPHY.

1. Cushing, H.: The basophil adenomas of the pituitary body and their clinical manifestations (pituitary basophilism). Bull. J. Hopk. Hosp., 50: 137, 1932.
2. Jonas, V.: Morbus Cushing. Cas. Lek. Cesk, 74: 1313; 1463, 1935.
3. Pardee, I.: Pituitary basophilism of Cushing-Syndrome of the basophilic adenoma. Bull. Neur. Inst. N. Y., 6: 183, 1937.

4. Kessel, F. K.: Morbus Cushing. Ein Uberblick uber Klinik und Kasuistik des basophilen Hypophysenadenoms. Erg. inn. Med. u. Kniderheilk., 50: 620, 1936.
5. Maclay, W. S., and Stokes, A. B.: Mental disorder in Cushing's syndrome. J. Neurol. and Psychiat., 1: 110, 1938.
6. Langeron, L.: Observation clinique rentrant probablement dans le cadre du basophilisme hypophysaire de Cushing. Bull. Soc. med. Hop., Paris, 3 ser., 51: 1116, 1935.
7. Giraud, G., Margarot, J., and Rimbaud, P.: Maladie de Cushing avec paraplegie spasmodique. Presse med., 43: 841, 1935.
8. Marinescu, G., Draganescu, S., and Banu, E.: Ueber einige Versuche bei Cushingscher Krankheit Endocr. Ginec. si Obstet., 2: 182, 1937. Ref. Zbl. Neur., 87: 658, 1938.
9. Cushing, H.: "Dyspituitarism": Twenty years later. Arch. Int. Med., 51: 487, 1933.
10. Walters, W., Wilder, R. M., and Kepler, E. J.: The suprarenal cortical syndrome, with presentation of ten cases. Ann. Surg., 100: 670, 1934.
11. Crooke, A. C.: A change in the basophil cells of the pituitary gland common to conditions which exhibit the syndrome attributed to basophil adenoma. J. Path. and Bact., 41: 339, 1935.
12. Rasmussen, A. T.: The relation of the basophilic cells of the human hypophysis to blood pressure. Endocrinology, 20: 673, 1936.
13. (a) Launois, P. E., and Cleret, M.: Le syndrome hypophysaire adiposogenital. Gaz. des Hop., 83: 57; 83, 1910. (b) Dattner, B.: Zwei Falle von Cushingschem Adenom. Wien. Klin. Wchnschr., 1: 809; 2: 1434, 1935. (c) Klein, F., and Wagnerova-Hatrikova, H.: Prispevok k patogenezi "Morbus Cushing." Rev. v Neurol. a Psychiat,. 32: 49; 65, 1935. Ref. Zbl. Neurol., 77: 581, 1935. (d) Fuller, C. J., and Russell, D. S.: Chromophobe adenoma associated with Cushing's syndrome. Lancet, 2: 181, 1936. (e) Schmidt, C.: Beitrag zum Cushingschen Syndrom. Klin. Wchnschr., 2: 1437, 1936. (f) Bland, P. B., and Goldstein, L.: Pituitary basophilism: Review of forty-two verified cases with report of personal case. Surg., Gynec. and Obstet., 65: 644, 1937. (g) Urban, H.: Ein Fall von "Basophilismus" (Morbus Cushing). Wien, klin. Wchnschr., 2: 1122, 1937. (h) Maclay and Stokes.[5]
14. (a) Bauer, T., and Wassing, H.: Zur Frage der Adipositas hypophysaren (Basophiles Adenom der Hypophyse.) Wien, klin. Wchnschr., 2: 1236, 1913. (b) Cushing.[9] (c) Rutishauser, E.: Osteoporotische Fettsucht (pituitary basophilism). Deutsche Arch. f. klin. Med., 175: 640, 1933. (d) Jamin, F.: Die hypophysare Plethora (Cushingsche Krankheit: pituitary basophilism). Munch. med. Wchnschr., 2: 1045; 1085, 1934. (e) Crile, G., Turner, H., and McCullagh, P.: Polyglandular disease. N. Y. St. J. Med., 36: 475, 1936. (f) Schilling, V.: Striae distensae als hypophysares Symptom bei basophilem Vorderlappenadenom (Cushingschem

Syndrome) und bei Arachnodaktylie (Marfanschem Symptomkomplex) mit Hypophysentumor. Med. Welt., 10:183; 219; 259, 1936. (g) Page, A. P. M., Roberts, L. V., and Biggart, J. H.: Cushing's syndrome in a mulatto. Lancet, 2:625, 1937.

15. Raab, W.: Analogien zwischen gewissen Alterserscheinungen und der Cushing'schen Krankheit. Wien. klin. Wschr., 49:112, 1936.
16. Harris, M. M., Horwitz, W. A., and Katz, S. E.: Endocrine studies in mental cases. Psych. Quart., 8:655, 1934.
17. Hutton, J. H., and Steinberg, D. L.: Endocrinopathies and psychoses. J. Ment. Sc., 82:773, 1936.
18. Cahill, G. F., Loeb, R. F., Kurzrok, R., Stout, A. P., and Smith, F. M.: Adrenal cortical tumors. Surg., Gynec. & Obstet., 62:287, 1936.
19. Campbell, C. M.: Psychical disturbances in the diseases of the glands of internal secretion. Endocrinology, 9:201, 1925.
20. Horwitz, W. A., and Harris, M. M.: Study of a case of cyclic psychic disturbances associated with menstruation. Am. J. Psychiat., 92:1403, 1936.

HEREDITARY AND ENVIRONMENTAL FACTORS IN THE CAUSATION OF MANIC-DEPRESSIVE PSYCHOSES AND DEMENTIA PRÆCOX *

By HORATIO M. POLLOCK AND BENJAMIN MALZBERG

In 1928 the Laura Spelman Rockefeller Memorial made a grant to the State Charities Aid Association for a series of investigations into mental disease in New York State. The New York State Department of Mental Hygiene collaborated with the Association in the investigations made. One line of research dealt with the pre-hospital histories of patients with mental disease. It was felt that such a study might clarify certain problems connected with the causation of mental disease. Social workers were therefore assigned to the Utica State Hospital and during the three years from 1928 to 1930 they conducted thorough investigations into the histories of two groups of patients admitted to that hospital. One group consisted of 155 first admissions with manic-depressive psychoses, of whom 60 were males and 95 females. The other consisted of 175 first admissions with dementia præcox, of whom 92 were males and 83 females. The patients were chosen for investigation in a random manner, there being no selective factor other than the existence of a well-defined psychosis. The Utica State Hospital was the locus of the investigation because its admission district includes a population easily accessible to investigation.

Information was obtained with respect to the mental and physical health of parents, grandparents, uncles, aunts and siblings of the patients, special attention being directed to the occurrence of mental and nervous diseases, mental defect, alcoholism and criminality in the family stock. The information was gathered, whenever possible, through the direct testimony of the patient, close

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

For a fuller discussion of the subject, see the book entitled, "Hereditary and Environmental Factors in the Causation of Manic-Depressive Psychoses and Dementia Præcox," by Horatio M. Pollock, Benjamin Malzberg and Raymond G. Fuller. State Hospitals Press, Utica, N. Y., 1939.

relatives and intimate associates. Interviews were held by the social workers at the hospital and in the field. All statements concerning the patient and his family were verified, whenever possible, through the direct testimony of first-hand sources; mere hearsay was avoided. Data were also gathered with respect to the developmental histories of the patients prior to the onset of the mental disorder. These also related to the health of the patient at stated periods in his life. There was a description of the home life and of the relations between the patient and members of the household and of the character of the community in which he lived. The histories also included the school record, the occupational record, sex and marital experiences, etc.

In analyzing this mass of data, we began with the thesis that there is no known organic basis for either a manic-depressive psychosis or dementia præcox. Innumerable conditions have at one time or another been declared to be causative factors in these diseases, but laboratory investigations have always failed to confirm the claims. Our investigations were therefore directed to the study of two sets of factors. One is found in the familial histories of the patients; the other deals with the effects of certain biopsychological experiences upon the individual.

We start first with the family background. The analysis of such data has proceeded along two lines. According to one method, we assume the fact of inheritance and we attempt to fit the observations to a specified law of heredity. Agreement of the two is considered proof of inheritance. According to the second method we assume no law of heredity, but we search for differences in the incidence of tainting factors among the families of the mentally diseased and among a corresponding healthy population. If significant differences are found, it is assumed, that, other things being equal, they are due to the force of heredity.

The first method has proceeded along the lines of Mendelian analysis. Assuming, for example, that mental disease is a recessive character, the absence of mental disease being considered dominant, the parental generations are classified according to the presence or absence of mental disease. Now, on the assumption of Mendelian inheritance none of the parents of our patients should be classified as DD, for from the union of DD with DR or RR no diseased offspring should result. Parents who are outwardly normal must

therefore be assumed to be of type DR. Mentally diseased parents must be considered of type RR. We must therefore consider the offspring resulting from the mating of DR with DR, of DR with RR, and of RR with RR. The first type should result in 25 per cent of the offspring being mentally diseased. From the second

TABLE 1.

OFFSPRING OF PARENTS OF ASSUMED TYPE DR × DR, CLASSIFIED BY AGE AT DEATH OR AGE AT TIME OF ADMISSION OF THE PROBAND TO THE UTICA STATE HOSPITAL.

Age (years).	Deceased siblings.*		Living siblings.*	
	Number of cases.	Number with mental disorder.	Number of cases.	Number with mental disorder.
Under 10....	57		1	
10–14.....	7		6	
15–19.....	4		11	
20–24.....	8		26	
25–29.....	3	1 general paresis	31	1 manic-depressive psychosis 1 dementia præcox
30–34.....	2		36	1 manic-depressive psychosis 1 unknown psychosis 1 psychopath
35–39.....	7	1 suicide	40	
40–44.....	5		38	1 nervous
45–49.....	4	1 general paresis	28	3 nervous 1 alcoholic
50–54.....	4	1 manic-depressive psychosis	37	1 feebleminded 1 nervous
55–59.....	2		22	2 nervous
60–64.....	..		13	
65–69.....	1		14	
70–74.....	..		5	
75–79.....	2		2	1 manic-depressive psychosis
Total...	106	4	310	15

* Exclusive of proband.

type of mating there should arise 50 per cent of diseased children. Progeny resulting from the third type of mating should all be mentally diseased.

Table 1 is based upon the histories of 82 patients with manic-depressive psychoses, and describes the siblings in those cases in which the parents were classified as DR × DR. In these families there should be mentally diseased offspring in the ratio of 1 dis-

eased to 3 normal. There were 416 children, exclusive of the probands. On Mendelian theory there should therefore have been 104 ± 6.0 diseased children. By defining mental disease as widely as possible, and by including such conditions as suicide, nervousness, depression, etc., as indications of a neuropathic taint, we obtain a total of 19 cases of mental disease. This is only 22 per cent of the minimum required on the hypothesis that mental disease is a recessive unit character. As age is an important complicating factor, several corrections were made, but in no case was it possible to obtain agreement with the requirements of Mendelian theory.

The hypothesis of DR × RR was tested in connection with 48 families. There were 230 siblings, exclusive of the probands. Half, or 115, should have been mentally diseased. Actually there were only 31 such cases, including 1 case of general paresis, 6 of manic-depressive psychoses, 1 of involution melancholia, 4 of dementia præcox, 2 unknown psychoses, 1 case of epilepsy, 7 cases of nervousness, 2 suicides, and 7 cases of alcoholism. Corrections for age did not bring about any significant changes in the results.

Finally, we may consider 8 families in which the matings were of the variety RR × RR. In 3 of these, the matings may be considered strictly of this type, since both parents were either feebleminded or mentally diseased. In the remaining 5 families, the matings represented unions of 2 nervous individuals, of a nervous individual with an alcoholic, and of an alcoholic with an alcoholic. On the assumption that these are of the same value from the Mendelian point of view, all the offspring should have been mentally diseased. There were 35 siblings, exclusive of the probands. Of these, only 9 were affected, of whom 8 were feebleminded, and 1 was a case of manic-depressive psychosis. The total is too small for significant comparison, but it is noteworthy, nevertheless, that once again fact and theory were not in agreement.

We turn next to dementia præcox. Table 2 considers 101 families in which both parents were described as normal and who must therefore be classified as latent dominants. In these families there were 458 siblings, exclusive of the 101 probands. There were 26 affected siblings, including 6 with psychoses, 4 alcoholics, 7 nervous, 2 suicides, 2 neurotic, 4 feebleminded and 1 epileptic. There should have been 114.5 ± 6.3 cases of affected siblings.

The actual total was thus far less than the minimum expected on the basis of random sampling.

In 47 families the matings were considered of the type DR × RR. The offspring totaled 165, exclusive of the probands. Of the siblings, 16 were classified as mentally tainted. On a Mendelian

TABLE 2.

OFFSPRING OF PARENTS OF ASSUMED TYPE DR × DR, CLASSIFIED BY AGE AT DEATH OR AGE AT TIME OF ADMISSION OF THE PROBAND TO THE UTICA STATE HOSPITAL.

Age (years).	Deceased siblings.*		Living siblings.*	
	Number of cases.	Number with mental disorder.	Number of cases.	Number with mental disorder.
Under 10....	50		9	
10–14.....	5		19	1 feebleminded
15–19.....	2		31	1 nervous
20–24.....	11		31	
25–29.....	8	1 alcoholic	44	
30–34.....	6	1 nervous	41	1 neurotic 1 nervous
35–39.....	2	1 suicide	56	1 alcoholic 3 feebleminded 1 nervous 1 unknown psychosis
40–44.....	3		42	2 nervous 2 alcoholic 1 epileptic
45–49.....	1		35	4 unknown psychoses
50–54.....	3	1 neurotic	24	
55–59.....	2	1 suicide	10	
60–64.....	2		5	
65–69.....	..		6	1 nervous 1 dementia præcox
70–74.....	..		2	
75–79.....	1		2	
Unknown....	5†		..	
Total...	101	5	357	21

* Exclusive of proband.
† All under 50 years of age.

basis there should have been 82.5 ± 4.3 such cases. Clearly, there is no agreement between fact and theory.

Finally, there was a series of 14 families in which both parents were tainted. In these families there were 66 children, exclusive of the probands. Among them were 5 cases of feeblemindedness, 1 of dementia præcox, and 1 of nervousness, a total of 7. According to the theory, all 66 should have been mentally diseased.

We conclude therefore that neither dementia præcox nor manic-depressive psychoses appears in frequencies that are in accord with the requirements of simple Mendelian inheritance. It has been suggested by several investigators that the hypothesis of dihybridism would give an adequate explanation of the frequency of certain types of mental diseases. But if we complicate hypotheses, it may always be possible to obtain concordance between fact and theory. However, such agreement cannot be the only criterion. One must seek underlying reasons justifying the application of a particular formula.

Search for such justification, however, only increases the distrust in the possibility of applying Mendelian laws to the inheritance of mental disease. In the first place, there should be clear-cut clinical entities with respect to the several types of mental disorders. We should, for example, study the offspring of parents who were dominant or recessive with respect to manic-depressive psychoses or dementia præcox. Among the offspring, there should be found individuals affected with such psychoses in ratios required by Mendelian theory. We have already shown that this requirement could not be met in our family data. In the second place, the development of the mental disorders should be independent of external circumstances. If this is not the case, we cannot hope to obtain the correct total of recessives, for it is conceivable that under a favorable environment no one might develop a mental disease. How different is the case with such characters as eye color, for example, where the trait appears in a constant proportion among the offspring, independently of environmental changes other than those of a pathological order, that may affect the entire organism. In the third place, there should be no differential mortality between the several genotypes. If those with the diathesis for a manic-depressive psychosis, for example, have higher rates of infant and child mortality than the normal children, then the frequency of appearance of the disease would be less than the minimum requirements of Mendelian theory. Finally, are we justified in assuming that a mental disease may be treated as a unit, the transmission of which through inheritance is independent of the transmission of other characters? Is it not at least conceivable that there is correlation instead of independence of mental traits, and that the frequency

of a mental disorder is therefore in part a function of the nature of the organism as a whole?

Thus far we have failed to establish the existence of a particular form of inheritance, but this does not necessitate the inference that heredity is not a factor in the origin and transmission of mental disorders. We must observe the frequencies of mental disease in the families of the patients, and compare them with frequencies in other populations. The first investigations of this type were reported by Koller and Diem. Both found higher percentages of individuals with so-called tainting factors in the families of patients with mental disease than among the families of healthy individuals, though the differences were smaller than had been anticipated. In recent years Rüdin and his associates in Munich have attempted to arrive at samples of the normal population through a consideration of the spouses of patients with general paresis or with cerebral arteriosclerosis. The selection of the spouses was made on the assumption that, since these disorders are presumably on a non-hereditary basis, the presence of the disease in husband or wife will not have been of genetic significance in the inheritance of the marital partner. However, it is unlikely that a satisfactory sample of a normal population (adequate in size and randomness of choice) can be secured for the purpose of comparison with the families of mental patients. The selective factors operating to vitiate the random choice of samples of a general population are extremely subtle, and one can hardly ever be certain that such factors have not entered into the choice of a numerically limited sample.

We may, however, proceed in the following manner. By means of our family histories we are able to count the total number of relatives of specified degree, and the corresponding numbers of affected individuals. The proportion of affected individuals may then be compared with the corresponding proportion for the general population. If each class formed only a random sample of the general population, the proportions of affected individuals should not differ sensibly from that of the general population.

In such an analysis it is necessary to know the expectation of mental disease. Under given conditions of mortality it is required to know how many in a given generation will develop mental dis-

ease in the course of a life time. This expectation has been determined for New York State, where it was found that in 1920 males had a chance of 4.7 in 100 of developing a mental disorder, females 4.4 in 100. On the theory of random sampling, approximately 4.7 per cent of male populations and 4.4 per cent of female populations should therefore develop mental disease. These may be called "crude" expectations for the families of the patients, however, since the assumptions underlying the expectations for 1920 cannot be applied directly to those of the older generations. The expectations for 1920 are the result of rates of mental disease and rates of mortality in that year. Both sets of rates have been subject to a secular trend. The rate of mental disease has been increasing from decade to decade and general mortality rates have been decreasing. Twenty and forty years ago admission rates were decidedly lower than they are today, whereas death rates were materially higher. Consequently, the expectation of mental disease in the several older generations must have been less than 4.7 per cent and 4.4 per cent, for males and females, respectively. The grand-parental generation included the period from 1820 to 1900; the parental generation and the collaterals included a period from approximately 1850 to 1930. The siblings embrace the period beginning at about 1890. Considering the changes in death rates and rates of mental disease it appears conservative to estimate, in comparison with the expectations of 1920, an average expectation of 4.0 for males and 3.5 for females throughout the earlier periods. Applications of such corrected expectations to the families of the patients will give figures legitimately comparable with the recorded findings.

The principal data are shown in Tables 3 and 4 for the families of the patients with manic-depressive psychoses. It will be noted that there were 2572 relatives in the histories. No details were recorded in 195 cases, leaving a total of 2377 with recorded histories. Among the latter there were 58 with mental diseases.

The total of recorded psychoses is undoubtedly too low. This is due in part to the fact that in each degree of relationship there are individuals still exposed to the chance of developing a mental disorder. We may compensate for this by applying the appropriate expectations of mental disease. We cannot compensate, however, for the incomplete histories of many of the patients. Not all were native-born with families easily accessible to investigation by the

TABLE 3.

FREQUENCY OF MENTAL DISORDERS AMONG THE RELATIVES OF 155 PATIENTS WITH MANIC-DEPRESSIVE PSYCHOSES.

Relationship.	Total number of relatives.	No details recorded.	Number of histories recorded.	Total without mental disease or other specified defect.	Totals.			Psychoses.										Other defects.					
					Number with psychoses.	Number with other defects.	Total defects or diseases recorded.*	Senile psychosis.	With cerebral arteriosclerosis.	General paresis.	Alcoholic psychoses.	Manic-depressive.	Involution melancholia.	Dementia præcox.	Epileptic psychoses.	Psychoneuroses.	Undiagnosed psychoses.	Neurotic traits.	Mental deficiency.	Epilepsy.	Alcoholism.	Drug addiction.	Suicide.
Father	155	1	154	115	3	36	41	..	..	..	..	3	..	..	..	..	..	13	1	1	19	..	4
Mother	155	1	154	122	6	26	36	..	1	..	..	1	..	2	..	..	2	25	2	..	2	1	..
Paternal grandfather	155	34	121	115	3	3	9	..	..	..	..	1	..	..	..	..	2	..	..	..	5	..	1
Paternal grandmother	155	37	118	115	1	2	3	..	..	..	..	..	..	..	..	..	1	2	..	..	..	..	..
Maternal grandfather	155	29	126	123	1	2	3	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	2
Maternal grandmother	155	26	129	125	1	3	4	..	..	..	..	..	..	..	..	..	1	3	..	..	..	..	..
Paternal uncles	229	17	212	201	5	6	11	1	..	..	1	..	..	..	..	..	3	..	..	1	5	..	..
Paternal aunts	187	12	175	171	2	2	4	..	..	..	..	..	..	..	..	..	2	1	..	1	..	..	..
Maternal uncles	228	10	218	210	2	6	9	..	..	..	..	1	..	1	..	..	..	..	..	..	6	..	1
Maternal aunts	231	6	225	217	5	3	8	..	..	..	..	1	..	..	..	..	4	2	..	1	..	..	..
Brothers	380	11	369	333	15	21	36	..	..	3	1	3	..	5	1	..	2	7	5	..	7	..	2
Sisters	387	11	376	346	14	16	32	..	..	1	..	7	1	2	..	1	2	11	3	1	..	..	3
Total	2,572	195	2,377	2,193	58	126	196	1	1	4	2	17	1	10	1	1	20	64	11	5	44	1	13

* An individual may have more than one defect.

field workers. Many histories were therefore based on incomplete evidence, and it is not beyond the bounds of reasonable probability that they include individuals who should be added to the total cases of mental disease. These errors will be apparent with respect to the ancestral generations, where it is shown in the accompanying table that the expected cases of mental disease are in excess of the actual plus anticipated cases, except for the mothers. Among the siblings, however, for whom the information is much more reliable, we find a total of 369 brothers and 376 sisters with known his-

TABLE 4.

COMPARISON OF EXPECTED WITH CORRECTED TOTAL OF ACTUAL CASES OF MENTAL DISEASE IN THE FAMILIES OF 155 PATIENTS WITH MANIC-DEPRESSIVE PSYCHOSES.

Relationship.	Number with known histories.	Expected cases of mental disease.	Actual cases of mental disease.	Anticipated cases.
Father	154	6.2	3	1.6
Mother	154	5.4	6	1.8
Paternal grandfather	121	4.9	3	...
Paternal grandmother	118	4.1	1	0.2
Maternal grandfather	126	5.1	1	...
Maternal grandmother	129	4.6	1	0.2
Paternal uncles	212	8.5	5	2.1
Paternal aunts	175	6.1	2	2.1
Maternal uncles	218	8.7	2	3.0
Maternal aunts	225	7.9	5	2.7
Brothers	369	14.8	15	11.0
Sisters	376	13.2	14	11.2
Total	2,377	89.5	58	35.9

tories. Among the former there were 15 cases of mental disease, and a further anticipation of 11 cases, a total of 26, compared with an expectation of only 14.8. Among the sisters there were 14 known cases of mental diseases, and an anticipation of 11.2 cases, making a total of 25.2, compared with a complete expectation of only 13.2 cases. Clearly, among the siblings, for whom the data are most adequate, there is an excess of mental disorders over the total expected in a random sample.

There were 39 families in which a father had a mental disease or some defect usually considered a tainting factor. In these 39 families there were, exclusive of the probands, 112 brothers and 85 sisters with adequate histories. The brothers included 4 known

cases of mental disease, and an anticipation of 2.7 cases, a total of 6.7, compared with a complete expectation of 4.5 cases. The sisters included 4 known cases of mental disease, with an anticipation of 2.0 additional cases, a total of 6.0, compared with a complete expectation of 3.0.

Thirty-two mothers were described as tainted. In these families there were 80 brothers and 65 sisters with adequate histories. Among the brothers there were 6 known cases of mental disease, with an anticipation of 2.2 cases, a total of 8.2, compared with a complete expectation of 3.2. Among the sisters there were 5 known cases of mental disease, with an anticipation of 2.0 cases, a total of 7.0, compared with a complete expectation of only 2.3 cases.

There were 9 families in which both parents were tainted. In these families the siblings were too few in number, however, to give significant results. There were 24 brothers, among whom there was 1 case of mental disease, and an anticipation of 0.4, a total of 1.4, compared with an expectation of 1.0. There were 19 sisters who included 1 case of mental disease and an anticipation of 0.4, a total of 1.4, compared with an expectation of 0.7 cases.

Finally, we may consider the frequency of manic-depressive psychoses among the siblings of patients with manic-depressive psychoses. The latter had 369 brothers and 376 sisters. Among the former there were 3 cases of manic-depressive psychoses, and an anticipation of 1.0 case, a total of 4.0, compared with a complete expectation of 1.8 cases. Among the sisters there were 7 cases of manic-depressive psychoses, and an anticipation of 2.9 cases, a total of 9.9, compared with an expectation of only 3.4 cases.

We shall next consider the incidence of mental disease among the families of the patients with dementia præcox. The necessary data are included in Tables 5 and 6.

The family histories included 2753 relatives, exclusive of the probands. The details were insufficient in 238 cases, leaving a total of 2515 individuals with adequately recorded histories. Among the latter were 74 psychotic individuals. With proper consideration of the age factor, we should add 47.5 cases, making a total of 121.5, compared with a complete expectation of only 94.6. Because of their accessibility to investigation, the results for the siblings were more reliable. Among the brothers there were 11 with known

TABLE 5.

FREQUENCY OF MENTAL DISORDERS AMONG THE RELATIVES OF 175 PATIENTS WITH DEMENTIA PRÆCOX.

Relationship.	Total number of relatives.	No details recorded.	Number of histories recorded.	Total without mental disease or other specified defect.	Totals.		Psychoses.														Other defects.			
					Number of psychoses.	Number with other defects.	Total defects or diseases recorded.*	Senile psychoses.	With cerebral arteriosclerosis.	General paresis.	Alcoholic psychosis.	With other somatic diseases.	Manic-depressive.	Involution melancholia.	Dementia præcox.	Paranoia or paranoic conditions.	Epileptic psychoses.	Psychoneuroses and neuroses.	With psychopathic personality.	With mental deficiency.	Undiagnosed psychoses.	Neurotic traits.	Mental deficiency.	Epilepsy.	Alcoholism.	Suicide.
Father	175	1	174	133	4	37	47	..	..	..	..	..	2	..	2	..	..	..	..	..	..	11	1	..	30	1
Mother	175	1	174	128	13	33	46	1	..	..	..	1	3	..	4	..	..	..	..	..	4	32	1	..	..	..
Paternal grandfather	175	40	135	123	2	10	14	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	3	..	8	1
Paternal grandmother	175	40	135	127	5	3	8	1	..	..	..	..	1	..	..	..	..	..	..	..	3	3	..	..	..	..
Maternal grandfather	175	41	134	122	5	7	13	1	..	1	..	..	1	..	..	..	..	..	..	..	2	1	..	..	6	1
Maternal grandmother	175	38	137	136	1	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..
Paternal uncles	259	22	237	215	6	16	26	..	1	..	1	1	..	..	2	..	..	..	..	..	1	2	..	16	2	..
Paternal aunts	211	11	200	189	4	7	11	..	..	..	..	..	1	..	2	..	..	..	..	1	..	6	1	..	..	..
Maternal uncles	254	12	242	223	7	12	22	..	..	..	..	..	4	..	..	1	..	..	..	1	1	1	3	..	9	2
Maternal aunts	245	14	231	222	5	4	10	..	..	..	..	..	1	..	..	..	..	1	..	..	3	3	..	1	..	1
Brothers	381	7	374	346	11	17	28	1	1	..	..	..	..	..	4	..	..	..	..	1	4	4	5	..	6	2
Sisters	353	11	342	309	11	22	34	1	2	..	..	..	2	..	3	1	..	..	..	..	2	16	4	1	2	..
Total	2,753	238	2,515	2,273	74	168	260	5	4	1	1	2	16	1	17	2	1	1	1	2	20	82	15	2	77	10

* An individual may have more than one defect.

psychoses, and an anticipation of 12.7 additional cases, a total of 23.7, compared with an expectation of only 15.0 cases. Among the sisters there were 11 known cases of mental disease and an anticipation of 11.4 cases, a total of 22.4, compared with an expectation of only 12.0.

We may next consider the frequencies of mental disorders among the siblings under specified conditions of mental tainting among the parents.

TABLE 6.

COMPARISON OF EXPECTED WITH CORRECTED TOTAL OF ACTUAL CASES OF MENTAL DISEASE IN THE FAMILIES OF 175 PATIENTS WITH DEMENTIA PRÆCOX.

Relationship.	Number with known histories.	Expected cases of mental disease.	Actual known cases of mental disease.	Anticipated cases.
Father	174	7.0	4	2.4
Mother	174	6.1	13	2.8
Paternal grandfather	135	5.4	2	0.2
Paternal grandmother	135	4.7	5	0.4
Maternal grandfather	134	5.3	5	0.2
Maternal grandmother	137	4.8	1	0.7
Paternal uncles	237	9.5	6	4.5
Paternal aunts	200	7.0	4	3.5
Maternal uncles	242	9.7	7	4.5
Maternal aunts	231	8.1	5	4.2
Brothers	374	15.0	11	12.7
Sisters	342	12.0	11	11.4
Total	2,515	94.6	74	47.5

In 27 families the father was tainted. In these families there were 144 siblings, exclusive of the 27 probands, consisting of 55 brothers and 59 sisters. Among the brothers there were 3 psychoses, and an anticipation of 2.0, a total of 5.0, whereas the expectation was only 2.2. Among the sisters there were 3 psychoses, and an anticipation of 1.8, giving a total of 4.8, compared with an expectation of 2.1.

In 32 families the mother was tainted. In these families there were 49 brothers and 44 sisters, exclusive of the probands. Among the brothers there were 2 psychoses, and an anticipation of 1.9 cases, a total of 3.9, compared with an expectation of 2.0. Among the sisters there were 4 psychoses, and an anticipation of 1.5, a total of 5.5, compared with an expectation of only 1.9.

There were 14 cases in which both parents were tainted. In these families there were 32 brothers and 34 sisters, exclusive of the probands. Because of the small totals the results cannot be considered significant. Among the brothers there was 1 psychosis, and an anticipation of 1.7, a total of 2.7, compared with an expectation of 1.3. Among the sisters there were no actual cases of psychoses. Those still exposed to a mental disease provided an anticipation of 1.2 cases, which is the same as the general expectation.

We shall consider, finally, the frequency of dementia præcox. Among the 374 brothers with known histories there were 4 cases of dementia præcox, with an anticipation of 4.2 cases, giving a total of 8.2, compared with an expectation of only 5.2. Among the 342 sisters there were 3 cases of dementia præcox, and an anticipation of 3.7, a total of 6.7, compared with an expectation of 4.2.

On the basis of family statistics of mental disease, we are now in a position to say that, though no specific law of inheritance of mental diseases has yet been proven, it does seem highly probable that there is a generalized familial basis for such disorders. The chance of developing a mental disorder is greater in some families than the corresponding chance for the general population.

Nevertheless, it would be a mistaken inference from the preceding data that a family predisposition, constitutional in nature, is an all-sufficient basis for the development of these mental disorders. The transmission of a mental disease from generation to generation is not a fatalistic process. The elements in the development of a mental disorder are not comparable to physical units which determine such characters as eye color or type of hair. The latter, so far as known, are the consequences of rigid and invariable laws, and appear at stated periods in the physiological development of the individual. Not so, however, are the facts with respect to mental disease. There is no evidence that mental disorders appear inevitably at certain life epochs with the regularity of physiological cycles. No one appears fated to develop dementia præcox because some ancestor had such a disease. It requires something in addition to a diathesis or predisposition. There must be not only a seed but a ground in which to plant the seed. Inferior human stock may still be enabled, through proper nurture, to achieve a life of a fair degree of usefulness. On the other hand, we know

that even the soundest of stock may succumb to the repeated onslaught of an unfavorable environment.

We conclude, therefore, that we cannot speak of hereditary and environmental factors as antithetic causes of mental disease. Both combine, often in subtle ways, to create such disorders. Persons with a diathesis for mental disease will undoubtedly succumb readily to many environmental stresses, which others, more fortunate in their family endowment, may be able to resist and overcome. But certain stresses are of such intensity that if repeated at sufficient length they may overcome the resistance of even the soundest constitutions. It must, furthermore, be borne in mind that siblings are affected by like environmental influences during their formative years. If faulty family habits, attitudes or conditions are factors in causing the mental breakdown of the probands, is it not probable that they would also unfavorably affect some of the other siblings? In the single family circle there is a blending of hereditary and environmental factors that renders it difficult to evaluate their respective influences on the development and health of the children.

In addition to inheritance or family background we must therefore consider two other elements, namely, the personality and the environment. Personality, though sometimes the result of inherited traits, usually develops to a great extent apart from such influences. More significant appears to be the psychic environment into which one is born and in which one grows up. The environment acts directly upon the mind through various types of stimuli, and it arouses the imitative processes. Environment also acts indirectly by encouraging the selection of traits. Thus, directly and indirectly, the environment brings about the various combinations which form the human personality. Some of these traits are correlated with unhealthy mental trends, and may be recognized in what Dr. August Hoch called the "shut-in" personality. The chief characteristics of the latter are the possession of a seclusive disposition and the gradual withdrawal of such persons from social contacts. This, in extreme cases, gives rise to certain types of reaction, emphasized by Dr. Adolf Meyer, which, if maintained over long periods of time, may result in abnormal behavior. We thus have a fusion of psychological and biological elements into the forma-

tion of a diseased personality. The process is fairly clear among the patients with dementia præcox. A large percentage exhibited well-marked seclusive traits. These traits appeared to be encouraged as a result of extremely restricted social lives of the patients and their families. A high percentage of these families were described as having no social contacts with persons outside the restricted family limits. As they grew older, the patients demonstrated lack of a normal degree of aggressive and adventurous spirit by failure to strike out for themselves and to create new careers and new homes. A surprisingly high percentage failed to marry and of those who did many failed to adjust and their marriages landed on rocky shores. In their economic activities many were failures in obtaining and in holding jobs. In a high percentage of the families there was a distinct lowering of economic status as the patients grew from childhood to manhood.

Among the manic-depressives we also found abnormal trends in personality, but in addition to seclusiveness, we found depressiveness, instability and overactivity. These mental characteristics were played upon by environmental forces until the breaking point was reached with respect to individual strength. Lowered physical resistance seems to play a significant part, whereby adverse circumstances which might otherwise be overcome cannot be borne. If we analyze those situations existing just prior to the mental breakdown, we find frequent references to financial difficulties, worry over death or sickness, and sex difficulties and disappointments. But merely because these precede the mental illness, it would be dangerous to infer that they are sufficient causes. Rather we seem justified in concluding that situations which may be of no significance to persons in otherwise sound health may prove decisive factors among those who are weakened by constitutional factors or physical disease.

If the immediate exciting factors in manic-depressive psychoses and dementia præcox are compared, it will be noted that there are no essential differences. The factors are not specific. Evidently, therefore, the type of mental disease depends primarily upon the nature of the organism upon which these external forces play. Persons who develop a manic-depressive psychosis must differ in essential traits from those who develop dementia præcox. What these differences arise from is still largely unknown. It is possible

that light may be cast upon the psychological processes in these mental disorders through a fuller understanding of the newer results being obtained from the application of insulin therapy. Used originally in connection with dementia præcox, hypoglycemic shock has also been used upon manic-depressive patients, and some startling results in therapy have been observed in both groups. Psychiatrists are beginning to study the psychological changes that accompany the processes of recovery, and it is possible that by reversing the approach they may obtain a valuable insight into the formation of those psychological trends which originally brought about the diseased mental condition.

Mental disease is a tragedy to the individual and his family, and a burden to society. It is important, therefore, that encouragement be given to all forms of research which may ultimately throw light upon the nature of these baffling disorders. We need more, not less, investigation into brain anatomy and physiology. We need additional studies into the nature of hereditary influences. Studies into the relation of constitution to disease should be carried out in greater detail. But we also need a better understanding of how human beings are molded by their environments. From a synthesis of such fields of research there will some day come a more adequate conception of the causative factors in mental disease.

DISCUSSION.

DR. KENNETH J. TILLOTSON (Waverley, Mass.).—Dr. Myerson and I are associated at the present time in a research study of inheritance in manic-depressive psychosis. We are proceeding by taking early family histories at McLean, going back to 1818, with the cooperation of the Genealogical Society in Boston and many important families whose ancestors can be traced. What we have found in possibly six families who have been pretty well traced down to the present time, is that in manic-depressive psychosis—and this has been reported in the literature—there is a tendency for families to run out, to stop. The single direct lineage is cut off after a while, and then, of course, they spread out into infinitesimal branches.

We do find, however, that the manic-depressive personality traits, as outlined by Dr. Pollock, can be pretty clearly seen throughout many generations. There is definitely a hereditary tendency of the manic-depressive features. There are many other illnesses coming in, but we are trying to confine ourselves for the moment to manic-depressive psychosis.

We feel also, as a result of our study, that had the outspoken manic-depressives who had recurrent attacks been cut off—that is, by sterilization—many valuable citizens would have been lost to society.

DR. POLLOCK.—Dr. Tillotson referred to an investigation going back into the early history of these families. One difficulty with a study of that kind is to make adequate comparisons with families of the general population. We have to study families on a comparative basis, and we all know that not only the manic-depressive families in New England, but all the old families originating with the Pilgrim Fathers and the Puritan Fathers are dying out. A few years ago I saw an interesting chart showing how the people that came over on the Mayflower had increased quite rapidly up to a certain point, then showing how those families began to decrease; and the investigator who made the chart said that in 2200, 600 years after the landing of the Mayflower, all of the descendants of the persons that came over could be carried back to England in a ship no larger than the Mayflower.

I think we can safely say that mental disease occurs more frequently in certain family stocks than in others, and that is true of both manic-depressive psychosis and dementia præcox—and probably true of nearly all types of mental disease, although we haven't studied the other types.

We are also certain that both manic-depressive psychosis and dementia præcox may appear in families in which there is no unfavorable heredity, in which mental disease has not previously appeared for three or four generations.

Comment.

PSYCHIATRY IN MEDICAL EDUCATION.

The Council on Medical Education and Hospitals of the American Medical Association has published its report of a survey on medical education in the United States.* The report was prepared by a committee consisting of men whose experiences render them significantly qualified to evaluate the present status of medical education in the United States. The committee consisted of Herman G. Weiskotten, M. D., dean of Syracuse University College of Medicine, whose background is that of a pathologist; Alphonse M. Schwitalla, Ph. D., dean of St. Louis University School of Medicine, whose background is primarily that of an educator; the secretary of the Council, William D. Cutter, M. D., whose wide experience in medical education rests upon a former deanship; and Hamilton H. Anderson, M. D., of the University of California, with a background of pharmacology, and as a member of the Council staff.

The foreword, signed by Dr. Ray Lyman Wilbur, chairman of the Council, points out that the present survey is but a part of a continuous process and represents, in a sense, a stock taking of the daily endeavors, purposes and activities of the Council. The report does not superimpose its own thoughts upon the subject of medical education but represents generally the composite opinions of representatives of various schools and depicts the present status of affairs in this field.

The introduction touches briefly upon the historical development and aims of medical education and the part which the Council has played during the thirty-six years of its work.

During the academic years, 1934-35 and 1935-36, representatives of the Council visited and surveyed all of the 89 medical schools in the United States and Canada. Of these, 77 were approved and

* Medical Education in the United States—1934-1939. Published by the American Medical Association, 1940. The volume contains 249 pages, 34 tables and 4 illustrations, with complete index.

two were unapproved medical schools in the United States and 10 were Canadian medical schools, surveyed at the latter's request. Of the 77 approved medical schools in the United States, 11 were schools dealing with training in the basic medical sciences only, leaving 66 with a four year course of study.

The report deals with the relationship of the university setting to medical education, a trend which is now more or less common in the United States since the advent of the Flexner Report on the subject in 1910. Of the 66 schools in the United States, 57 have university affiliations. It deals also with the organization and administration of medical schools with special reference to the administrative aims, purposes, objectives and duties of deanships. The relationship of the medical school to the university and the administrative functions of the deanships appear to be crucial factors in the future evolution of greater uniformity in medical education. While considerable progress has been made in the fields of administration, nevertheless it would appear from the tenor of the report that greater uniformity in these fields would result in significant progress affecting the entire field eventually.

The report deals with the administrative and instructional personnel; with student administration; educational programs; library; clinical facilities and finance; and with the subject of the basic medical sciences and clinical instruction, including those embraced by what is termed, "clinical specialties." Under the latter heading the subjects of neurology and psychiatry are discussed as follows: "There was a separate department of neurology in 14 of the 66 medical schools; in 26 schools psychiatry was combined with neurology; and in 8 of these the two subjects were taught in the department of medicine. In 22 other schools neurology was included with medicine, while in the remaining 4 schools neurology was taught in medicine, surgery, pediatrics, medicine and surgery, anatomy, and in the department of mental and nervous diseases." "Personnel in neurology and psychiatry was largely part-time; the number of hours devoted to these subjects varied from 40 to 196 with approximately one-half of the schools devoting 90 or more hours to their teaching." "Psychiatry has not yet found itself in the medical curriculum." "In some schools efforts were made to introduce a psychiatric examination generally, into the study of all patients even though the patient's present complaints did not seem

to involve mental derangement." "There was a growing demand for the inclusion in the preclinical curriculum of introductory courses in medical psychology and in psychobiology." "Psychiatry . . . . is believed to be one of the most undeveloped areas in the medical curriculum and certainly in medicine."

W. L. T.

MEDICAL EDUCATION IN CHINA.

The admirable and courageous manner in which those responsible for medical work and education in China have met the extraordinary tasks imposed upon them by the war is apparent in a communication from T. L. Yuan, acting director of the National Library of Peiping.

In his letter Dr. Yuan says:

> With the removal of medical schools and hospitals from the coast cities to the interior of China as a result of Japan's aggression, Kunming has become one of the important centres of medical education and research. There are already five medical schools and over a dozen hospitals established here in the course of last two years.
>
> Owing to the strict control over foreign exchange by the Chinese Government, none of these institutions is able to purchase foreign medical books and journals. Their scientific work has been greatly handicapped by the lack of reference books and current medical literature.
>
> In order to facilitate them in their work, the National Library has been building up a small medical collection which is made available to a large number of physicians and medical students generally.

The need for current medical literature in China is urgent, and we are glad to give publicity to the appeal for assistance by the National Library of Peiping. The National Library now located at Kunming has been placed on our mailing list for a complimentary subscription to THE AMERICAN JOURNAL OF PSYCHIATRY, and we have no doubt that other publishers of journals and books are making their contributions to advance medical education and research in China, and thus to lighten, however little, the load of those so hard pressed through the acts of the aggressor.

News and Notes.

PENNSYLVANIA PSYCHIATRIC SOCIETY.—The Pennsylvania Psychiatric Society held its regular mid-winter dinner meeting February 27, 1940, at the Penn-Harris Hotel, Harrisburg, Pennsylvania.

Dr. William C. Sandy, Harrisburg, president, presided. Dr. Charles H. Henninger, Pittsburgh, president of the Medical Society of the State of Pennsylvania, gave the opening address.

The program was devoted to the subject of "Shock Therapy in the Psychoses." Dr. Earl D. Bond, Philadelphia, read a paper on "Metrazol." Dr. Howard K. Petry, Harrisburg, spoke on "Insulin." Formal discussion by Drs. Robert W. Staley, Pittsburgh, Joseph A. Cammarata, Danville, Robert S. Bookhammer, Norristown, and Lauren H. Smith, Philadelphia, was followed by general discussion from the floor.

Sixty-four members and guests attended the meeting. Dr. Henry I. Klopp, Allentown, is president-elect of the Society. The secretary-treasurer is Dr. LeRoy M. A. Maeder, Chancellor Hall, 206 South Thirteenth Street, Philadelphia, Pennsylvania.

RORSCHACH COURSE AT MICHAEL REESE HOSPITAL.—S. J. Beck, Ph. D., head of the psychology laboratory of the department of neuropsychiatry at Michael Reese Hospital, Chicago, will give a course on the Rorschach method in personality study and clinical diagnosis.

The course will include teaching the technique of administering the Rorschach method, scoring the responses, and interpretation with especial emphasis on clinical classification. Response records as obtained from various healthy personality groups and from clinical groups (schizophrenia and some neuroses to be included), will be scored, analyzed and interpreted. Primary aim of the course will be to demonstrate the test's practical application in investigating the whole personality, with particular reference to its clinical use.

The course will be presented in two two-hour sessions daily for five days, June 24-28, 1940, inclusive. Interested persons are invited to communicate with the medical librarian, Michael Reese Hospital, 2908 Ellis Avenue, Chicago, Illinois.

EXPANSION OF THE COMMITTEE ON PUBLIC EDUCATION.—Acting on the recommendation of the Committee on Public Education, the Council has approved the establishment of regional representatives of the Committee for the dissemination of authentic psychiatric information, and the gathering of information of psychiatric import.

Each district will be composed of several states with more or less geographical solidarity and will be headed by a regional representative acting for the Committee. Later, representatives will be appointed in each of the states, but the Committee is anxious to build its organization slowly, preferring to have the skeleton organization well established and its work well defined before proceeding further.

It is contemplated that the localization of some of the public education work of the Association may make it possible in time to play a more active rôle in the shaping of public opinion in matters directly concerning the Association or having a bearing on professional standards or methods which it endorses. Regional representatives will not be called upon to enter into local controversies on psychiatric matters beyond the re-statement of standards and policies already established by the Association.

Such representatives will also report from time to time on psychiatric affairs within their district, including administrative changes, important legislation, changes in state policy, important meetings of local or state groups,—in fact, any significant data which would be helpful or of interest to members of the Association throughout the country.

It is felt that in time each district may be the nucleus for an active and authoritative program of psychiatric education which will make the aims, standards and policies of the Association well-known everywhere.

The first meeting of the newly appointed regional representatives will be held during the Cincinnati meeting of the Association at which plans for future development of the work will be discussed.

Stressing the intention of the Committee on Public Education to build this program slowly, Dr. Burlingame states:

> The Committee is traveling over new ground and in the beginning we are purposely avoiding any effort to stimulate a sudden and concerted program for public education through this new organization, preferring to have such a program develop slowly and adapting it to the needs as they do become known.

Regional representatives appointed by the president, Dr. Sandy on recommendation of the Committee on Public Education are as follows:

Dr. Franklin G. Ebaugh, Denver, Col.
Dr. C. B. Farrar, Toronto, Canada.
Dr. Ralph C. Hamill, Chicago, Ill.
Dr. Titus H. Harris, Galveston, Texas.
Dr. Richard H. Hutchings, Utica, N. Y.
Dr. George S. Johnson, San Francisco, Calif.
Dr. William C. Menninger, Topeka, Kans.
Dr. Merrill Moore, Boston, Mass.
Dr. Arthur P. Noyes, Norristown, Pa.
Dr. Winfred Overholser, Washington, D. C.
Dr. Thomas A. Ratliff, Cincinnati, Ohio.
Dr. William W. Young, Atlanta, Ga.

NORTH CAROLINA MENTAL HYGIENE APPOINTMENT.—Announcement has been made of the appointment of Dr. James Watson as director of the mental hygiene division of the Board of Charities and Public Welfare of the State of North Carolina.

Dr. Watson, a graduate of Northwestern University Medical School, has served as superintendent of the Ryder Memorial Hospital in Puerto Rico, later on the staff of the Elgin (Illinois) State Hospital and most recently on the staff of the Worcester (Massachusetts) State Hospital.

THE "BABOON BOY" OF SOUTH AFRICA.—*Science* (March 22, 1940), reports what it considers "relatively convincing evidence" of the authenticity of accounts which have appeared from time to time during the last few years of a native South African boy who had lived with and had been reared by a group of baboons. It is

announced that a fully documented report of the case will appear in a forthcoming issue of the *American Journal of Psychology.*

The record appears to supply a unique instance of a human child early removed from all contacts with its kind and made accustomed only to the life and ways of infrahuman primates whose habits and characteristics he had adopted. A special interest also attaches to the fact that having been captured and transferred to a human environment the process of change in personality and behavior as one set of culture influences was replaced by another could be watched and described.

SALMON MEMORIAL LECTURES.—Announcement is made by Dr. C. Charles Burlingame, chairman of the Thomas W. Salmon Committee for Psychiatry and Mental Hygiene, that because of uncertainties of transportation due to the international situation the eighth series of Salmon Memorial Lectures which were to have been given by the eminent Russian psychiatrist Dr. Alexander Luria have been postponed.

In view of this fact, Dr. Burlingame stated, the 1941 lecturer, Dr. Nolan D. C. Lewis, director of the New York State Psychiatric Institute will advance the date of his series of lectures to November 1940. Dr. Lewis, who will be the eighth Salmon Memorial Lecturer will deliver a series of three lectures at the New York Academy of Medicine on successive weeks, November 8, 15 and 22 of this year.

Salmon lecturers are selected each year on the basis of their scientific achievement with particular reference to constructive contributions in the fields of psychiatry and neurology. Dr. Lewis has long been a leader in the field of psychiatric research and for the past five years has been in an unusual position to gain knowledge of the trend of present-day research as coordinator of research in dementia præcox for the Scottish Rite Masons. This organization has sponsored a nation-wide assay of research in dementia præcox under Dr. Lewis' leadership.

Book Reviews.

THE PATIENT AS A PERSON. A STUDY OF THE SOCIAL ASPECTS OF ILLNESS. By *G. Canby Robinson, M.D., LL.D., Sc.D.* (New York: Commonwealth Fund, 1939.)

This volume embodies the results of an enquiry undertaken because of the conviction that there is today general neglect of social and emotional disturbances of persons suffering from various diseases and with various specific diagnoses.

Many important factors contributing to the social incapacity of individuals with organic disease or with disability due to illness of psychogenic or emotional origin were elicited by the study of the *patient as a person* in all his relationships. It is this study of "total individuals" or the investigation of the social, economic and environmental status of sick persons as well as of their physical pathology which is described as enquiry into "illness" rather than the much more limited consideration of "disease." This distinction between illness and disease is of prime importance and is continually emphasized. The first chapter is devoted to a consideration of the elements of illness and their relation to medical service. The only factors determining the selection of most of the 174 patients who were observed during the period between the spring of 1936 and June 1937, were residence in the Eastern Health District of Baltimore and admission to the medical service of the Johns Hopkins Hospital. They were typical of persons admitted either to the public wards or dispensary of the hospital.

The study of each patient as a total individual was initiated by an interview. Prior thereto the medical history, the positive objective findings, record of any previous hospital admission, diagnosis and prescribed treatment were assembled and reviewed. At the interview the patients were encouraged to discuss freely their illness, daily routine, work habits, associates, family, church and other topics of interest or containing a problem.

At that juncture effort was made to separate the symptoms of the present illness into those caused by organic disease from those of psychogenic or emotional origin. Search was then undertaken for adverse social conditions in the life of the patient. Any possible relation of such conditions to the production of symptoms was sought. The background of the patients was reviewed with special reference to factors determining personality. In addition nearly all dispensary patients were visited in their homes. The home visits almost always yielded valuable information. Representatives of social and public health organizations to whom the patient was known were consulted. There was always close cooperation with the physician in charge of the patient. The behaviour and thought content of all patients were recorded.

Consideration is given in subsequent chapters to patients with circulatory symptoms, respiratory symptoms, digestive symptoms, the diabetic, the syphilitic, the epileptic, the psychoneurotic patient and patients with various miscellaneous conditions.

Adverse social conditions were divided into disturbances of subsistence which were external and objective, and those of satisfaction which were internal or subjective. The external were due to inadequate physical or economic protection and the internal to faulty personal habits.

Eighty per cent of patients revealed some type of adverse social condition and in 36 per cent emotional disturbances due to adverse social conditions were the chief cause of illness. In many patients excessive emotional reactions were the sole cause of the illness. The adverse social conditions produced emotional disturbances which in turn produced symptoms, the reaction depending first upon inherent qualities, personality and stability of the patient and then upon the severity and duration of the stimulus. The chapters devoted to patients with various organic diseases and the psychoneuroses consist of abstracts of case histories. They illustrate the emotional status and social conditions of such patients. They also bring out the etiological importance of poverty, emotional shock and family friction in the production of certain conditions.

After consideration of the first group of case histories the conclusion is reached that "the study of social and emotional disturbances is essential as a basis for the correct diagnosis and treatment of patients with circulatory symptoms." It is further concluded that excessive emotional reactions may produce harmful effects which outweigh those caused by physical strain.

The special social and emotional disturbances of tuberculosis and syphilis are considered in detail. Each of these conditions may give rise to particularly harmful emotional reactions or anxiety states. Such reactions are said to be to some extent the same whether a patient has tuberculosis or only fear thereof.

Patients with digestive symptoms were found to be the most readily disturbed of all. As a group these patients also demonstrate well the relationship of emotional disturbance to symptoms both in the presence and absence of organic symptoms.

Those patients with discomfort, pain and weakness, whose symptoms are general in character and with no demonstrable organic disease, were represented by a group of 20 individuals. They have in common "those physical manifestations of excessive emotional reactions to adverse social conditions which we designate as psychoneuroses." They are representative of "pure" functional disturbance. With reference to these individuals the author writes:—"There is one conviction, however, that has come from the personal intimate study of the psychoneurotic patients in our series and from a comparison of them with other patients similiarly studied. This conviction is that these patients suffer greatly both in body and mind and that they are often in greater need of medical care in the broader sense than any other type of ambulatory patients."

Finally there is considered in a separate chapter a group of 29 patients with miscellaneous conditions. In reviewing the treatment of the patient as a whole in these various groups, in a number of cases the treatment of the social problem was the essential element in the patient's medical care.

In a further analysis of the present situation it is pointed out that provision for the care of acute and serious illness is usually good. However the great bulk of illness remains to be provided for, namely, progressive disease and the process of aging. There are also the chronic inflammations, infections, diseases of metabolism and bodily functions of psychogenic origin, the psychoneuroses and the psychoses.

The essential difficulty with which the patient is confronted is social incapacity. He is concerned with being restored to his former place in society. The peculiar and individual character of the problems in various diseases is emphasized and the social, economic and occupational adjustments required are stressed. The question is raised as to whether a new type of "health" doctor may be necessary, to deal with the social aspects of illness. The conclusion is later reached that a "general" physician may best meet the needs of the present time.

The importance of medical student training to meet the situation is emphasized. Every student should be introduced to the study of the patient as a total individual. This may be done by a home visit, by an interview, conference and a review of the social, emotional and hygienic problems of at least one individual. These problems, as well as the purely medical, are presented at an informal weekly conference at Johns Hopkins Medical School by each student. In this way the social and personal problems are integrated with the medical. The solution of those problems is regarded as the responsibility of the doctor as is the purely medical. The place of the medical social worker in the study of the total individual is indicated. It is believed too that medical social service awaits greater understanding, cooperation and stimulation from the medical profession. Respecting the relation of social adversity to illness, it is anticipated that in the future the medical social worker will be relied upon more generally for supplying information of fundamental significance and in directing the treatment of many patients.

The new outlook which may come about as a result of these possible developments might be of profound importance for preventive medicine and mental hygiene. The changed outlook is reflected in the following observation of the author: "In a recent discussion of the study of the patient as a person it was recalled that it was true in our student days that the ultimate aim was to follow the patient to the autopsy table. We were keen to follow him into death. Now we are facing about and are beginning to be keen to follow the patient into life." This stimulating enquiry raises many problems, not the least important of which is the development of a routine procedure in the complex services of clinics and hospitals whereby the difficulties of the patient as a person as they are related to a given illness may be ascertained. Whether in private practice or in the hospital, an understanding of these problems is as necessary as the medical history, the physical diagnosis and

all other procedures now available, if the correct diagnosis is to be made and suitable treatment undertaken.

This book should be recommended to medical students as well as to general practitioners. It contains much that is of interest and importance also to all those concerned in the provision of medical services generally. The constructive suggestions relating to medical education, if adopted, should be of immense value to the medical practitioners of tomorrow.

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THE KELLEY STATISTICAL TABLES. By *Truman Lee Kelley*. (New York: MacMillan Co., 1938.)

To review a book of statistical tables is indeed no enviable task. One might attempt it by pointing out errors in the author's calculation, but I am confident that this would prove a barren search in tables compiled by Dr. T. L. Kelley. Although a professor of education, Dr. Kelley is a mathematical statistician of first rank and one who has a predilection for accuracy. To point out that the tables are published in a very usable and legible form, the print being large and the columns well spaced, covers a description of their layout. It would appear that the only useful review will be one which indicates some of the purposes for which such a set of tables is designed. It is true the author states that "the uses are so numerous and they will be so obvious to statistical workers that illustrations in the main are superfluous." There is a strange and naïve axiom among mathematicians that we who are not of their ilk always understand what they are talking about. It is time to draw to their attention that on occasion many of us do not, and indeed sometimes we suspect that we are being unnecessarily baffled. Along with others Dr. Kelley has too great a faith in the mathematical understanding of most of us. It is with this in mind that I wish to outline some of the rather simple uses I would find for such a set of tables.

Table 1 covers 100 pages and provides eight-place values for the normal curve, probability and correlation. Columns p, q, x, and z provide values for: (1) obtaining a quantitative evaluation of test items, scores, grades or classes from the per cent of cases passing the item, obtaining the score or falling in the grade or class; (2) transforming judgments into quantitative values; (3) determining what per cent of cases in a normal distribution falls between certain given scores; (4) fitting a normal curve to a distribution of data; (5) interpreting standard deviations in terms of probability. Column $\sqrt{pq}$ provides values for: (1) computing the probable error of a proportion or of a per cent; (2) the calculation of biserial r and its probable error. Columns $\sqrt{1-p^2}$ and $\sqrt{1-q^2}$ provide values for; (1) calculating probable errors of estimate for predicted values; (2) calculating partial coefficients of correlation and partial standard deviations as used in multiple correlation; (3) calculating correlation ratios for non-linear relationships.

Table 2 provides four-place values of the χ^2 function which can be used for determining goodness of fit between an obtained distribution and a

theoretical one such as a normal curve. It may also be used in the calculation of the coefficient of mean square contingency for measuring the relationship between non-measurable variables such as eye-colour, neurotic symptoms, etc.

Tables 3, 4 and 5 provide cubic, quintic and septic coefficients which will yield interpolated values to as high a degree of accuracy as would be required in any statistical calculations.

Table 6 is an eight-place table of squares and square roots from 1 to 100.

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YOU CAN SLEEP WELL. By *Edmund Jacobson, M. D.* (New York: Whittlesey House, 1938.)

That insomnia is one of the grave ills of our modern civilization is pretty generally recognized. If the good sleeper is oblivious of the problem or the victim regards it as his own peculiar curse some of the figures on drug consumption, which Dr. Jacobson quotes, are convincing proof of the wide extent of the malady. For example, between 1930 and 1932 the sales of a single legitimate sedative, phenobarbital, rose from 25,000 to 45,000 pounds. Since as little as three grains may produce sleep, enough was sold in one year, as he points out, to provide most of the population of the United States with a night's rest. This book, as the cover proclaims, tells you "How to go to sleep promptly at night—wake up rested and refreshed in the morning—overcome fatigue resulting from wakefulness and insomnia fears." And all this without medicine or essential change in the pattern of your life, by the practice of six seemingly simple steps in relaxation.

The thesis upon which the book is built is that mental activity is inseparable from tension in the eye and speech muscles and that if this tension is relaxed mental activity ceases and you sleep well. In a certain sense these are "key muscles" and when they relax the other muscles will relax automatically. While relatively complete relaxation can be maintained perhaps only from 10 to 30 seconds, sleep will come during this interval. To be sure, such a doctrine seems like putting the cart before the horse but if the technique works, one can have no quarrel with the procedure.

As early as 1908, while working with Münsterburg at Harvard, Dr. Jacobson became interested in the problem of scientific relaxation. Since then he has studied the subject extensively both in the laboratories of the University of Chicago and more recently in his own laboratory for clinical physiology, equipped with all the most accurate devices for measuring nerve tension. This book, while written primarily for the lay reader, contains a review of all the important studies which have been made of sleep and therefore should be of value to the medical practitioner as well. It will be noted that the data presented are directly opposed to H. M. Johnson's widely discussed findings regarding "sleep motility," viz., that healthy sleepers change position from 20 to 45 times in a single night.

The form in which the author couches his contribution is that of a dramatic dialogue—between an insomniac auto salesman and the doctor. In the

concluding chapters there is a general discussion of the problems of sleep as carried on by the workers in the laboratory to which the doctor takes his patient to demonstrate his methods. While a more direct and compact style would greatly shorten the book, it would undoubtedly lose much in readability for the layman. As it is, interest is well maintained and much homely and practical advice and comment administered relatively painlessly. While few may be able effectively to follow the rules laid down for relaxation, it is worthwhile, on the one hand, to have emphasized the part played by continual strain and tension in the production of insomnia and nervous disease and, on the other, to have exploded our faith in current nostrums. To those unfortunates who rely on drugs to obtain a good night's sleep and to those who have vainly experimented with various techniques of "suggestion" the author's condemnation of all such methods will have special significance. Parents and educators will be particularly interested in the discussion of children's sleep as affected by such institutions as the radio and the movies.

For the psychologist, psychiatrist and philosopher, this book though it rests upon sound scientific research and experimentation, will probably be unsatisfactory because it too easily disposes of some of the fundamental problems with which their disciplines deal, and because it neglects the dynamic factors underlying the nerve tensions preventing sleep. Thus, the statement that "The 'will' is no longer the incomprehensible principle that philosophers have painted it for many generations past. It is simply and chiefly what your muscles do—those lying external to your bones" may not pass unchallenged. Or the author's position, long a subject of debate among psychologists, that there can be no thinking without movements, however slight, of the speech muscles. However it is to the psychiatrist that the book will prove most unsatisfactory. Not only is Freud's theory of dreams dismissed as "a development from the imagination of a speculative genius" and psychoanalysis disapproved because "you should learn to shoulder normal burdens and should not desire to be relieved of them", but no adequate consideration is given to such dynamic factors as desires and conflicts which, in the last analysis, are responsible for the mental activity innervating the muscles of eye and speech and making relaxation difficult, if not impossible. In this sense the book offers only a "muscle philosophy", a cure imposed from without; it fails to point the way to a real resolution of the tensions producing insomnia.

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POPULATION, RACE, AND EUGENICS. By *Morris Seigel, M. D.* (Privately printed in Hamilton, Ontario, 1939.)

Books touching on eugenics are legion and their approaches most varied. This one aims to cover the field in an elementary way, emphasizing the medical angles, and belongs definitely to the serious, scientific and unsentimental class. It provides an adequate, if rather conventional compilation of the usual

subject matter of positive and restrictive eugenics, being packed throughout with citations from the best genetic and psychiatric researches on the causes, eugenic significance and possible remedies for the unfavorable differential birth rates and for the various mental defects and disorders. Some heat is generated as the author stresses the inaccuracy of the "racial" theories.

To make marriage more rational and eugenic more medical coöperation is urged in premarital health examinations, social and mental hygiene clinics and expert consultation boards. To meet the social menace of increasing numbers of hereditary defectives the author regards marriage legislation as ineffective, and prefers sterilization to segregation, which he says "most eugenists consider, on a wide scale, as neither practical nor humane." The reader is warned, however, that sterilization is "applicable to only a small proportion of defectives," and will effect no material saving in the cost of maintaining them and will cause "no significant reduction in the incidence of feeblemindedness or insanity." The reviewer suggests that this account be supplemented by the newer democratic eugenic proposals appearing currently in such periodicals as the *Journal of Heredity*.

It is to be hoped that this book may indicate and may also help to stimulate a growth of interest among medical men in the social aspects of problems of human heredity.

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OUTLINES OF PSYCHIATRIC CASE-STUDY. By *Paul William Preu, M. D.* Foreword by *Eugen Kahn, M. D.* (New York: Paul Hoeber, Inc., 1939.)

This small book is intended by its author as a "comprehensive manual of psychiatric case-study." After a few pages of general introduction, detailed outlines are given for the taking of the psychiatric history, the personality study, the medical history, the present illness, the outline of mental examination and physical examination, nurses' records and notes and progress notes. The material is covered systematically and, in general, along the lines accepted by American psychiatry. Certain individual attitudes appear from time to time, but no more than would be expected from any author of a book of this type. The reviewer finds himself in hearty accord with the statement: "Technical medical and psychological terms should not be used if it is possible to avoid them." The general directions given for taking a history and for making a mental examination are in keeping with the best psychiatric thought.

The book is a very satisfactory one to place in the hands of all beginners in psychiatry.

K. M. B.

NURSING MENTAL DISEASES. By *Harriet Bailey, R.N.*, Fourth edition. (New York: The MacMillan Company, 1939.)

Everyone who is engaged in teaching psychiatric nursing will greet with pleasure the appearance of the fourth edition of Miss Bailey's well-known

textbook, the only one on this subject, we believe, written by a nurse, and perhaps the only one which really deals with the *nursing* of the mentally ill.

Miss Bailey has spent the greater part of her professional life in the field of psychiatric nursing. A graduate of the School for Nurses of Johns Hopkins Hospital, she was associated for some years with the Phipps Psychiatric Clinic there. Lately she has made a survey of a large number of mental hospitals, in connection with the inclusion of psychiatric experience in the undergraduate courses of general hospital schools.

The general arrangement of the material in this edition is the same as formerly, but two new chapters are added. These are Chapter XIII, "Nursing the Psychoses with Convulsive Disorders," and Chapter XV, "Nursing the Psychoses due to Metabolic and other Somatic Diseases." These are very useful additions.

During her survey Miss Bailey constantly asked for suggestions which would make her book more useful. She tells us that "the reply was almost unanimous; 'there should be more description of nursing procedures, more information as to what to *say* and what to do'; and more concrete suggestions for approaching the patient and establishing the necessary rapport." This is of course the heart of the problem. We have gradually acquired an extensive list of things which we tell our nursing students not to say and do; but positive suggestions have been largely lacking. The result has too frequently been that the nurse is afraid to say anything. "An earnest effort," says Miss Bailey, "has been made to meet these suggestions." It has resulted in a few definite models of the more desirable verbal approaches for gaining the co-operation of patients. To say that there are only a few is surely no disparagement of this book, since no other gives any. There are, however, many questions which nearly all psychiatric patients ask again and again, and for the answers to which the nurse is apt mechanically to refer them to a doctor who is no longer present. There are surely answers to these questions which the small medical staffs of our mental hospitals would be more than glad to leave to nurses if they could feel certain that they would be intelligently handled. Let us hope that in a later edition Miss Bailey will put us still further in her debt by giving us some of these answers.

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Injuries of the Nervous System Including Poisonings. By *Otto Marburg and Max Helfand.* (New York: Veritas Press 1939.)

In this small volume a survey of the causes, symptoms, diagnosis and treatment of injuries of the brain, spinal cord and peripheral nerves is presented, together with a brief discussion of various chemical agents which poison the central nervous system. In view of the wide experience of the authors it should be of value to have this expression of their opinions.

It is pointed out by the authors that certain personality types may be predisposed to trauma, a fact not so frequently recognized. The opinion is emphasized that the mental symptoms following trauma are largely dependent

upon the mental makeup of the individual before the injury and that "a normal man with an intensive head injury tends to remain normal after the injury."

While the book has several points in its favor its brevity has led to the omission of important details. There is a failure to discuss adequately some of the neurological phenomena commonly associated with head injuries. In the preface it is stated that the practical standpoint would be emphasized. However the sections on such subjects as blood pressure and pulse changes, for example, evidence a lack of perspective for dealing with the problem of head injuries from a practical standpoint. In general, similar defects are evident in the sections devoted to injuries of the spinal cord, the peripheral nerves and to poisonings of the nervous system. Often no proper distinction is made between trivial and important facts. The discussion of therapeutic procedures is often too brief to be of practical value.

A lack of fluency with the English language might well be condoned in view of the tumultuous life which the authors may have experienced in beginning their profession in a new country, but it would seem that they might have obtained some help in correcting the ambiguous language with which their pages are filled. Even if such ambiguities can be condoned there are not a few evidences of haste and carelessness in proof reading in the form of mispelled words and errors in reference. In addition, references are made to authors who are not listed in the bibliography. While no pretense is made of giving a complete bibliography it would seem that references to many helpful and important articles, particularly in the English and American literature, have been omitted. Not only have many important references been omitted but results previously written in the English language have been restated in remarkably obscure language.

In conclusion, this book cannot be given an unqualified or general recommendation. It leaves much to be desired as a practical survey of the subject. Many more readable surveys of the problems of head injuries are available in the literature. Those readers who are very familiar with the subject material will, however, find some opinions and facts of interest and of value.

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. . . . AND THE STUTTERER TALKED. By *A. Herbert Kanter, M. D., and A. S. Kohn, B. A., B. Sc.* (Boston: Bruce Humphries Inc., 1938.)

This book presents an interesting human document. It is a graphic account of the life and personality development of a stutterer. Beyond this, it is of little value to the speech therapist, who would require a more specific outline of treatment and a clearer intimation of how the authors arrived at their conclusions.

It would appear that the reason for writing this book in biographical style was to attract the attention of the lay reader. But if this book is primarily for the stutterer and his parents it may be criticized from the standpoint of comprehension. The description of the boyhood struggle against his handicap

is excellent and will find a sympathetic echo in the lives of many stutterers. But the latter part of the book would not be readily understood by the average reader. When the authors come to the actual treatment of the adult stutterer they tend to become obscure.

The value of the book lies in its strong emphasis on the psychological and emotional aspects of stuttering. The authors also stress the need for the early treatment of the stutterer and a clearer understanding of his problem by parents and teachers. And, lastly, they do not hesitate to point out the uncertainty of cure. They emphasize the factor of hereditary predisposition in the stuttering condition, and conclude that the adult stutterer rather than discovering a miraculous cure attains the power of control over his stuttering. He learns to adjust himself to his stuttering, and manage it in such a way that he is not handicapped in normal social intercourse with his fellowmen.

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SCIENTIFIC CONTRIBUTIONS OF THE FIRST PSYCHIATRIC HOSPITAL OF MOSCOW, U. S. S. R. (Second issue, dedicated to the occasion of the 130th anniversary of the founding of the hospital.) Edited by *M. A. Djagaroff*. (Moscow: Bolshaya Gruzinskaya, 1939.)

The first paper in this collection is a historic sketch of the hospital which was the first to be founded for mental cases not only in Moscow but in the Russian Empire. Here we find a detailed duplication of the "Bedlams" of western countries, including the eventual emergence of a well arranged and equipped institution in which competent workers apply the most modern techniques in their practice, teaching and research.

The bulk of the volume of over 400 pages, well printed and illustrated and attractively bound, consists of 23 papers dealing mainly with forms of active therapy of the commoner psychoses.

Of special interest is a contribution by Vasilyeva, Dikovitzky, and Zelyeva on "Treatment of Schizophrenia by Combined Method (Insulin and Cardiazol)." The authors have tried, on 15 resistant cases, several ways of combining both remedies, simultaneously and alternately. Following are their principal conclusions:

At the beginning of treatment one cannot prescribe combined therapy for a given patient, inasmuch as neither the form, nor duration, nor course of the illness affords any indication for it.

Cases which prove to be resistant to the one or the other therapy alone may, upon thorough investigation of both its physical and psychic features, be subjected to combined treatment.

Some cases, in which neither insulin nor cardiazol alone yields good results, unquestionably change their course for the better only under the combined action of insulin and cardiazol.

Under the combined treatments the results reported in the above-mentioned 15 resistant cases were as follows:

Cases under 1 year's duration, full remission 4, no improvement 1, total 5.

Cases over 1 year's duration, full remission 1, remission with defect at time of release from the hospital 2, improvement within the hospital 5, no improvement 2, total 10.

A. J. R.

SEGUNDA REUNIÓN DE LAS JORNADAS NEURO-PSIQUIÁTRICAS PANAMERICANAS. Publicadas por *Honorio Delgado y J. O. Trelles.* (Lima: Imprenta Torres Aguirre, 1939.)

This volume of approximately eleven hundred pages contains the official contributions which appeared on the program of the second Neuro-Psychiatric Pan-American Congress, which was held at Lima, Peru, March 20-25, 1939. (The first Congress was held under the auspices of the University of Chile in 1937).

The officers of the Congress were: President, Dr. Honorio Delgado; Secretary-General, Dr. J. O. Trelles; Chairman of Sections, Drs. Nerio Rojas, Arturio Vivado O., Edgard Pinto César, F. Herrera Guerrero, Horatio M. Pollock, Demetrio E. García, Isaac Horvitz, Elas Malbran, José B. Odoriz, Aurelio Loret de Mola, Alejandro Busalleu.

Official delegates represented the following countries: Argentina, Brazil, Cuba, Chile, Peru, Santo Domingo, United States, Venezuela. The delegates from the United States were Dr. Horatio M. Pollock and Dr. Robert B. Lamb.

The scientific program resolved itself mainly into a series of symposia. One of these dealt with the anatomy, pathology, physiology, psychology and psychotherapy of pain. Another surveyed the various aspects of hypochondriasis. One session was devoted to the newer treatments of schizophrenia. The subjects of social service and medical organization and policy were dealt with in other sessions. Professor Nerio Rojas discussed the biological foundation of civil incapacity in mental illness, and Dr. Enrique Encinas presented an elaborately illustrated paper on the citoarchitectural variability of the cortex of the human frontal lobe.

In addition to the countries represented by delegates to the Congress members were also present representing Ecuador, Bolivia and Uruguay.

C. B. F.

PRELIMINARY ANALYSIS OF GROUPING BEHAVIOR IN PATIENTS WITH CEREBRAL INJURY BY THE METHOD OF EQUIVALENT AND NON-EQUIVALENT STIMULI.*

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Unsatisfactory material and methods have jointly hindered progress in analyzing the extent to which circumscribed cerebral lesions in man are associated with alteration of the "higher" mental functions. Prior to developments in the field of neurosurgery, particularly during the past ten years, the human cranio-cerebral cases available for study rarely presented circumscribed neurological lesions. While studies of developmental anomalies, vascular cases, traumatic head injuries (including injuries resulting from gunshot or shell-wounds), cases with brain tumor and infectious and degenerative diseases of the central nervous system have in some instances yielded constellations variously useful in neurological diagnosis,[11] they have not thus far revealed definite relationships of the "higher" mental functions to the brain.[21] However, with the rapid developments in recent years in the field of neurosurgery, partial and complete lobectomies have become routine operations in several centers. With the increasing availability of such cases for purposes of intensive study, the problem of relatively satisfactory material is in some measure diminishing.

Concerning available methods, however, it is but comparatively recently that the need for analysis of the basic psychological functions involved in various forms of behavior has been clearly conceived as being linked to the problem of cerebral function.[9, 17, 19] Moreover, the adequacy of traditional psychological methods for revealing truly basic mechanisms of behavior has been questioned by Klüver, who in seeking such mechanisms in the behavior of

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

monkeys found it necessary to develop the duplex method which he subsequently termed the "method of equivalent and non-equivalent stimuli." [19] It has not yet been generally recognized, unfortunately, that the considerations which Klüver has raised concerning methods of studying behavior apply to *any analysis of behavior*, whether the organism involved be insect or rat, monkey or man.[12]

The problem of equivalence in the analysis of behavior has been dealt with in its various aspects by Klüver [19] and will be considered here only in a general way. This problem arises most prominently in connection with various forms of behavior which remain unaltered in the presence of marked change in the environmental stimulus constellation. Such forms of behavior do not persist unaltered in the presence of *all* changes in the stimulus constellation, however. Using Klüver's terminology, it becomes convenient to refer to the stimuli in the first situation as "equivalent stimuli" and to those in the second (in which the response in question is altered) as "non-equivalent stimuli." Interest in such differentially stable forms of behavior does not end with the discovery (for a particular response) of a number of "equivalent" stimuli, *i.e.*, an "equivalence range." In fact, primary concern in a given situation is an answer to the question: "What property or characteristic of equivalent stimuli is responsible for equivalence?" It may be observed, for example, that under certain conditions: (A) solid squares, solid and outline ellipses, solid and outline circles and line drawings of a "star" of certain sizes, when projected symmetrically at the monocular fixation point of a *nulle* vision hemianopic patient, will be seen by him as whole or "completed" figures; whereas (B) squares or ellipses of a particular size, straight lines, diagonal lines, less than half a circle, or a picture of a dog, face, bottle or butterfly will be seen as part figures or "uncompleted." [7] In line with the preceding discussion, the stimuli of A, in terms of "completion" behavior, may be referred to as "equivalent stimuli" whereas the stimuli of B with reference to "completion" behavior may be termed "non-equivalent stimuli." Clearly, where questions of such biological importance as "seeing" and "not seeing" are involved, it is not sufficient to know that A stimuli and B stimuli exist. It becomes important to know what property or characteristic of the A stimuli makes it possible for them to be

"seen," *i.e.*, "completed." It should be clearly recognized that whatever this property or characteristic is, it is, in a certain sense, present in the A stimuli only and yet cannot be defined without analyzing the properties of the B stimuli.

Present Investigation.

During the past four years at the University of Chicago Clinics, we have been investigating by experimental methods the behavioral effects of brain lesions in carefully selected neurosurgical patients. With some exceptions noted elsewhere, these patients have come to our attention through the cooperation of Drs. Percival Bailey, Paul C. Bucy, and A. Earl Walker.

As one aspect of the general investigation, we have been interested for various reasons in securing information as to ways in which perceptual material of various kinds can be organized or integrated by normal individuals and by patients with cerebral injury. In securing such information, test-situations requiring "grouping" or "sorting" of heterogeneous test-objects according to individual preference have been developed and applied in conjunction with Klüver's method of equivalent and non-equivalent stimuli. Heterogeneous test materials in many respects similar to those used in the present investigation have been used by other investigators in studying grouping behavior [*cf.* Ach, 1905,[1] Dearborn, 1916,[5] Franken, 1917,[6] Stern, 1917/18,[23] Bouman and Grünbaum, 1925,[3a] Gelb and Goldstein, 1925,[8] Weigl, 1927,[24] *et al*]. However, the methods employed in connection with such materials differed considerably from those which we have employed.

On the basis of their studies of grouping behavior of World War cases with cerebral injuries Gelb and Goldstein [8] and Weigl [24] concluded that of the various factors influencing such behavior the factor of *attitude* was the predominant one in limiting the performances of patients with cerebral injury. It is clear that such a conclusion, if valid, would be of great significance for further analysis of cortical functions. Examination of the evidence upon which this conclusion was based reveals, however, that in no instance did these investigators carry analysis of the behavioral situation far enough to disclose a *basic* or controlling principle. The

preliminary analysis of grouping behavior of normal individuals and patients with cerebral injury by the method of equivalent and non-equivalent stimuli made in the present investigation indicates that under certain conditions factors other than attitude may be predominant in controlling such behavior.

Subjects Used in Present Investigation.

Of the twenty-six neurosurgical cases considered in this report, (*cf.* Figs 1 and 2) eleven (Cases 1 to 11) have a considerable lesion in one frontal lobe; one (Case 12) has an undetermined amount of cortical atrophy residual to removal of a small meningioma from the right frontal lobe; fourteen (Cases 13 to 26) have lesions in the brain posterior to the frontal lobes, including one instance (Case 26) of cerebellar lesion. The 11 normal individuals examined ranged in age from 12 to 56 years. By spreading the ages of the control subjects through so wide a range, the chances of getting relatively consistent performances in the test situation were arbitrarily given a strong negative bias.

It is not our purpose to present here a detailed history of each of the patients studied. Those cases which best approximate physiological preparations, in the sense of our knowledge of the probable extent of the lesion, and which have been available for extended investigation, will be the subject of a series of individual reports to be published later.

Patients With Brain Injury.

Case 1.—48-year-old American-born male; occupation: salesman. Entered University of Chicago Clinics (UCC) 9/4/35 with a history of severe headaches, occasional vomiting, blurred vision and loss of weight. Operation performed 9/17/35 by Dr. Bailey involved partial extirpation of the left prefrontal lobe for removal of an olfactory groove meningioma. Recovery uneventful; patient discharged from hospital 9/28/35. Post-operative I. Q. 110 (Stanford-Binet.)

Case 2.—63-year-old American-born female; occupation: housewife. Entered UCC 2/1/31 with a history of severe headaches, loss of memory and sense of smell and loss of vision in the left eye, continued drowsiness and difficulty in walking. Operation performed by Dr. Bailey in two stages 2/5/31 and 2/13/31 involved partial extirpation of the left prefrontal lobe for removal of an olfactory groove meningioma. Recovery uneventful; patient discharged from hospital 3/3/31. Post-operative I. Q. 96 (Stanford-Binet).



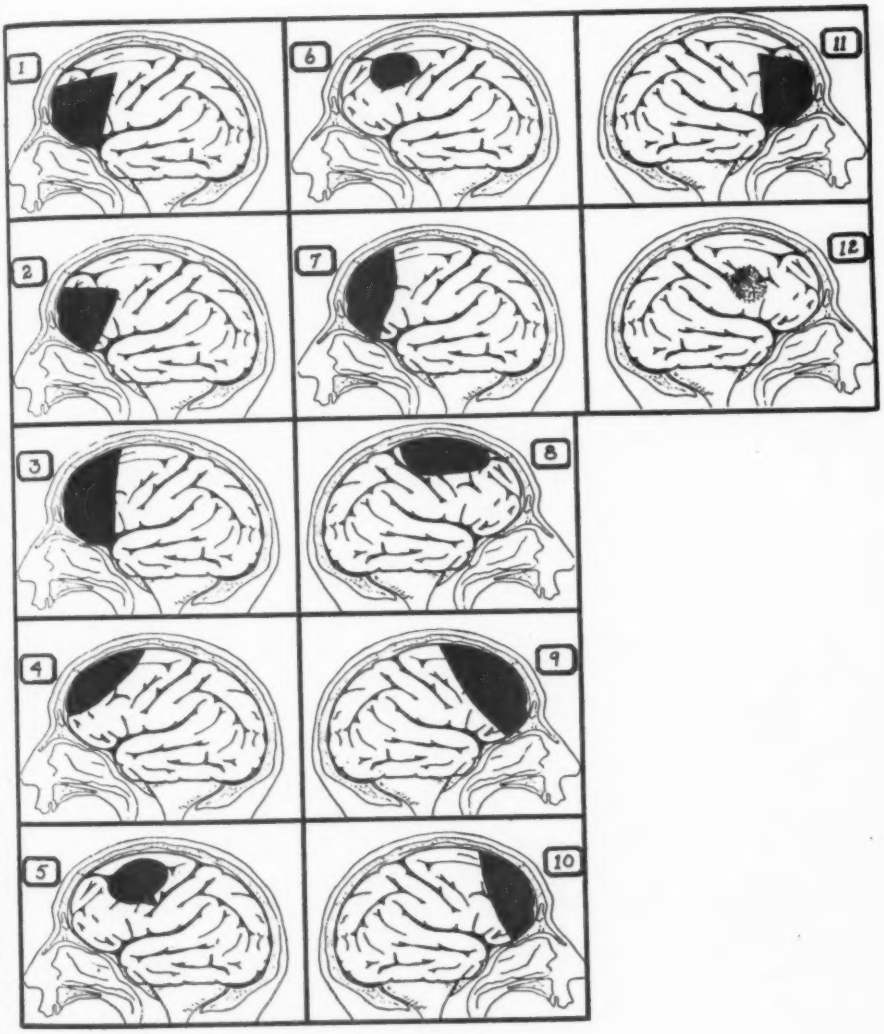


FIG. 1.

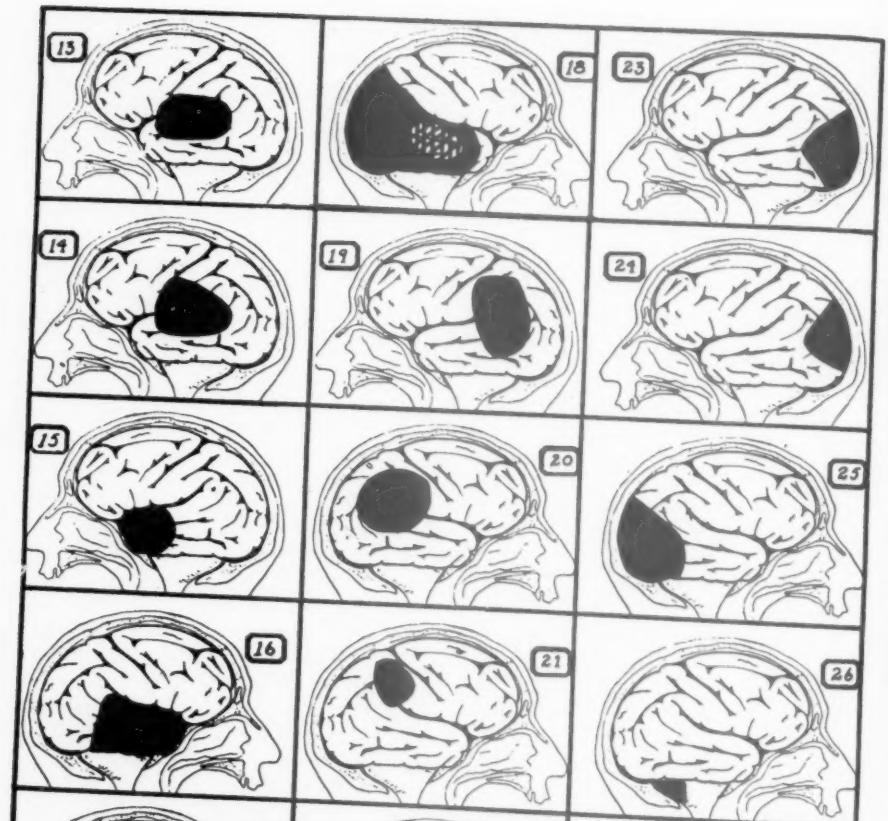


FIG. 2.

CASE 3.—58-year-old Canadian-born female; occupation: retired school teacher. Entered UCC 8/31/36 in coma following a series of major convulsions during the past eight hours. A history of definite personality changes during the past year was obtained from the relatives. Operation performed 9/10/36 by Dr. Bucy involved extirpation of the left prefrontal lobe for partial removal of a calcified glioma. Recovery, except for persistent motor aphasia, was uneventful. Patient discharged from hospital 10/6/36.

CASE 4.—32-year-old American-born male; occupation: truck-driver. Entered UCC 12/3/37 with history of generalized convulsions for past two years which during the past six months had become progressively more severe and were followed by difficulty in speaking and weakness of the right arm. Operation performed 12/9/37 by Dr. Bucy involved partial extirpation of the left prefrontal lobe for removal of an astrocytoma. Recovery uneventful; patient discharged from hospital 12/21/37.

CASE 5.—36-year-old American-born male; occupation: salesman. Entered UCC 6/23/37 with history of generalized convulsions and progressive difficulty in speaking for past seven years. Operation performed 6/29/37 by Dr. Bucy involved partial extirpation of the left prefrontal lobe for removal of an astrocytoma. Recovery uneventful except for transitory motor aphasia. Patient discharged from hospital 7/10/37.

CASE 6.—51-year-old American-born male of Irish descent; occupation: traffic policeman. Entered UCC 6/17/36 with a history of severe headaches, spells of dizziness and unsteadiness for past four months, and progressive failure of memory. Operation performed 6/17/36 by Dr. Bucy involved partial extirpation of the left prefrontal lobe for removal of a benign glioma of the septum pellucidum. Recovery uneventful; patient discharged from hospital 7/20/36. Post-operative I. Q. 105 (Stanford-Binet).

CASE 7.—53-year-old American-born male; occupation: machinist. Entered UCC 10/17/37 with a history of generalized convulsions, progressive weakness of right arm and leg, and general listlessness for past six months. Operation performed 10/26/37 by Dr. Bucy involved nearly complete extirpation of the left prefrontal lobe for partial removal of an oligodendroglioma. For one week following operation patient was aphasic, irrational and apathetic; he repeatedly urinated on the walls of his room or on the bedposts. Recovery rapid during second post-operative week. Patient discharged from hospital. 11/7/37.

CASE 8.—32-year-old Austrian-born male; occupation: unskilled laborer. Entered UCC 9/27/28 with a history of severe headaches, weakness and numbness of the left side of the body, and difficulty in walking, all for the past five months. Operation performed in two stages 10/2/28 and 3/19/29 by Dr. Bailey involved partial extirpation of the right frontal lobe for removal of a glioblastoma. Recovery uneventful except for residual left hemiparesis. Patient discharged from hospital 10/22/28 and 3/29/29.

CASE 9.—47-year-old Lithuanian-born male; occupation: journalist. Entered UCC 2/1/38 with a history of major convulsions, progressive difficulty in speaking and failing memory during the past six years. Operation performed 2/8/38 by Dr. Bucy involved nearly complete extirpation of right prefrontal lobe for removal of a glioma found to involve the corpus callosum. Recovery uneventful except for residual left hemiparesis. Patient discharged from hospital 3/2/38.

CASE 10.—31-year-old American-born female; occupation: housewife. Entered UCC 7/24/39 with a history of severe throbbing headaches, numbness and tingling of upper and lower extremities during past six months. Operation performed 7/29/39 by Dr. Walker involved partial extirpation of right prefrontal lobe for removal of a cerebral aneurysm. Recovery uneventful; patient discharged from hospital 8/15/39.

CASE 11.—46-year-old American-born male; occupation: farmer. Entered UCC 5/3/37 with a history of severe headaches, occasional spells of vomiting, and failing memory during past two years. Operation performed 5/13/37 by Dr. Bailey involved partial extirpation of the right prefrontal lobe for removal of a sphenoidal ridge meningioma. Recovery uneventful; patient discharged from hospital 6/12/37.

CASE 12.—33-year-old American-born female; comptometrist. Entered UCC 4/10/39 with a history of focal convulsive attacks, slight slurring of speech and failing of memory for past six months. Operation performed 4/20/39 by Dr. Bucy involved decompression and removal of small meningioma from the right frontal lobe. The cortex under the tumor was noted to be markedly depressed. Recovery uneventful; patient discharged from hospital 5/15/39. Pre-operative I. Q. 126; post-operative I. Q. 126 (Stanford-Binet).

CASE 13.—19-year-old American-born male; occupation: farmer. Entered UCC 12/2/35 with a history of frequent headaches, progressive loss of memory, loss of ability to concentrate and vision. Operation performed 12/5/35 by Dr. Bailey involved partial extirpation of left temporal lobe for partial removal of a glioma. Recovery was uneventful except for persistent motor aphasia. Patient discharged from hospital 12/15/35.

CASE 14.—35-year-old America-born male; occupation: farmer. Came to the laboratory 8/22/38 after being seen by Dr. Bailey. Ten years ago patient was completely paralyzed. Operation performed at another hospital involved the removal of a cyst from the left temporal lobe. For 18 months following the operation the patient could not speak. Since then there has been progressive improvement from the paralysis and loss of speech.

CASE 15.—33-year-old American-born female; occupation: housewife. Entered UCC 6/12/39 with a history of severe headaches, occasional spells of vomiting and progressive difficulty in speaking during the past year. Operation performed 7/6/39 by Dr. Walker involved partial extirpation of the left temporal lobe for partial removal of a glioblastoma. Recovery uneventful ex-

cept for transitory nominal aphasia; patient discharged from hospital 7/11/39. Pre-operative I. Q. 108; post-operative I. Q. 108 (Stanford-Binet).

CASE 16.—51-year-old German-born male; occupation: truck driver. Entered UCC 7/7/36 with a history of generalized convulsions, strange tastes and odors during the past four months. Operation performed 7/11/36 by Dr. Bucy involved extirpation of the right temporal lobe for partial removal of an astrocytoma. Recovery uneventful; patient discharged from the hospital 8/3/36. Post-operative I. Q. 91 (Stanford-Binet).

CASE 17.—35-year-old American-born male; occupation: farmer. Entered UCC 1/7/36 with a history of severe headaches and attacks of dizziness for the past four months. Operation performed 2/1/36 by Dr. Bucy involved nearly complete extirpation of the right temporal lobe for partial removal of an infiltrating glioma. Recovery uneventful; patient discharged from the hospital 2/13/36.

CASE 18.—This case came to our attention through the courtesy of Dr. Harold Voris of Chicago. 31-year-old American-born male. Occupation: none. Entered Mercy Hospital 9/12/35 with a history of major and minor seizures for the past 4½ years and severe frontal headaches during past year. Operation performed 9/13/35 by Dr. Voris involved partial extirpation of the right temporal lobe for removal of a "hard fibrous infiltrating tumor located 2 cm. below the surface. A cavity about 4 x 4 x 6 cm. was produced in the posterior part of the right temporal lobe." Recovery was uneventful except for residual focal seizures which could not be completely controlled with medication. Post-operative I. Q. 110 (Stanford-Binet). A second operation performed 2/4/39 involved partial extirpation of the left occipital and parietal lobes. Recovery uneventful except for left hemiparesis.

CASE 19.—19 year old American-born male; occupation: student. Entered UCC 1/15/36 with a history of a fall at the age of five resulting in a compound depressed fracture of the skull, which was repaired by operation. Following the fracture, the patient was unable to speak for one week and experienced marked weakness of the right arm and leg which has persisted. Generalized Jacksonian seizures began at the age of 13 and persisted thereafter. Operation performed 1/24/36 by Dr. Bucy involved partial extirpation of the left parietal and temporal lobes for removal of a cortical scar. Recovery uneventful; patient discharged from the hospital 2/11/36.

CASE 20.—28-year-old America-born female; occupation: graduate nurse. Entered UCC 5/17/37 with a history of severe headaches since childhood and generalized convulsions during the past year. Weakness of the left side of the body and occasionally disorientation for time, place and person. Operation performed 5/20/37 by Dr. Bailey involved partial extirpation of the right parietal lobe for partial removal of a glioblastoma. Recovery uneventful; patient discharged from the hospital 6/13/37.

CASE 21.—21-year-old American-born female of Italian descent; occupation: none. Entered UCC 7/13/36 with a history of almost daily generalized convulsive seizures during the past year with marked weakness on the left side of the body and marked impairment of stereognosis on the left. Operation performed 7/25/36 by Dr. Bucy involved partial extirpation of the right parietal lobe for removal of a cortical scar. Recovery uneventful; patient discharged from the hospital 8/17/36.

CASE 22.—25-year-old American--born female; occupation: file clerk. Entered UCC 9/27/37 with a history of visual disturbances during the past ten years consisting of flickering lights and colors, and headaches and spells of vomiting for the past 18 months. Operation performed 10/23/39 by Dr. Bucy involved complete extirpation of the left occipital lobe for removal of a spongioblastoma polare. Recovery uneventful; patient discharged from hospital 11/11/37.

CASE 23.—33-year-old American-born female of Danish descent; occupation: stenographer. Entered UCC 4/22/32 with a history of failing memory and spells of dizziness during past year and blurring of vision during past four months. Operation performed 4/28/32 and 5/10/32 by Dr. Bailey in two stages involved partial extirpation of the left occipital lobe for removal of an angioblastic meningioma. Recovery uneventful; patient discharged from hospital 7/6/32.

CASE 24.—22-year-old American-born female of Italian descent; occupation: housewife. Entered UCC 7/29/29 with a history of severe headaches, spells of nausea and vomiting and blurring of vision with diplopia for the past month. Operation performed 8/2/29 by Dr. Bailey involved partial extirpation of the right occipital lobe for removal of a glioma. Recovery uneventful; patient discharged from the hospital 8/26/29.

CASE 25.—22-year-old American-born female; occupation: stenographer. Entered UCC 9/27/39 with a history of severe headaches and attacks of nausea which were relieved by induced vomiting, during the past three years. Operation performed 10/10/39 by Dr. Bucy involved complete extirpation of the right occipital lobe for removal of an oligodendroglioma. Recovery uneventful; patient discharged from the hospital 11/3/39. Pre-operative I. Q. 133; post-operative I. Q. 133 (Stanford-Binet, Forms L and M).

CASE 26.—15-year-old American-born male; occupation: student. Entered UCC 10/4/38 with a history of severe headaches, attacks of dizziness and unsteadiness, and a feeling of pressure in the right ear. Operation performed 10/6/38 by Dr. Bucy involved the nearly complete extirpation of the right hemisphere of the cerebellum for removal of an astrocytoma. Recovery uneventful; patient discharged from the hospital 10/19/38.

REPRESENTATION OF THE LESIONS.

The distribution of the lesion with reference to the lateral plane for each of the cases is indicated in Figs. 1 and 2 (the number

shown in the figure of each brain represents the case number). In reconstructing the lesions use has been made of the following types of data: (1) lateral X-ray plate in which silver clips outline the lesion, (2) operative notes of the surgeon, (3) sketch of the operative field made by the surgeon at the time of the operation, and (4) diagram of the operative fields made by the writer at the time of the operation. Among these various sources of information greatest use has been made of (1). To facilitate translation of the post-operative X-ray plates, Bailey's figure of a normal brain [2] was photographed and enlarged to life size.* Lithoprinted forms of this figure for both right and left-sided orientations were prepared and used in direct contact with the X-ray plates. The line of section for each lesion was then determined either by Dr. Paul C. Bucy or by Dr. A. Earl Walker.

Method and General Procedure.

The test-objects consisted of sixty-two objects which differed in such objective properties as size, shape, color, brightness, weight, material, hardness and position (see Fig. 3). These objects were presented in a predetermined arrangement on the surface of a table (2′ x 3′ at a height of 30″) which was covered by a dark brown linoleum mat. The subject remained seated at the table during the progress of the test. Illumination of the test-objects was from above by incandescent lamp and averaged 5 foot candles.

Every effort was made to establish and maintain a comfortable, relaxed "atmosphere" for the subjects. The test-procedures were never begun until the examiner was convinced that good rapport had been established. Any suggestion to the subject that speed or rate of performance was a consideration in the procedures was deliberately avoided. The length of time during which the various subjects were willing to participate without interrupting the test procedures varied widely, particularly in the patients with a cerebral lesion. In the case of the normal subjects, test procedures were terminated after 1½ to 2 hours on the average; in only two instances was it considered necessary to have more than one session devoted to this particular test-situation. The "work unit" for the

* This drawing of the normal brain was found to compare quite well in its various proportions with Berry's dioptrographic tracing of a normal brain.[3]

cerebral injury patients, on the other hand, averaged in the neighborhood of 30 minutes; in some instances as many as seven sessions were required before the necessary information was secured.

In setting the test-situation for the subjects, the following sequence of procedures was used.

Part I was concerned with establishing the range of interest and familiarity of the subject with the test-objects. The objects were distributed in a predetermined arrangement on the table before the subject. He was then asked: "Have any of these things *ever* been of interest to you—if so, pick them up one at a time." A record was made on a coded data sheet of the objects picked up by the subject. He was encouraged to verbalize the nature of his interest, particularly as to whether pleasant or unpleasant in recollection. These two types of interest were noted differentially. As soon as a preliminary interest range had been indicated by the subject, the examiner pointed to each of the objects not mentioned by the subject, and asked: "What is this—has it ever been of interest to you?" Any object with which the subject was unfamiliar was thus discovered at the outset of the test procedures; in such instances he was told the common name of the object and its customary use. Evidence of anomia was carefully looked for by the examiner.

Part II was concerned with having the subject make a "spontaneous" grouping of some of the test-objects. The objects were again distributed in random

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LEGEND FOR FIGURE 3.

1. Glass bottle labeled "Camphor."
2. Glass stopper.
3. House key.
4. Wooden pulley.
5. Colored picture of a rooster.
6. Glass bottle and stopper labeled "Bergamot."
7. Pink yarn.
8. Yellow yarn.
9. Multicolored cube.
10. Metal whistle.
11. Colored picture of a bell.
12. Cancelled foreign postage stamp.
13. Toy metal spoon.
14. Bone chess pawn.
15. Miniature electric light socket.
16. Blue yarn.
17. Coarse sandpaper.
18. Smoked glass lens.
19. Pipe stem.
20. Playing card.
21. Metal jar lid.
22. Small cork.
23. Pipe bowl.
24. Picture of a house key.
25. Small wax candle.
26. Rubber-covered wire.
27. Lipstick.
28. Wax crayon.
29. Metal hairpin.
30. Red wool cloth.
31. Blue poker chip.
32. Piece of thin roundish stick.
33. Flashlight bulb.
34. Abstract design.
35. Colored picture of a doll.
36. Ping-pong ball.
37. Card labeled "Pipe."
38. Fine sandpaper.
39. Pink candle holder for cakes.
40. Bakelite bracelet.
41. Rubber grommet.
42. Red paper stock.
43. Small padlock.
44. Red poker chip.
45. Card labeled "Hairpin."
46. Metal thimble.
47. Piece of thick roundish stick.
48. Red poker chip.
49. Colored picture of a rabbit.
50. Black wooden cube.
51. Small glass jar.
52. Toy metal fork.
53. Small metal key.
54. Metal pulley.
55. Blue bakelite earring.
56. Canvas doll slipper.
57. Toy metal knife.
58. Small metal key.
59. Miniature electric light bulb.
60. Metal puzzle.
61. Round wooden box lid.
62. Round wooden box.

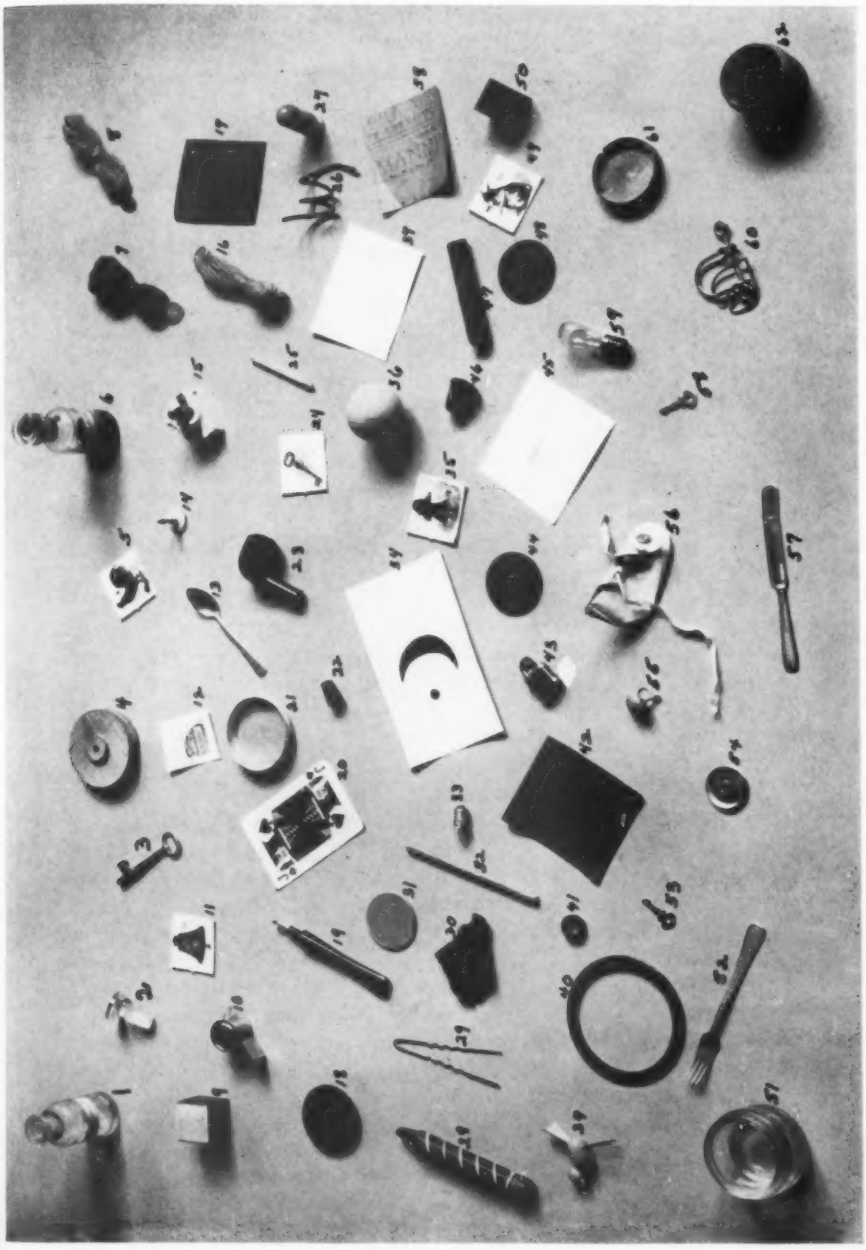


FIG. 3.

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fashion on the table. The subject was told: "Place those together which seem to you to belong together." In every instance the subject began at once to sort the objects before him into groups or piles. Careful observation was made of the manner in which he proceeded. At the first pause or slowing in rate of progress,* but only after a minimum of five groups had been made, the subject was interrupted at his task and told to rest. The table supporting the objects was then moved to one side of the room out of the line of vision of the subject. The examiner then said: "Just relax while I jot down some notes," and proceeded to record the content of the groups made by the subject. The latter was encouraged to smoke if he wished and to converse on any topic he cared to, except any aspect of the test procedures. The rest period was limited to five minutes for all subjects.

Part III was concerned with testing for recall of the test-objects by the method of imminent recall. When the five minute rest period had elapsed, the examiner placed a cover over the objects on the table. (In preliminary experiments it was found that covering the objects earlier in the rest period led the subject to anticipate the test for recall.) The examiner next told the subject: "Mention the things you saw on this table." The objects mentioned by the subject were then noted on the coded data sheet. The subject was permitted to mention as many of the objects as he could without a time limit.

Part IV was concerned with testing for nominal equivalence. The cover was removed from the table thus again exposing the test-objects. The subject was aked: "Why did you put these (indicating the objects of a particular group) together?" This question was repeated for each of his groups until a grouping principle, for example, "metal objects" or "games," had been verbalized by him. The test-objects were then redistributed in random fashion on the table by the examiner and the subject instructed: "Show me another way of grouping them." This instruction was repeated after each grouping of the objects until the subject indicated that he could think of no further way to group them.

Part V was concerned with determining in a preliminary way the basis of equivalence of the test-objects in groups. We used, (1) the group of test-objects previously made by the subject, and (2) five groups prepared by the examiner. First one and then another of the objects of a particular group was selected by the examiner and the question asked: "Could I take this away?" (from the group without disturbing its principle). The responses of the subject were noted and the object was then either removed or left in its place depending on the reaction of the subject. All objects removed from the group were replaced before proceeding with the next stage of the examination. The examiner next selected other objects from the table and offered them one at a time to the subject with the query: "Could this go here?" (with the group in question). If acceptable to the subject, the object

* Only one subject worked at such a uniform rate that no "natural" breaking point could be discerned by the examiner for interrupting the grouping behavior.

was added to the group and then another object offered. The examiner recorded the reactions of the subject and noted whether the various objects offered were accepted or rejected.

Before considering the results of this study a word of caution against casual interpretation is in order. In the first place, all cases which were given a favorable prognosis by the neurologist at the time of operation (as well as some that were not) are living and well.

TABLE 1.

ANALYSIS OF GROUPING BEHAVIOR OF NORMAL INDIVIDUALS.

Subjects.	Age.	Per cent objects grouped.	Total per cent recalled.	Per cent grouped recalled.	Per cent ungrouped recalled.	Groups created. Type I.	Type II.	Type III.	Type IV.	Grouping principles recognized.
SF	21	90	68	71	33	24	3	4	7	5
ER	23	81	76	86	30	13	6	0	5	5
ES	37	76	61	68	40	13	3	1	4	4
DC	23	69	50	58	32	18	2	0	6	5
KL	23	92	89	97	0	14	7	2	6	4
MH	36	82	71	78	36	22	6	0	4	5
CS	16	89	76	85	0	14	5	1	5	5
JM	46	63	69	97	22	12	5	0	3	4
AG	56	73	47	56	23	24	3	0	4	5
EM	12	77	39	48	7	13	3	0	4	5
GL	24	84	87	98	30	14	6	1	5	5
Average	80	67	77	23	16	5	1	5	5	

We have elsewhere noted the unusually "good" post-operative social adjustments which some of them have made.[13] Secondly, many of these cases are neurologically negative in the sense that they present no neurological "signs" of their intracranial lesion. Claims in the literature concerning the absence of psychological changes in personality following operations upon the brain must be interpreted with great caution. It deserves special emphasis that in the present investigation, perhaps for the first time in the history of modern neurosurgery, a group of neurologists and neurosurgeons have given full cooperation in making available their cases for careful study by experimental methods.

Results of Test Procedures.

In the following we shall present the results of applying the test procedures to groups consisting of normal individuals, individuals with a frontal lobe lesion, and individuals with a cerebral

TABLE 2.

Analysis of Grouping Behavior of Individuals with a Frontal Lobe Lesion.

Case No.	Per cent objects grouped.	Total per cent recalled.	Per cent grouped recalled.	Per cent ungrouped recalled.	Groups created. Type I.	Type II.	Type III.	Type IV.	Grouping principles recognized.
LEFT FRONTAL LESIONS.									
1	47	32	31	33	9	0	5	1	3
2	23	23	14	25	8	1	3	0	3
3	15	*	*	*	10	1	2	0	*
4	36	27	32	25	8	1	4	1	4
5	27	27	18	31	14	0	4	0	5
6	23	27	36	25	11	1	3	0	3
7	15	16	22	15	9	2	5	0	3
Average	27	25	26	26	10	1	4	0	4
RIGHT FRONTAL LESIONS.									
8	18	24	27	24	14	1	3	0	3
9†	50	36	39	32	11	2	5	1	5
10†	55	40	41	39	18	3	6	2	5
11	23	29	36	27	7	1	3	0	3
12	58	42	28	40	8	3	4	0	4
Average	41	34	34	32	12	2	4	1	4

* Not tested (motor aphasia).
† Lesion in "dominant" hemisphere in terms of "handedness" and "eyedness."

lesion posterior to the frontal lobes. The quantitative data are summarized in Tables 1 to 4. The significance of the various headings in these tables is indicated below.

Per Cent Objects Grouped.—In this column is indicated the percentage of the 62 test-objects "spontaneously" grouped by the various types of subjects in response to the instruction: "Place those together which seem to you to belong together" (Part II of Procedures). At the first pause or slowing in rate of grouping,

but only after a minimum of five groups had been made, the subject was interrupted and told to rest for a while. Only Cases 3, 7, and 18 required urging from the examiner to produce five groups. The

TABLE 3.

ANALYSIS OF GROUPING BEHAVIOR OF INDIVIDUALS WITH A CEREBRAL LESION POSTERIOR TO THE FRONTAL LOBE.

Case No.	Per cent objects grouped.	Total per cent recalled.	Per cent grouped recalled.	Per cent ungrouped recalled.	Groups created. Type I.	Type II.	Type III.	Type IV.	Grouping principles recognized.
TEMPORAL LOBE LESIONS.									
13	58	42	39	46	6	4	0	2	3
14	50	32	35	29	14	5	0	3	5
15	77	52	58	29	8	3	1	3	4
16	60	52	68	28	15	5	0	3	5
17	82	39	43	18	9	4	0	4	5
18	44	29	48	14	8	3	1	2	5
Average	62	41	49	27	10	4	0	3	5
PARIETAL LOBE LESIONS.									
19	84	61	67	33	22	3	2	5	5
20	86	65	74	11	16	4	1	4	4
21	100	58	58	..	15	4	0	2	5
Average	90	61	71	22	18	4	1	4	5
OCCIPITAL LOBE LESIONS.									
22	58	50	70	23	22	6	2	4	5
23	52	58	100	20	15	4	0	3	4
24	66	45	51	33	13	4	1	3	5
25	87	82	93	13	23	6	1	5	5
Average	66	59	79	22	18	5	1	4	5
CEREBELLAR LESION.									
26	87	66	72	25	24	5	1	4	5

average number (for all subjects excluding Cases 3, 7, and 18) of groups of test-objects created prior to the interruption was eight. While marked individual variations were observed in the approach of the various subjects to the task of grouping the test-objects,

it was found that only two general modes or styles of grouping could be differentiated under the conditions of the test-situation. It was also found to be characteristic of each individual to employ one mode of grouping only.

The first of these modes, Mode *A*, was characterized as follows: (a) the subject began to group the test-objects *as soon as* the instruction designed to induce grouping was given; (b) usually three or four test-objects were picked up in rapid succession and

TABLE 4.

SUMMARY OF GROUPING BEHAVIOR OF NORMAL INDIVIDUALS AND INDIVIDUALS WITH A CEREBRAL LESION.

Subjects.	Per cent objects grouped.	Total per cent recalled.	Per cent grouped recalled.	Per cent ungrouped recalled.	Groups created.				Grouping principles recognized.
					Type I.	Type II.	Type III.	Type IV.	
Normals	80	67	77	23	16	5	1	5	5
Left frontal	27	26	26	26	10	1	4	0	4
Right frontal	41	34	34	32	12	2	4	1	4
Temporal	62	41	49	27	10	4	0	3	5
Parietal	90	61	71	22	18	4	1	4	5
Occipital	66	59	79	22	18	5	1	4	5
Cerebellar	87	66	72	25	24	5	1	4	5

placed together at the periphery of the table; (c) the subject then quickly selected other objects from the table and either added these to the first group or started an additional group; (d) as groups accumulated, objects were sometimes shifted from one group to another; (e) the tempo or rate of grouping was relatively rapid throughout and extensive and rapid eye-movements [14] were frequently observed. Mode *B* was characterized as follows: (a) the subject did not immediately begin grouping the test-objects after the instruction had been given but only after a delay ranging from 5 to 15 seconds; (b) following the delay, the subject successively picked up *all* of the objects of a given group and placed them together at the periphery of the table before starting an additional group; (c) shifting of a test-object from one group to another occurred rarely; (d) the tempo of grouping was relatively slow and eye-movements made during grouping tended to be relatively slow and of small magnitude.

With reference to the above modes of grouping, our subjects were distributed as follows:

Mode *A*
Normals—ER, DC, CS, JM, AG, and EM;
Cerebral Injury Cases—1, 4, 5, 8, 11, 12, 13, 14, 15, 16, 17, 20, 21, 23, 24, and 26;
Mode *B*
Normals—SF, ES, KL, MH, and GL;
Cerebral Injury Cases—2, 6, 9, 10, 19, 22, and 25.

The average percentage of test-objects grouped by normals according to Mode *A* was 75 per cent and according to Mode *B* 88 per cent. The average percentage of test-objects grouped by patients with cerebral injury according to Mode *A* was 56 per cent and according to Mode *B* 54 per cent. It follows, therefore, that irrespective of the mode of grouping employed the normal individuals tended to group more test-objects "spontaneously" than patients with cerebral injury. From Table 4 it can be readily seen that the frontal lobe cases contributed most to this difference in that they grouped fewer test-objects on the average than other cases with cerebral injury.

Total Per Cent Recalled.—In this column of Tables 1 to 4 is indicated the total percentage of the 62 test-objects recalled by the subject when tested by the method of imminent recall (Part III of Procedure). Inspection of the averages given in Table 4 reveals the fact that the frontal lobe cases not only grouped fewer objects, but recalled fewer objects from the test-field than either the normal individuals or individuals with other types of cerebral injury.

Per Cent Grouped Recalled.—In this column of Tables 1 to 4 is indicated the percentage of those objects grouped by the subject (before being interrupted) which he subsequently recalled when tested by the method of imminent recall.

Per Cent Ungrouped Recalled.—Under this heading is indicated the percentage of those objects not grouped by the subject (before being interrupted) which he subsequently recalled in the test for recall.

Providing recall of the test-objects by the various subjects had proceeded independently of the dichotomy "grouped" versus "ungrouped" objects, the average percentage of objects recalled for these two classes should be the same. On the whole the data do not

bear out this assumption. This becomes apparent if we express the above dichotomy as a ratio:

Subjects.	Per cent grouped recalled. / Per cent ungrouped recalled.
Normal	3.3
Right frontal	1.0
Left frontal	1.1
Temporal	1.8
Parietal	3.0
Occipital	3.6
Cerebellar	2.9

From these ratios it may be seen that with the exception of the frontal lobe cases, all subjects tended to recall from the test-field a considerably larger number of *grouped* as compared with ungrouped objects. This effect of preferential recall of grouped over ungrouped objects will henceforth be referred to as the GR effect.

Since for most of the subjects more objects were originally grouped than were left ungrouped, it is of interest to compare the individual differences in the number of objects originally grouped with those in the magnitude of the GR effect. A low relationship would suggest that the GR effect was relatively independent of the original grouping performance. This would leave the matter open to further investigation as to the essential factors involved. For data such as the present, degree of relationship between two variables is commonly expressed by some measure of correlation such as Rho.

Rho values for the variables in question for (1) the normal individuals, (2) the frontal lobe cases and (3) the cases with cerebral injury posterior to the frontal lobes proved to be 0, −.33 and .33 respectively. The last two values do not differ reliably from zero. These values indicate that the GR effect is independent of the original grouping performance, and that further investigation is required to determine its exact nature.

It is of considerable interest that the GR effect is either absent or markedly diminished in the recall performance of the frontal lobe cases. To a less extent this is also true of the temporal lobe cases. All our temporal lobe cases had residual tumor tissue which

may have involved the frontal lobes, but until such time as autopsy findings are available the diminished GR effect in these cases can scarcely be interpreted.

We have thus far considered results which indicated that individuals with a frontal lobe lesion, when given the task of sorting or grouping certain heterogeneous stimulus objects according to individual preference, in comparison with normal individuals and individuals with a cerebral lesion posterior to the frontal lobes, (1) employed fewer objects "spontaneously," (2) recalled fewer objects after an interval of five minutes, and (3) manifested little or no differential recall of grouped as opposed to ungrouped objects (GR effect). There remains for consideration, however, the question as to whether the various individuals differed as to the number and type of organization of the groups of test-objects created in our test-situation. It was for the purpose of securing a preliminary answer to this question that use was made of the method of equivalent and non-equivalent stimuli. Examination of the groups made by the various subjects in terms of this method revealed the existence of four general types. Examples of these four types are represented in a condensed form in Table 5. The Arabic numerals in the table indicate individual test-objects as shown in Fig. 3.

Type I.—This type of group was differentiated objectively by the fact that subtraction or removal of some of its content (without disturbing its organizing principle) was rejected by the subject. We may consider as an example of this type a group created by subject MH which is included in Table 5. Its content consisted of a metal pulley, a glass stopper, a metal padlock, a picture of a house key, and a small metal key. The subject accepted subtraction from the group of the small metal key and the picture of the house key. He rejected subtraction of the metal pulley, the glass stopper, and the padlock. While the five objects were objectively equivalent in the sense of belonging to the same group for the subject, it thus became apparent that they were not equally dispensable to the group without disturbing its organizing principle.

Of the additions to the group offered by the examiner the subject accepted (as not disturbing the organizing principle) another small metal key, an electric light socket, a miniature light bulb, and a flashlight bulb. He rejected (as disturbing the organizing principle) a metal thimble, a metal spoon, some blue yarn, a piece

TABLE 5.

REPRESENTATIVE GROUPS CREATED BY THE VARIOUS SUBJECTS ALONG WITH THEIR

TABLE 5.

REPRESENTATIVE GROUPS CREATED BY THE VARIOUS SUBJECTS ALONG WITH THEIR EQUIVALENT AND NON-EQUIVALENT STIMULI.

The Arabic numerals refer to test-objects shown in Fig. 3.

Type.	Content.	Name given by subject.	Subtractions accepted.	Subtractions rejected.	Additions accepted.	Additions rejected.
I	54, 43, 3, 24, 53	"Hardware"	53, 24	54, 3, 43	58, 15, 59, 33	46, 13, 16, 26, 41, 57, 52, 60, 17, 28, 6, 51, 4, 11
I	46, 30, 7, 8, 16	"Sewing box"	30	46, 7, 8, 16		29, 40, 42, 26, 56, 14, 11, 10, 55, 27
I	1, 6, 2, 51, 40, 55, 27	"Dressing table"	51	1, 6, 2, 40, 55, 27	62, 29, 17, 38, 32	3, 45, 20, 24, 28, 52, 47, 18, 59, 15, 60, 42
II	11, 5, 49, 35, 24	"Pictures"	11, 5, 49, 35, 24			45, 12, 37, 20, 34, 42, 36, 17, 18, 38, 60, 57, 15, 19, 28, 25, 30
II	51, 1, 6	"Containers"	51, 1, 6		62	23, 15, 46, 27, 4, 32, 10, 2, 13, 26, 43, 61, 39, 59, 22, 21
III	5, 50, 9, 42	"Square things"	5, 50, 9, 42		20, 17, 37, 12, 11, 43, 15, 10, 54, 59, 2, 21, 60, 28	7, 16, 30, 51, 62, 19, 58, 3, 57, 46 22, 35, 49, 34
III	44, 48, 9, 23, 30 42	"Red things"	44, 48, 9, 30, 23, 42		28, 20, 7, 39, 19, 61, 11, 5, 49, 35, 24	29, 15, 12, 17, 40, 51, 60, 26, 59, 31, 50, 45, 18, 22
IV	10, 57, 54, 60, 3	"Metal objects"	10, 57, 54, 60, 3		29, 55, 15, 52, 43, 59, 27, 13, 53, 39, 58, 19, 7, 8, 16, 21, 33, 56, 26, 46	34, 9, 1, 2, 42, 38, 18, 50, 4, 36, 11, 51, 62, 40, 23, 44, 14, 28, 47, 41, 17, 24
IV	44, 4, 61, 36, 48, 18	"Round things"	44, 4, 61, 48, 36		62, 21, 1, 6, 27, 46, 54, 31, 59, 10, 2, 41, 40, 28, 47, 25, 19, 26, 23, 39	50, 15, 57, 9, 12, 20, 37, 42, 30, 56, 60, 7, 29

of rubber-covered wire, a rubber grommet, a metal knife, a metal fork, a metal puzzle, a piece of coarse sandpaper, a wax crayon, a bottle labeled "Bergamot," a glass jar, a wooden pulley and a picture of a bell. By way of summary it may be said, therefore, that certain additions or subtractions did not disturb the organizing principle whereas others did. In examining the characteristics of the objects in the equivalent and non-equivalent situations, such as size, shape, material, use, color, hardness, and weight, it became apparent that none of these provided the basis of organization for the group in question. The subject "named" the group "hardware."

Type II.—This type of group was differentiated objectively by the fact that while all subtractions of content of the group were accepted by the subject, all additions to the group were rejected. One of the examples of this type given in Table 5 is a group created by Case 25. Its content consisted of a picture of a bell, a picture of a rooster, a picture of a rabbit, a picture of a doll, and a picture of a house key. Subtraction of each of these objects from the group was accepted without disturbing the principle of the group for the patient. Among additions to the group offered by the examiner, all of which were rejected by the patient, were a card labeled "Hairpin," a cancelled foreign postage stamp, a card labeled "Pipe," a face-card playing card, a card with an abstract design (black dot and crescent), a piece of red paper stock, a ping-pong ball, a piece of coarse sandpaper, a smoked glass lens, a piece of fine sandpaper, a metal puzzle, a metal knife, an electric light socket, a pipe stem, a wax crayon, a wax candle, a piece of red cloth. The subtractions from the group accepted by the patient indicate that the various objects were equally dispensable without disturbing the basis of the organization for the group. Since characteristics as to size, shape, color, material, weight and use found in this group and in the additions rejected from this group did not seem to be essentially different, such characteristics apparently did not constitute the basis of organization of the group in question. The fact that no additions of test-objects were accepted made it impossible to determine objective extensions of the equivalence range of the group without drawing upon material outside that provided in the test-situation. The patient "named" the group in question "pictures." An examination of the additions to the group

rejected by the patient shows that "pictures" for her did not include such objects as abstract designs, printing, and colored designs although these were objectively similar in size, shape and material to objects of the group in question.

Type III.—This type of group was differentiated objectively from the types thus far considered in that all subtractions of content were accepted, and that of the additions to the group offered by the examiner, some were accepted whereas others were rejected. A sample of this type given in Table 5 is a group created by subject ES. The content consisted of a picture of a rooster, a black cube, a multicolored cube, a square piece of red paper stock. Subtraction of each of these objects from the group was accepted by the subject without disturbing the principle of the group for him. Among the additions to the group offered by the examiner, the subject accepted a face-card playing card, a piece of coarse sandpaper, a card labeled "Pipe," a cancelled foreign postage stamp, a picture of a bell, a small padlock, a miniature electric light socket, a metal whistle, a metal pulley, a miniature electric light bulb, a glass stopper, a metal jar lid, a metal puzzle, and a wax crayon. He rejected some pink yarn, some blue yarn, a piece of red cloth, a glass jar, a round wooden box, a pipe stem, a small metal key, a house key, a metal knife, a metal thimble, a small cork, a picture of a doll, a picture of a rabbit, a card with an abstract design (black dot and crescent). The subject had "named" the group in question "square things." Included among the additions accepted were such objectively round objects as the metal jar lid and the wax crayon. Likewise, among the additions rejected were objects similar in objective size, shape and material to some of the content of the group in question. Groups of this type proved to be objectively unstable upon examination of their equivalent and non-equivalent stimuli.

Type IV.—Groups of Type IV were differentiated objectively from the foregoing types with respect to stability and the magnitude of the equivalence range. Two groups of this type created by Case 10 are given in Table 5. The content of one of these consisted of a metal whistle, a knife, a metal pulley, a metal puzzle, and a house key. Subtraction of each of these objects from the group was accepted by the patient as not disturbing the organizing

principle of the group for her. Of the additions to the group offered by the examiner, the patient accepted a metal hairpin, an earring, a miniature electric light socket, a metal fork, a small padlock, a miniature electric light bulb, a lipstick, a metal spoon, a small metal key, a candle holder, a small metal key, a pipe stem, some pink yarn with a metal tab attached, some yellow yarn with a metal tab attached, a metal jar lid, a flashlight bulb, some blue yarn with a metal tab attached, a doll slipper with a metal buckle, a piece of rubber-covered wire bared at each end, and a metal thimble. The patient rejected a card with an abstract design (black dot and crescent), a multicolored cube, a glass bottle labeled "Camphor," a glass stopper, a piece of red paper stock, a piece of fine sandpaper, a smoked glass lens, a black wooden cube, a wooden pulley, a ping-pong ball, a picture of a bell, a glass jar, a round wooden box, a bakelite bracelet, a pipe bowl, a red poker chip, a bone chess pawn, a wax crayon, a piece of thick roundish stick, a rubber grommet, a piece of coarse sandpaper, and a picture of a house key. Upon examining the characteristics of the additions accepted and rejected by the patient, it became apparent that all of the objects accepted as additions to the group in question were either part or all metal in material whereas no object rejected by the patient was even part metal. The patient in this instance "named" the group in question "metal objects."

The relative frequency of these four types of groups as created by the various subjects in this investigation is shown in the corresponding columns of Tables 1 to 4. The most striking feature is the divergence of the frontal lobe cases both as to the total number of groups created and the relative frequency of Types III and IV. It will be recalled that groups of Type III were characterized chiefly by objective instability and groups of Type IV by objective stability. In comparison with the normal individuals, the frontal lobe cases produced a relatively large number of groups characterized by objective instability and a relatively small number of groups characterized by objective stability.

Grouping Principles Recognized.—That the frontal lobe cases investigated here did not differ markedly from the other types of subjects in recognizing (in the sense of "naming") organizing principles of groups submitted by the examiner is revealed by

inspection of the corresponding column in Tables 1 to 4. The five groups submitted (Part V of Procedures) were comprised of the test-objects here referred to in terms of the Arabic numerals shown in Fig. 3 (Group A: 4, 36, 41, 40, 62, 1, 18, 44; Group B: 9, 42, 24, 50, 12; Group C: 3, 29, 54, 57, 10, 60; Group D: 41, 22, 55, 14, 53; Group E: 30, 42, 7, 44, 9, 28). This aspect of grouping behavior has been made the subject of a separate investigation and will be given further consideration elsewhere.[22]

Discussion of Results.

In comparison with the performances of the normal individuals, the greatest deviation in the performances of the patients with a cerebral injury occurred in the grouping behavior of individuals with a lesion in one frontal lobe, regardless of whether the primary lesion was on the right or left side of the brain. We shall therefore confine our discussion to cases with injury in the frontal lobe.

Reduced Number of Objects "Spontaneously" Employed.—Inspection of Tables 1 to 4 reveals that in comparison with the other types of subjects our frontal lobe cases tended to employ *fewer* test-objects in their first "spontaneous" grouping. This tendency was found to be relatively independent of the mode of grouping used (*i.e.*, whether a rapid, "trial-and-error" or a deliberate, "planned" approach). It should be recalled that the "task" was not limited as to length (or quantity) by the instructions. Thus the percentage of the test-objects grouped by the subject before slowing markedly or before pausing "spontaneously" represents within the reliability of the criterion an individual performance unit. The performance unit "spontaneously" produced by our frontal lobe cases tended to be smaller on the average than that produced by our other subjects. It is of interest that examination of the Rorschach performances of such cases reveals evidence of "constriction."[4]

Reduction in Recall of Objects from the Test-field.—When tested for recall after an interval of five minutes, our frontal lobe cases recalled on the average considerably fewer objects from the test-field than did the other types of subjects. While this tendency towards a reduction in recall is marked in the results of the present investigation, it should be noted that the test for recall made was

in terms of material of a particular kind, a single interval of delay (five minutes) and a single indicator of recall (verbal). Thus only further investigation can reveal to what extent the tendency noted here is a general one for memory functions of individuals with a frontal lobe lesion.[9, 21]

Absence of GR Effect in Recall Performance.—A relationship of certain types of content to availability in recall was demonstrated for the first time in the present investigation. In normal individuals and in patients with certain cerebral lesions this relationship is reflected in "preferential" recall of grouped as compared with ungrouped objects (*i.e.*, objects which had been grouped by the subject prior to the test for recall). Elsewhere in this report we have referred to this relationship as the GR effect. Incidentally, an effect somewhat similar to the GR effect has been demonstrated for reactions to situations carried to various stages of "completion."[25] The fact that in comparison with the other types of subjects investigated, the recall performances of our frontal lobe cases are characterized by a marked reduction or absence of the GR effect implies that the number of objects recalled from the test-field tended to be uninfluenced by the fact that some of the objects had been grouped prior to the test for recall whereas others had remained ungrouped. In the GR effect, forces are operative which determine selective recall of certain types of content. The behavior of the frontal lobe cases indicates that the GR effect can be reduced and even eliminated by a cerebral lesion occurring in some part of the brain.

Differences in the Distribution of the Groups Produced.—The frontal lobe cases tended to produce a smaller total number of groups despite the fact that the time they were exposed to the test-field was on the average thirty per cent longer than for other subjects. Tables 1 to 4 also indicate differences in the distribution of the various types of groups produced. By examining further the nature of the organization of the various type groups we may hope to throw some light on functional equivalence,[18] that is, on the factors accounting for the various groups of equivalent and non-equivalent situations.

Examination of the distributions of the groups produced by the various subjects (Tables 1 to 4) reveals that for every subject the

groups most commonly produced "spontaneously" were those of Type I. In this respect, our frontal lobe cases present no exception. They are, however, differentiated in our findings from the other types of subjects in the relative frequency of Types III and IV. Our frontal lobe cases produced relatively *more* groups of Type III and relatively *fewer* groups of Type IV. From our description of Type III it is apparent that groups of this type involve organizing principles not easily defined. In further publications an attempt will be made to determine the organizing principles underlying groups of this type.[16] With reference to Type IV our normal individuals produced on the average five such groups (Table 1). Eight of our frontal lobe cases, on the other hand, produced no groups of this type (Table 2) while three cases (Cases 1, 4 and 9) each produced one such group and one case (Case 10) produced two (*cf.* the examples of this type given in Table 5). In every instance the Type IV groups produced by the frontal lobe cases were produced after groups of another type. (This was also true in many instances in the performances of the normal individuals and patients with a lesion elsewhere in the brain). The production of Type IV groups by patients known to have a large lesion in one frontal lobe under the conditions of the present investigation, calls into question the conclusion of Goldstein,[8, 10] and Weigl [24] that such performances cannot occur since such individuals cannot adopt the "categorical attitude." In this connection the performances of Case 10, cited above, may be summarized as follows: (1) Groups of Type IV were "spontaneously" created by an individual with a large frontal lobe lesion; (2) such groups were created *after* groups of other types had been created by the patient; (3)the capacity for "shifting to an alternative way of grouping" was present even when such shifting was in the direction of groups of Type IV. Thus it must be inferred either that (1) the *attitude* requisite for such shifting in grouping performance could, under certain conditions, be "voluntarily adopted" by this patient or (2) the factor of attitude was not the dominant one in determining the organization of the groups created by Case 10.

In the foregoing we have focussed our discussion on the results obtained in patients with lesions in the frontal lobe. It remains to be seen, of course, whether future work will support the finding

that the grouping behavior of frontal cases is characteristically different from that of other cerebral cases. We insist that for an analysis of functions it seems at the present time more important to study the existence or non-existence of certain types of grouping behavior in cerebral cases in general than to make statements about the symptomatology of different lobes of the brain.

BIBLIOGRAPHY.

1. Ach, N.: Ueber die Willenstätigkeit und das Denken, pp. X + 294. Göttingen: Vandenhoeck & Ruprecht, 1905.
2. Bailey, P.: Intracranial tumors, pp. XXII + 475. Springfield: Thomas, 1933.
3. Berry, R. J. A.: A cerebral atlas, pp. XXII + 425. London: Oxford Univ. Press, 1938.
3a. Bouman, L., and Grünbaum, A. A.: Experimentelle-psychologische Untersuchungen zur Aphasie und Paraphasie. Zeitschr. f. d. ges. Neurol. u. Psychiat., 96: 481-538, 1925.
4. Brosin, H. W., and Halstead, W. C.: Rorschach performances of patients with cerebral injury. Presented on the program of the Midwestern Psychological Association, Madison, Wisconsin, May, 1938. (To be published).
5. Dearborn, W. F., Anderson, J. E., and Christiansen, A. O.: Form board and construction tests of mental ability. J. Ed. Psychol., 7: 445-458, 1916.
6. Franken, A.: Bilderkombinationen. Ein Beitrag zum Problem der Intelligenzprüfung. Zeitschr. f. ang. Psychol., 12: 173-329, 1917.
7. Fuchs, W.: Untersuchung über das Sehen der Hemianopiker und Hemiamblyopiker. II. Die totalisierende Gestaltauffassung. Zeitschr. f. Psychol., 86: 1-143, 1921.
8. Gelb, A., and Goldstein, K.: Ueber Farbennamenamnesie. Psychol. Forsch., 6: 127-186, 1925.
9. Goldstein, K.: Die Lokalisation in der Grosshirnrinde. Handb. d. norm. u. path. Physiol., vol. X: 600-842, 1927.
10. ———: The significance of the frontal lobes for mental performances. J. Neurol. and Psychopathol., 17: 27-40, 1936.
11. Grinker, R. R.: Neurology, pp. 979. Springfield: Thomas, 1934.
12. Halstead, W. C.: The effects of cerebellar lesions upon the habituation of post-rational nystagmus. Comp. Psychol. Monogr., 12: (No. 56), 130, 1935.
13. ———: Experimental analysis of the effects of localized cerebral injury in man. J.A.M.A., 108: 1624, 1937.
14. ———: A method for the quantitative recording of eye movements. J. Psychol., 6: 177-180, 1938.
15. ———: Behavioral effects of lesions of the frontal lobe in man. Arch. Neurol. and Psychiat., 42: 780-783, 1939.

16. ———: Psychological analysis of a case of prefrontal lobectomy. (To be published).
17. Klüver, H.: Visual disturbances after cerebral lesions. Psychol. Bull., 24: 316-358, 1927.
18. ———: The equivalence of stimuli in the behavior of monkeys. J. Genet. Psychol., 39: 3-27, 1931.
19. ———: Behavior mechanisms in monkeys, pp. XVII + 387. Chicago: Univ. of Chicago Press, 1933.
20. ———: The study of personality and the method of equivalent and non-equivalent stimuli. Character and Personality, 5: 91-112, 1936.
21. Lashley, K. S.: Brain mechanisms and intelligence, pp. XIV + 186. Chicago: Univ. of Chicago Press, 1929.
22. Settlage, P. H., and Halstead, W. C.: Responses to serially presented stimuli in the development of "concepts." A study of patients with cerebral injury. (To be published).
23. Stern, W., and Wiegmann, O.: Methodensammlung zur Intelligenzprüfung von Kindern und Jugendlichen. Zeitschr. f. ang. Psychol., 1920, Beiheft *20*. Pp. V + 256.
24. Weigl, E.: Zur Psychologie sogenannter Abstraktionsprozesse. Zeitschr. f. Psychol., 103: 1-45, 1927; ibid, 257-322.
25. Zeigarnik, B.: Ueber das Behalten von erledigten und unerledigten Handlungen. Psychol. Forsch., 9: 1-85, 1927.

DISCUSSION.

Dr. Roy R. Grinker (Chicago, Ill.).—I don't know why Dr. Halstead has given me the honor of discussing this paper, except perhaps because of the fact that some years ago I had a minor rôle in getting him interested in the problem. My recompense for this is indicated in the beautiful work which he has presented to you today.

I think that the amount of experimentation which has been done on animals by Klüver and Jacobsen has been sufficiently worked out, sufficiently controlled, but the application to the human subject has lagged behind. The pioneer work of Goldstein and others has given us a great deal of insight into the difficulties that occur from various lesions in the human brain; but I think Dr. Halstead has made a tremendous advance with his very careful scientific studies, particularly of exactly localizable lesions in the human brain.

He is fortunate in being associated with a group who are equally interested in the problems, so that surgical lesions can be described exactly by the surgeon at the operation and by the neuropathologist indicating the particular areas of the brain removed. The results of such cooperation are of much greater value than psychological studies alone, without anatomical controls.

It is, of course, important to recognize, as he has stated in this paper, that there is one problem which is brought out very simply without much technical confusion. The results are clear-cut in the general statement which he

has made. It is obvious, as he promises us, that he must sharpen his localization and go further in bringing out the exact relationship between smaller areas of removal and the particular psychological behavior.

I am particularly pleased by the fact that Dr. Halstead has also not neglected the emotional problems that occur in the presence of the frustrating situation that an individual with this categorical disturbance shows. He is studying not only what the patient cannot do, but what he does do under certain stress. It seems to me that some of the reports which have lately come from certain Russian laboratories in relation to the problem of schizophrenia—I am referring to the Vigotsky test—indicated the same type of research of analysis of the category behavior.

I have no doubt that such exact studies as Dr. Halstead has made will at some future date be applicable to the study of functional disturbance in which emotional factors enter into the problem—disturbance of behavior, and the various psychoses.

Dr. Spafford Ackerly (Louisville, Ky.).*—Dr. Halstead's paper is a very interesting contribution to the field. He is particularly fortunate in being able to study Dr. Bailey's carefully selected series of posterior and anterior lesions of the brain in man, checked against normal controls. It is lack or such coordinated research in the past that has retarded real progress in clearing up the confusions of misconceptions that have crept into the literature from earliest times. Animal experimentation the last five years, such as the work of Lashley, Fulton, Jacobsen and others has clarified many concepts and greatly helped to simplify clinical research.

Frontal lobe extirpation in apes showed conclusively that while the animals could enter into simple performance such as pulling a lever to get food, they could not carry out a series of acts leading to the food. They lacked ability to take in the total situation apparently, as I observed in studying Dr. Spurling's case of bilateral frontal lobe damage (in 1933). Up to that time concepts of integration and synthesis were the only ones mentioned in the literature. Drs. Fulton and Jacobsen labelled the defect in these animals as loss of memory for immediate events, leaving remote memory intact. These animals could manipulate what was in sight at the moment very well, but could not remember where the food was placed a few seconds before.

Then there was confusion concerning such terms as "distractibility." One observer would find it and another not. What had that to do with memory or lack of interest, etc.?

Drs. Goldstein and Katz in a paper on Pick's Disease in 1937, did much to state the issues clearly. Dr. Goldstein said, "While we must agree in considering performances of the higher type as impaired and those of the more primitive as still being retained, such an interpretation would be incorrect if it meant that so-called elementary performances are preserved and that only the syntheses of the elementary performances forming the higher processes are impaired. When we speak of higher and lower performances,

* Read by Dr. Wm. K. Keller in Dr. Ackerlys absence.

we mean a performance is embodied in a higher (abstract) or lower (concrete) attitude. Thus, a patient cannot be said to have lost the ability to attend or to form combinations, etc., but he is unable to be attentive in a situation which demands an abstract attitude. In concrete situations all these functions may be normally retained." Then he goes on to say, "From this point of view, it appears understandable that one can find, in the same patient, symptoms that seem so contradictory,—on the one hand abnormal excitability, unrest, distractibility, lack of interest, bad memory, lack of orientation in space and time, and on the other hand, dullness, lack of initiative, abnormally fixed attention, an intense tendency to cling to some objects or actions, and good memory in certain situations." While these issues are stated, they are still far from solved.

This brings us to Dr. Halstead's work in adapting for man Klüver's method of equivalent and non-equivalent stimuli, or the process of grasping, or abstracting essential similarity in the presence of dissimilarity, or the converse which he states is the essence of normal thinking. The very simplicity of Dr. Halstead's experiment intrigues us. When we look back over some of the patients who have had a good deal, if not most, of the prefrontal lobe tissue removed and know how cleverly some have managed to carry on their daily living, it is rather hard to believe that they cannot make these simple groupings of the test objects. But Dr. Halstead's findings are especially significant since these patients have been studied from many angles, both psychological and physiological, so that the fruits of his studies over the last five years have had an opportunity to ripen. From individual cases it would be impossible to draw conclusions, but his average figures for the series are quite striking.

I wish Dr. Halstead would do more than just state his findings. Who is better qualified than himself to draw conclusions and try to interpret his own findings? I would like to ask Dr. Halstead if this defect showed itself in the patients' behavior in the hospital, or at home with members of the family and others? Is not the basis of good judgment the ability to appreciate likenesses and differences? Is not the ability to group things one of the organism's economizing processes?

I regard this paper as a real contribution to the growing knowledge of brain functioning.

DR. HALSTEAD.—I am extremely grateful to Dr. Grinker for his generous appraisal of our activities.

Dr. Ackerly suggested that we have been somewhat conservative in interpreting our findings, and I must reply that if I knew less about these cases, it would have been possible to draw more conclusions. It is true, as we have pointed out, that the patient with the frontal lobe lesion may get along well in the hospital; he may hold a job; he may adjust readily to the members of his family and to his friends, who may not notice the slightest difference in him. He may even convince his physician that there has been no change, if we are to judge by some of the literature in this field.

However, it is equally true that when confronted by specific test situations such as we have described, and by others with which pre-school children have no difficulty, he may fail badly.

Dr. Ackerly raised the interesting question whether the basis of good judgment is the ability to appreciate liknesses and differences. He might have extended this question to include the ability to distinguish likenesses or differences in terms of a concrete attitude or an abstract attitude, in the sense of Dr. Goldstein, whom he mentioned. From our studies, it is clear that the point is not that certain similarities and certain differences are or are not recognized by the patient—they are not abstract or concrete because they are recognized—but rather, what similarities, how many and under what conditions? In short, what is the psychological structure of the categories that are availabe to the patient in terms of the inner and outer fields?

This is the question to which our report was directed, and upon which our knowledge of the functions of the frontal lobe will ultimately depend.

SYPHILIS IN SERUM NEGATIVE FEEBLEMINDED CHILDREN.*

A Histologic Study in Meningoencephalitis Syphilitica and in the Nissl-Alzheimer Endarteritis.

By CLEMENS E. BENDA, M. D., Wrentham, Mass.

The rôle which congenital syphilis plays in the causation of mental deficiency has always been a subject of much discussion. Those investigators who rely on a positive outcome of the Wassermann reaction are mostly inclined to deny the importance, especially if the blood Wassermann is the only criterion. Penrose,[1] however, considers the frequency of congenital syphilis in the feebleminded population, judged by positive reactions, about 10 per cent. If the Wassermann test is not thought to be of definite value, speculation finds an open door, according to the criteria on which any investigator depends and no general agreement has been reached.

The introduction of family studies has advanced our knowledge to a great extent. Raeder[2] studied several families in which one or both parents had syphilis and he proved that congenital syphilis may be present in the offspring in spite of the fact that sero reactions were to be found negative.

In order to gain more insight into the problem, a careful microscopic study of the autopsy material of the Wrentham State School, an institution which houses 2000 patients, has been started. This study is far from being completed and it will not be until a sufficient number of autopsies have been studied that I shall be in a position to enter into a discussion of the percentage of syphilis found and the importance of its pathology. The autopsy material is still too small to draw conclusions as conjectures based on an insufficient number are misleading.

* Read at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

From the Research Department for the Study of Mental Deficiency, Wrentham State School, Wrentham, Mass., and the Department of Neuropathology, Harvard Medical School, Boston, Mass.

Because of limitations in space and in the number of pictures, it is the aim of this paper to present two well studied instances out of a number of cases to demonstrate two types of manifestations of congenital syphilis. It is my purpose to call attention to a type of pathology which decidedly indicated congenital syphilis and which was found in cases previously considered to be due to birth injury. Both cases which will be presented were sero-negative. This observation is in accordance with cases of the same type which were previously published in literature by other investigators. A comparative study of the three groups of congenital syphilis (sero-positive instances which are mostly cases of general paresis, children of parents whose infection was ascertained, and cases in which the type of pathology indicates a specific infection) will eventually lead to definite conclusions regarding the importance of congenital syphilis in the causation of mental deficiency.

CASE I.—This female child was born prematurely at seven months; labor was easy, and of two hours duration. She was the first child and there was no history of stillbirths or miscarriages given. There is one other child five years younger than our patient about whom nothing is known.

Two weeks after birth, the baby was admitted to a Children's Hospital where she remained for five months. During this period, she had a very irregular temperature going from 95° to 104°. In the 19th week of her hospitalization, a pyelitis appeared. It was noticed that the baby had a severe hypertonicity and exophthalmos. She lay with her head retracted and spine arched with a moderate degree of opisthotonus. When supported under the axillæ, the legs assumed a scissors position. The hospital diagnosis was prematurity, chronic pyelitis, hypertonicity and question of a birth injury. The Wassermann reaction was negative.

She began to talk at about two years of age, could not use her limbs properly and had convulsive seizures. She was reexamined at an age of 4 years, 8 months in one of the state schools for the feebleminded. Her I. Q. was 77 but she appeared seriously handicapped by her spastic diplegic paresis.

At the age of five years, she was admitted to the Wrentham State School. She adapted herself very well. She was able to talk and understand everything said to her. She made no attempt to stand as her legs were decidedly crossed.

Neurologically, she had alternating strabismus; the pupils were round and equal, but reacted sluggishly to light. The knee jerks were exaggerated and there was marked spasticity and crossing of legs.

She died at an age of eight years with clinical signs of meningitis and increased inter-cerebral pressure.

Five hours after death, an autopsy was performed but unfortunately had to be restricted to the central nervous system. The head appeared asymmetrical, the right side of the skull being smaller than the left. Much spinal fluid was dropping out under pressure when the brain was removed. The brain weighed 890 grams. An area of pachymeningitis with yellowish-gray discoloration was found upon the inferior surface of the brain and brain stem. The ventricles were enlarged.

A microscopic study of the central nervous system revealed pathology spread all over the brain and spinal cord. There was a lymphatic meningitis upon the brain stem and upon the region between pons and optic chiasma. The infiltration consisted of lymphocytes mingled with some plasma cells imbedded in a network of connective tissue. The arteries showed a marked periarteritis with some endarteritic alterations. There was also phlebitis.

An analysis of the area of acute lymphatic meningitis would certainly not decide the question what kind of meningitis was present but a study of the whole central nervous system revealed that the acute meningitis was superimposed upon a chronic meningo-vascular encephalitis. Sections through the spinal cord including the dura revealed that the small vessels of the dura showed infiltration which extended beneath the sheaths of the dura. There was also a general thickening of the dura. Pia and arachnoid showed a chronic lymphatic pachymeningitis mingled with many plasma cells and imbedded in a network of increased connective tissue fibers. The infiltration penetrated into the nerve sheaths of the peripheric nerves producing a perineuritis. A study of the smaller and medium sized vessels appeared of special interest. All these vessels revealed periarteritis and a marked endarteritis of the Heubner type. In some vessels, the lumen was almost occluded. A higher power study of some arteries upon the medulla revealed miliary gummata between intima and media. There was also periarteritis with many plasma cells and few Langerhan's giant cells. Silver stains showed that the cells were suspended in a dense network of proliferated connective tissue fibers. The arteries penetrating into medulla and brain tissue showed marked cuffing of lymphocytes and plasma cells. Silver stains revealed again that there was a remarkable increase in silver fibers. All the smaller capillaries showed marked sclerosis. The same sclerosis of the capillaries was visible all over the cortex. Not only the inferior surface of the brain but also the convexity showed a chronic arachnoiditis with increase of fiber tissue and lymphatic infiltration of the vascular system. The brain tissue was devastated of nerve cells. Most of them had disappeared or were sclerotic. The nerve cells were replaced by astrocytes. The ventricle walls, especially the fourth ventricle and the aqueduct showed widespread ependymitis granulosa. The choroid plexus were atrophic.

If I summarize these observations, I may say that the cause of death was a lymphatic meningitis, superimposed upon a chronic meningoencephalitis of long duration. Decisive for the diagnosis of congenital syphilis were periarteritis and endarteritis gummosa; the

Heubner type of arteritis present in arteries of the spinal cord; a phlebitis, capillary sclerosis, lymphatic infiltration of the dura, chronic arachnoiditis with lymphatic infiltration of the meningeal vessels penetrating into the cortex and spinal cord, ependymitis granulosa, and degeneration of nerve cells with replacement through astrocytes. All these pathologic signs were present in confusing abundance and although no one of these signs would suffice for establishment of a definite diagnosis, the combination of all these alterations, above mentioned, renders overwhelming evidence for the presence of congenital syphilis. There is, in my opinion, no other disease known which could produce the same combination of pathology. On the other hand, the occurrence of these signs is sufficiently observed in cases of undoubted syphilis thus justifying the filing of the first case into the same group.

The next case represents another type of pathology which is generally considered as the sequel of syphilitic infection. Since the so-called Nissl-Alzheimer endarteritis of the small brain vessels offers a very impressive and definite histologic picture and the literature to which I shall return later does not report many cases of this type, it may be worth while to dwell on this case a little more in detail.

Case 2.—This case differed from the preceding case insofar as there was in the history the remark that the mother was syphilitic. It was, however, not possible to ascertain more information. The father and maternal grandfather of the patient were alcoholic. There were two brothers, the history of whom is unknown. There were four more pregnancies; two ended in miscarriages, one sister died at the age of two days, and the other at the age of five and one-half months.

Our patient was born at full term, breech-presentation. She began to walk at the age of two and one-half years and is said to have talked at two years. The mother did not realize the retardation of the child until the school authorities brought it to her attention.

At the age of eight years, the child was carefully studied in an outstanding hospital. The physical examination showed poor development and posture, 13 per cent underweight and mental deficiency. The patient had an abnormal hair growth over the whole back, internal strabismus and nystagmus. The eye condition showed sluggish papillary reaction, snuff-fundi. The conclusion of the examination was that there was an intracranial lesion of some sort. The statement was made that "The snuff fundi are strongly suggestive of congenital syphilis though they do not absolutely define it." The final conclusion of the Superintendent was: "I report that the child has

an intra-cranial lesion of unknown character with strong suggestion of both congenital lues and thyroid failure."

The child was admitted to our institution at an age of ten years. At that time she appeared well developed and well nourished. However, her height was only 3′ 7½″, the head measurements show a microcephaly of 17¾″ circumference, and her weight was 40 pounds.

During the next years, the child failed progressively. The expression of the face was that of an old woman and she exhibited the condition known as progeria. In the last year of her life, at an age of 16 years, she developed the habit of falling down and stiffening her body or becoming limp, and she uttered strange sounds. The last weight taken was 38 pounds, when she was almost 17 years of age.

From the autopsy findings, I shall report only a few data. The calvarium weighed 330 grams and the skull bone measured 8 mm. in thickness, which is extremely thick in proportion with the smallness of the head. After removal of the calvarium, much spinal fluid dropped out. The dura was adherent to the calvarium—especially upon the frontal lobes. The brain appeared hard in consistency. Between the brain and the inside of the skull, there was a space of about half an inch filled with spinal fluid. The arachnoid was of grayish-white color and thickened. The brain weighed only 600 grams. The ventricles were enlarged.

The microscopic examination revealed pathologic alterations of extreme degree. The dura spinalis was thickened and in stained preparations, the broad band of dura appeared dotted. These small black spots represented blood vessels which showed intima and adventitia proliferation, the cells penetrating into the spaces between the dura sheaths. The lumen of many vessels was narrowed or even occluded. There was also a cellular proliferation of the vasa vasorum in larger arteries. Around the convexity of the brain and upon the inferior surface, pia and arachnoid were greatly thickened, filling the spaces between the atrophic convolutions. Every smaller and medium sized vessel showed the same proliferation of intima and adventitia, forming dense granulation tissue. The small vessels revealed a swelling and proliferation of the endothelium; the capillaries showed increased silver fibers. In the basilary artery, two gummatous granulations were found between intima and media.

Besides these vascular alterations, the first description of which attracted much interest because of the peculiar appearance of those vascular "parcels," the case revealed numerous alterations which added new evidence to the specificity of that process. The wall of the fourth ventricle was covered with mushroom-like granulations representing an ependymitis granulosa. Both nuclei lentiformis revealed a status spongiosus in which the brain tissue had mostly disappeared and only a network of capillaries with many sprouts had survived. The remaining tissue was rich in large protoplasmatic glia. The cortex of the brain showed atrophy and degeneration of nerve cells. The parenchyma was replaced by greatly increased astrocytic glia. The lateral ventricles were enlarged and their walls gliotic. The surface of the

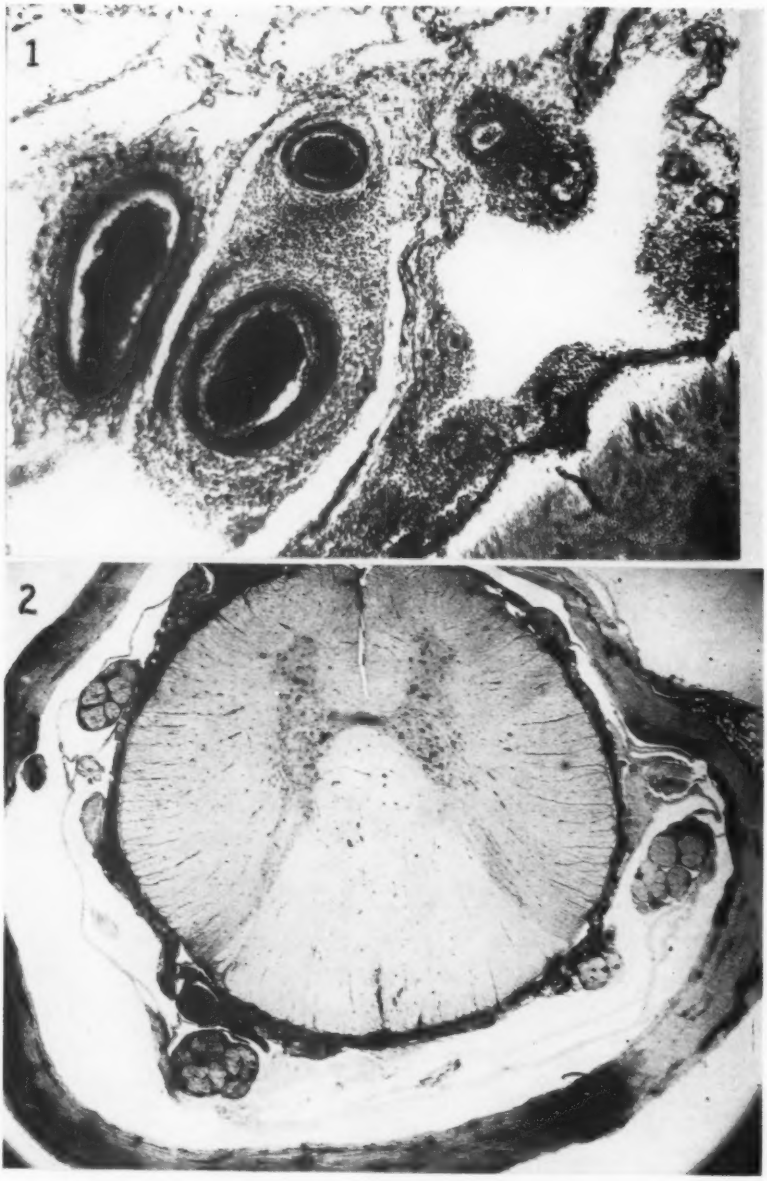
brain stem and medulla appeared felt-like from abundant gliosis. The choroid plexus were atrophic.

The pathology of this case is very characteristic and impressive. The outstanding feature is an extreme proliferation of endothelium and adventitia cells which form dense conglomerations around the vessels and penetrate into the meningeal sheaths. Moreover, the smaller vessels are increased in number, forming vascular "parcels" and showing sprouting of many smaller branches. Even the smallest vessels show endothelium proliferation. Nowhere is true infiltration of round cells recognizable. That was the reason why Jakob [3] used the term "arteriosis."

It is not the purpose of this paper to discuss whether this type of vascular disease ever occurs without alterations in the meninges and in the brain tissue and whether such an isolated arteriosis would be of any pathognomic significance. In the presented case, the pathology of the vascular system was accompanied by alterations of the brain parenchyma and glia and it is again the combination of that pathology which I consider as evidence of a syphilitic disease.

Discussion.

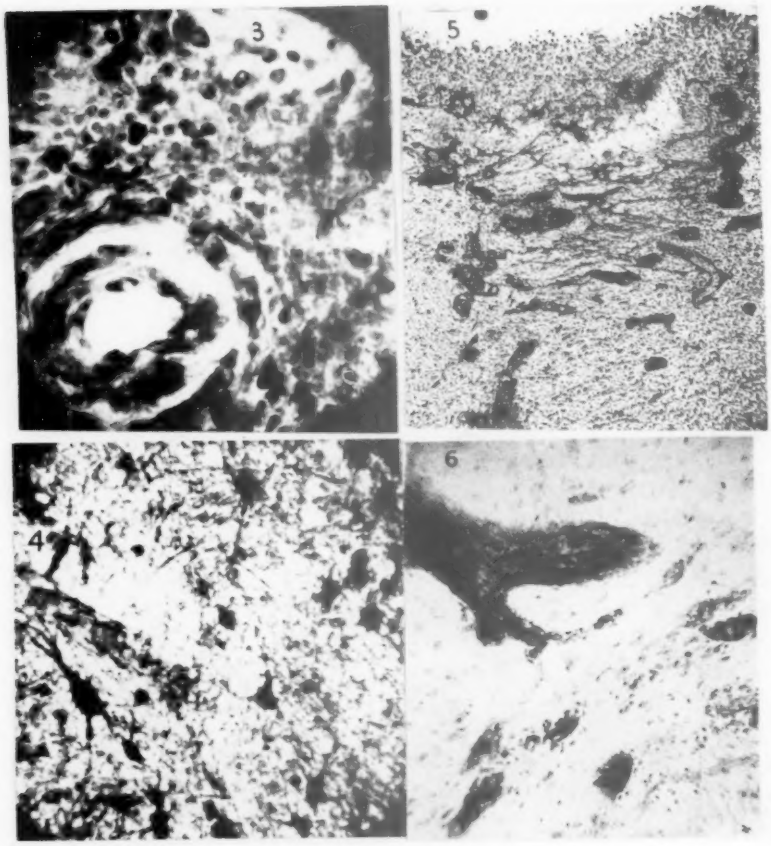
It has already been briefly discussed why there seems to be sufficient evidence in the first case to establish the diagnosis of congenital syphilis. The acute lymphatic meningitis upon the brain stem would certainly not suffice to draw conclusions concerning the etiology and a tuberculous meningitis may produce a picture very similar to that which was observed in the case presented. The chronic alterations, however, which were also present seem to render sufficient evidence for the specificity of the pathology. As far as the more acute changes are concerned there are some points of differential interest. It is surprising to observe, time and again, how little developed the vascular reactions are in tuberculous meningitis. Two cases which recently came to the attention of the writer gave ample evidence in that direction. Although several miliary tuberculoma could be located and there was a dense lymphatic pachymeningitis, no endarteritis could be observed and reactions of the connective tissue were almost absent. The round cell infiltration was almost entirely cellular while in a syphilitic process,



CASE I.

FIG. 1.—Lymphatic periarteritis and phlebitis upon the brain stem. Endarteritis of the smaller and of one larger artery. Notice the felt-like gliosis of the brain tissue. (H. and E. stain.)

FIG. 2.—Section through spinal cord. Patchy infiltration of dura due to round cell infiltration. Dense pachymeningitis. Round cell infiltration of peripheric nerves. The capillaries of the cord are infiltrated, the nerve cells still rather well preserved. (Nissl stain.)



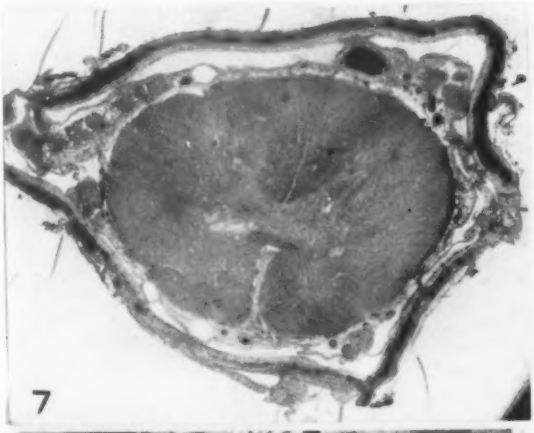
CASE I.

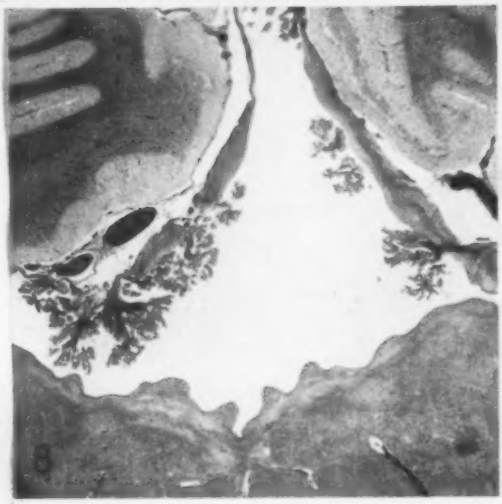
FIG. 3.—High power picture of small artery upon brain stem. Periarteritis consists of lymphocytes and many plasma cells, one giant cell at the right upper corner of picture. Endothelium swollen, miliary gummata between intima and media. (Nissl stain.)

FIG. 4.—Dense gliosis of astrocytes in deeper layers of cortex. (Cajal gold sublimate method.)

FIG. 5.—Section through floor of fourth ventricle. Notice profuse ependymitis. Capillar sclerosis with increase of silver fibers. Round cell cuffing. (Achucarro-Klarfeld tannin silver method.)

FIG. 6.—Same section as Fig. 5. High power (Nissl stain). Cuffing of vessels with lymphocytes and plasma cells. Miliary gummi in one artery.





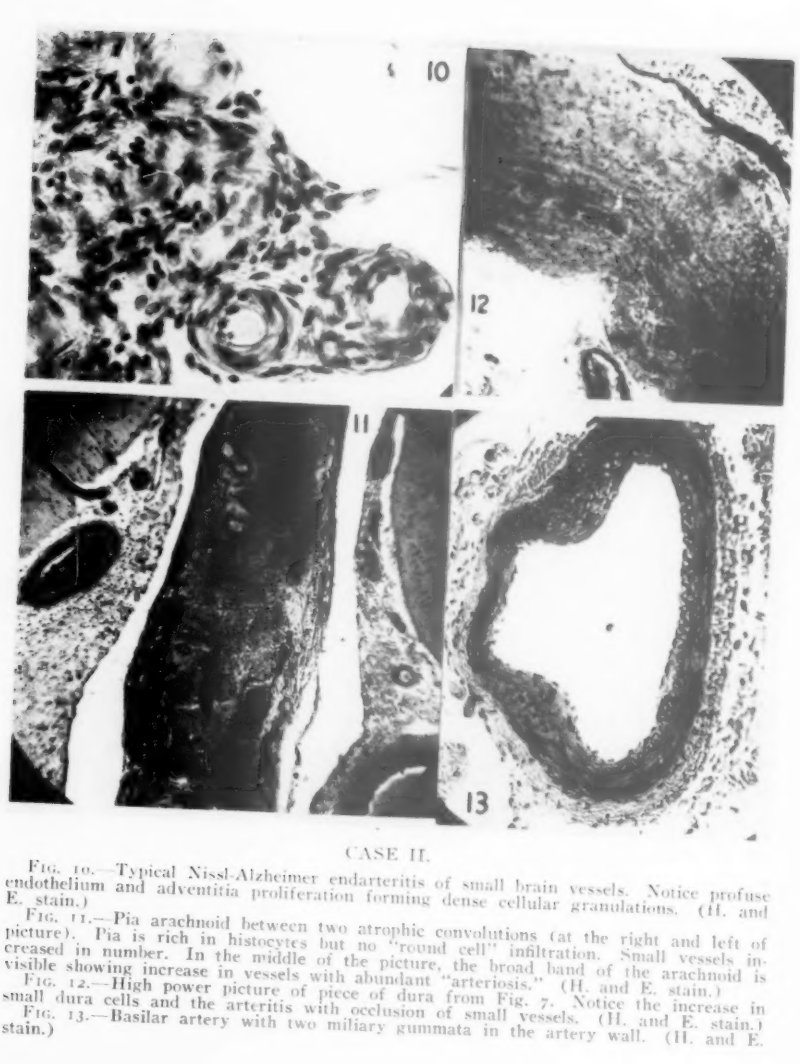


CASE II.

FIG. 7.—Section through spinal cord. Patchy endarteritis of small dura vessels. Arteritis within pachymeninges. White matter edematous. Enlargement of perivascular spaces. Softening of gray matter. (H. and E. stain.)

FIG. 8.—Floor of the fourth ventricle covered with papilliform ependymitis granulosa. Choroid plexus degenerated. Atrophic cerebellum. (H. and E. stain.)

FIG. 9.—High power picture of ependymitis granulosa upon the floor of fourth ventricle. (H. and E. stain.)



CASE II.

FIG. 10.—Typical Nissl-Alzheimer endarteritis of small brain vessels. Notice profuse endothelium and adventitia proliferation forming dense cellular granulations. (H. and E. stain.)

FIG. 11.—Pia arachnoid between two atrophic convolutions (at the right and left of picture). Pia is rich in histocytes but no "round cell" infiltration. Small vessels increased in number. In the middle of the picture, the broad band of the arachnoid is visible showing increase in vessels with abundant "arteriosis." (H. and E. stain.)

FIG. 12.—High power picture of piece of dura from Fig. 7. Notice the increase in small dura cells and the arteritis with occlusion of small vessels. (H. and E. stain.)

FIG. 13.—Basilar artery with two miliary gummata in the artery wall. (H. and E. stain.)

marked reactions from the side of the connective tissue are to be expected. On the other hand, the intima proliferation of syphilis is of a cellular nature while even in long lasting tuberculous processes, endarteritis is almost absent and the vascular changes consist in multiplication of the elastica interna. This consideration is of some interest because it may help in establishing the diagnosis but, of course, one needs more evidence to reach a final conclusion. In the first case presented, the gummatous granulations in the vascular walls, the Heubner's endarteritis, and the ependymitis granulosa indicated so definitely the diagnosis that there is no need to discuss every single symptom in detail. There is, however, a strong inclination among pathologists to deny the "specificity" of any pathologic lesion. Every argument of that kind ought to be taken seriously.

In 1932, in an instructive paper, Frederic Wertham [4] discussed the "nonspecificity" of the histologic lesions of dementia paralytica. He pointed out that the three cardinal histologic signs: infiltration of small vessels with plasma cells, proliferation of Hortega cells with formation of rod cells, and iron deposits in intra-adventitial spaces and in the Hortega cells, usually considered as specific of general paresis, may be found in nonsyphilitic infections. In experiments with spirocheta gallinarum, he discovered that in his control cases, some chickens showed a spontaneous disease that produced similar changes to that seen in the syphilitic experimental animals. He drew the conclusion that the histologic signs, mentioned above, are not sufficient to establish a definite diagnosis. At the 93d annual meeting of The American Psychiatric Association in 1937, Ferraro, Berrara and Gregory [5] presented a well studied case of juvenile paresis. They discussed at length the differential diagnosis and the probability of syphilis. Their final decision was that their case did not represent a syphilitic paresis, because some of the main signs, especially meningitic reactions, were completely absent. I think one ought to agree with these authors that there was not sufficient evidence in their case, to establish a final diagnosis in regard to the cause of the general paresis. In the first case, however, which I presented, there were so many more symptoms present which are generally considered as pathognomic that the diagnosis of syphilitic origin of those alterations seems more justified. There remains, however, the objection that

the negative sero reactions do not support such an interpretation. The child was admitted two weeks after premature birth to a Children's Hospital where the Wassermann reaction was negative. At that time, however, severe neurologic symptoms and epileptic seizures were present.

In a recent paper discussing the value of different serologic tests, Walter F. Lever and William K. Massie [6] quote several statistical investigations which showed that the Wassermann test was positive only in 41-49 per cent of cases of unselected syphilis. In that material, syphilis of the nervous system was not separately considered and it seems that very few cases of that type were included at all. As far as the literature on syphilis of the nervous system is concerned, it is always observed that the Wassermann reaction is frequently found to be negative. At the beginning of my paper, I mentioned the investigation of Raeder who demonstrated impressively that in syphilitic families the first children may show positive sero-reactions while the later ones show negative blood and spinal fluid. The outcome of the reaction is in no relation to the clinical manifestations and children with positive Wassermann may show less clinical symptoms than congenital syphilitic children with negative reactions. William Meninger [7] in his book on juvenile paresis gives a scrutinizing survey about the outcome of sero-reactions. He quotes a recent study, in which he and Bromberg reported "on the negative blood findings in 500 cases of neurosyphilis with positive spinal fluid Wassermann tests. Approximately 30 per cent of that group had negative blood Wassermann tests, and of 38 cases of general paresis included, eight had negative blood Wassermann tests (only one of these had been untreated)."

Concerning the special type of syphilitic manifestations which were present in our case, the percentage of sero-negative reactions is higher than in any other type. Grinker [8] writes, "The blood and spinal fluid Wassermann in vascular syphilis may be negative" and he adds regarding Erb's spinal lues, "Spinal fluid studies show that there is no blocking of the sub-arachnoid space. Cerebral fluid contains only a few more cells than normally and a trace of globulin may be noted. Blood and spinal fluid usually show negative Wassermann reaction." The same experience is expressed by A. Jakob: [9] "In the syphilitic chronic vascular disease, the serologic findings in blood and spinal fluid may be entirely negative." Kafca,

according to Jakob, expressed his opinion: "The syphilis of the vascular system of the brain is characterized by extremely weak sero-reactions, the spinal fluid shows either slight increase in cells or is entirely negative, the Wassermann reaction is positive only with 1.0, in several cases however, entirely negative. The hemolysin reaction is negative; the colloid reactions show a curve indicative of lues cerebri or they are almost negative.

This brief discussion indicates that negative sero-reactions do not rule out congenital syphilis. This is especially true if the sero-reactions are not frequently repeated and different tests are not used.

An even more complicated subject is the demonstration of spirochetes. I did not succeed in finding spirochetes in the brain tissue. It would certainly be of great help if one would eventually succeed in this search, but the attempt was unsuccessful in most of the cases reported in literature. Negative results are even to be expected and are in accord with observations of the most reliable investigators in this field. As early as 1906, Verse [10] called attention to the fact that spirochetes are most numerous in such organs which show slight or no reaction at all, but that spirochetes are not present in organs which appear much involved and reveal proliferative changes of connective tissue. These observations have been ascertained by several investigators (Carl Benda,[11] Schneider,[12] Hoffmann [13]). In those regions in which the proliferative, inflammatory reactions are visible, the parasites vanish. Some investigators (Carl Benda,[11] Hoffman,[13] and Warthin,[14]) have fostered the theory that the inflammatory reactions are the response of the tissue to the destruction of spirochetes. Whether the spirochetes break down because of the inflammatory reaction of the tissue, or the reaction of the tissue is the response to toxin production of dying spirochetes, is still a matter of discussion. Whatever the final decision will be, most writers mention countless unsuccessful attempts of demonstrating spirochetes in late stages of congenital syphilis or in chronic acquired syphilis. Grinker goes so far as to state, "This type of vascular syphilis is not associated with the presence of spirochetes and seems to be an endarteritis stimulated by toxins elaborated by the spirochetes lodged in other organs. Its effect on the brain is secondary to diminution and occlusion

of the vascular lumens producing perivascular atrophies and softenings."

The second of the cases reported, represents a very definite pathologic entity known as "endarteritis syphilitica of the small brain vessels." This disease was first described under the name of "diffuse brain lues" by Nissl [15] in 1903. In 1904 and 1909, Alzheimer [16] added nine further cases to the first description. In 1920, Jakob [8] published 13 observations, and Malamud [17] described four new cases in 1926. The outstanding feature of this disease is a proliferation and swelling of the endothelium and of the adventitia cells which stand out through proliferation and sprouting. The picture as a whole is very characteristic. The pia vessels form dense conglomerations which contain a large number of small and medium sized vessels. The smallest vessels are mostly affected. The swelling of the endothelium is so pronounced that the lumen of many vessels is closed. Pia and arachnoid are greatly thickened but no round cell infiltration is present. The alterations of the parenchyma are diffuse, the picture being very similar to those seen in general paresis. The nerve cells show swelling or sclerosis, but most of them have disappeared. The normal layers of nerve cells are no longer to be made out, the remaining cells lie confused and sparsely scattered. The glia shows increase in macro-glia and protoplasmatic swelling. Within the deeper cortical layers, areas of softening and honeycomb formation are noticeable. The center of both lentiform nuclei forms a network of fibrotic capillaries and glia cells while the nervous parenchyma is entirely degenerated. The picture is that of a "status spongiosus." These alterations involve mostly the gray matter of the cortex; the white matter being far less affected. The pathology of the vascular system and parenchyma is associated with marked thickening of pia arachnoid. The thickening of the sheaths is found all over the nervous system including the spinal cord. Here, degeneration of the gray matter is even more marked and small hemorrhages are not rare.

Clinical progress of our case was extremely slow and this is in complete accord with observations reported in literature. This child was the only one in our material who reached the age of 17 years, although her condition was desperate for almost five years.

It may be of interest to mention that Jakob [8, 9] in his description of this disease emphasized that spirochetes were not found

in any case, hitherto observed. Sioli and Westphal [18] reported spirochetes in cortex and vascular walls in one case which they considered as instance of the Nissl-Alzheimer disease, but it was a case of acquired syphilis with rather quick progress and more of a type of general paresis.

According to the literature, the sero-reactions vary at different times, the Wassermann test, however, is in almost 50 per cent of cases negative, at least, sometimes. In the 13 cases recorded by Jakob,[3] the blood Wassermann was negative four times, questionable two times and positive six times and one time weakly positive. The spinal fluid is recorded for ten cases; in seven cases the Wassermann test was positive with cells 20/3 varying to 400/3, in three cases it was entirely negative.

The differential diagnosis has to consider chronic poisonings in the first place, lead, arsenic and manganese, but even chronic alcoholism and malaria-tropica may produce similar arterial proliferations. The intima and adventitia proliferation is therefore not pathognomic in itself. In poisonings, however, the arteriosis will hardly amount to such a degree and will not be associated with other signs of active connective tissue reactions. Because of the fact that the vascular pathology may be seen in non-syphilitic conditions Pentchew [19] expressed some doubt whether the Nissl-Alzheimer endarteritis is a syphilitic disease after all. But, he had no experience of his own in that disease and he disregarded the fact that the vascular changes will not alone establish the diagnosis.

Conclusions.

If we consider sero-positive reactions in the feebleminded population as indication of congenital syphilis, the percentage of congenital syphilis is very low—less than 2 per cent. This estimate is, however, based on usually one test and it is known that this percentage is much too low. It is generally agreed that the sero-reactions in congenital syphilis vary to a great extent and tests must be repeated several times and qualitative tests ought to be used. If we consider those cases which are suggestive of syphilis, the percentage is much higher and would amount to almost 15 per cent of the patients. Various investigators have found various per-

centages. Most agree at the present time that a fair estimate would be between 7 per cent and 10 per cent.

Investigations under way at the Wrentham State School approach the problem from another angle. In microscopic studies of autopsy material, several alterations were found which strongly indicated congenital syphilis. The number of cases is still too small to dwell on any speculation about the percentage of these syphilitic cases in relation to the whole population.

The microscopic examination of two cases, studied on the basis of several hundred slides each, has been presented. The first case was an instance of vascular meningoencephalitis, associated with Heubner's arteritis and peri- and endarteritis gummosa. The second case was a case of the so-called Nissl-Alzheimer endarteritis-syphilitica of the small brain vessels. The sero-reactions in congenital syphilis were discussed in detail.

These cases have been presented to demonstrate the difficulties which are still outstanding in establishing the diagnosis of congenital syphilis. The discussion may stimulate clinical and pathologic studies of this type of children who represent rather a considerable percentage of the population in schools for the feebleminded and may eventually lead to a definite understanding of the rôle played by congenital syphilis in the production of mental deficiency.

BIBLIOGRAPHY.

1. Penrose, Lionel: Mental Defect, Farrar & Rinehart, 1934.
2. Raeder, Oscar J.: Amer. J. Dis. Child., 21: 240, 1921.
3. Jakob, A.: Ztsch. ges. Neur., 54, 1920.
4. Wertham, Frederick: Arch. Neur., 28: 1117, 1932.
5. Ferraro, Barrera and Gregory: Am. J. Psychiat., 94: 1291, 1938.
6. Lever, W. I. and Massie, W. K.: Arch. Derm. and Syph., 40: 45, 1939.
7. Menninger, William C.: Juvenile Paresis. Williams & Wilkins Co., 1936.
8. Grinker, Roy R.: Neurology. Chas. Thomas, 2d. ed., 1937.
9. Jakob, A.: Normale und Pathologische Anatomie und Histologie des Grosshirns, 2d. Vol., Leipzig und Wien, 1929.
10. Versé, Max: Münch. Med. Wo., 29: 1906.
11. Benda, Carl: Allgemeine Pathologie, patholische Anatomie und Serologie der Syphilis; Bd. 15. 2 Teil Berlin, Jul. Springer, 1929.
12. Schneider, Paul: Lubarsch-Ostertag Erg. Path. 20, II. I, 1923.
13. Hoffmann, E.: J. Pediat., 9: 1936.

14. Warthin, Aldred Scott: Harvey Lectures, Series XIII and XIV., 1917-1919, J. B. Lippincott Co.
15. Nissl, K.: Neur. Ztbl. 1904 and Histopath. Arb., Jena, 1904.
16. Alzheimer: Histopath. Arb. 1904 and Zentralbl. Nervenheilk, 20: 676, 1909.
17. Malamud: Ztsch. ges. Neur. 102, 1926.
18. Sioli and Westphal: Arch. Psych., 63: 1922, 66: 1922.
19. Pentschew: Nervenarzt, 8: 393, 1935.

DISCUSSION.

DR. ADOLF MEYER (Baltimore, Md.).—Here we are confronted with a condition in which, undoubtedly, we have to use inference as well as study of the detail of the lesion. We have to recognize that it is not possible to demonstrate the spirochaete in a very large percentage of lesions that we have every reason to attribute, however, to syphilitic conditions. Dr. Potter, in New York, has undertaken studies to demonstrate the frequency of syphilis among patients in institutions for feebleminded. I do not know whether these studies were particularly pursued also in the postmortem material, but where one finds such a combination of data as Dr. Benda has described, one really has no choice, practically, but to await the impression given by the total picture. It makes it very much more desirable than most people realize that in our institutions for the feebleminded we pursue postmortem studies and establish what lesion types one gets, and it may be that with particularly advanced stages of this condition it will be possible to give the positive and final and incontrovertible evidence that one actually does deal with what Dr. Benda infers.

DR. SAMUEL EPSTEIN (Boston, Mass.).—This material which Dr. Benda has presented affords to my mind a very nice demonstration of the existence of syphilis among the feebleminded; in fact, it is perhaps the only evidence of which I am aware of a conclusive nature that syphilis plays a rôle in the mental deficiencies. It has always been a problem to determine the presence of syphilis in feebleminded patients. Even the introduction of the Wassermann reaction has not helped us very much; in fact, some workers in the field of mental deficiency and many neurosyphilologists consider that negative serologic reactions exclude the presence of syphilis. The evidence presented by Dr. Benda today proves conclusively that that is not so.

The illustrations of the histologic changes shown here are, in my opinion, quite conclusive, and Dr. Benda is to be congratulated upon his excellent demonstrations.

This work has another great value I think, namely, that it brings up an important line of investigation. I refer to the matter of brain punctures. It would be perfectly feasible, I think, to do brain punctures in the living and to examine such material histologically and; secondly, in such cases one may give antiluetic treatment and, thirdly, brain punctures might again be done to see what changes have occurred. This would afford not only some

conclusive evidence of the value of antiluetic treatment in such cases of mental deficiency, but it would also throw considerable light upon the action of our antiluetic remedies in various forms of neurosyphilis. I think that the closer we get to such factual considerations, the sooner we are going to learn about at least one aspect of the problem of mental deficiency.

Dr. Oscar J. Raeder (Boston Mass.).—I am very glad that Dr. Benda has done this nice piece of work. While he has not demonstrated spirochaetes which probably would doubly clinch the diagnosis it seems to me that the evidence is so strong that we must conclude that these lesions are syphilitic.

I have been attacked for a great many years for saying that a great deal of the feeblemindedness is syphilitic in spite of the fact that the serum reactions are negative. The paper that I wrote, and that Dr. Benda mentioned, was an effort to prove that there were still lesions, demonstrable by both the psychological reactions and also physical reactions, in spite of the fact that the third or fourth birth, after a number of syphilitic abortions, lived and had serum negative reactions.

Let me say first that I do not know whether the Hinton test has been tried out and compared with the Wassermann test. The Hinton test is supposed to be more sensitive in many cases, but I do not know whether a comparison has been made in these syphilitic children, or presumably syphilitic children, to see whether the Hinton test would give us some information. I think that should be done if it has not been done.

Another point and that is the matter of treatment and prophylaxis. I think it is brought out more prominently than anything else, that we must not wait for treatment until these children get to be of school age where the ordinary child is usually discovered to be feebleminded. Ordinarily parents do not recognize feeblemindedness until the child is in kindergarten or school or is old enough to talk and think and is in some way comparable with his playmates and contemporaries; therefore, I should think that the challenge would confront the general practitioner and obstetrician and especially the family physician to institute some sort of antiluetic treatment in these children early in life, even right after birth. We know that there is still pretty active mental growth taking place in the first year after birth and perhaps that is just the time when such treatment would do good.

Dr. Clemens E. Benda (Wrentham, Mass.).—I am sorry that the time is not sufficient to discuss the extremely interesting question of the relationship between spirochaete and syphilis-infection and between syphilis and serum negative test. Some details will be given in the published paper.

From a clinical standpoint the study of these cases was not extremely careful. As I told you, we did not expect syphilis at all. We made the routine Wassermann and the Wassermann was negative one time and then we were satisfied and thought of other problems. So I do not know whether the Hinton test, especially that which is now developed for the spinal fluid would give us more information.

Of extreme interest is a suggestion which Dr. Epstein gave in regard to puncture of the brain, especially of the meninges. It seems that this method, which Dr. Solomon also suggested in a personal conversation with me, is used in the Boston Psychopathic Hospital with great success. If this method would provide us with the possibility of an early diagnosis in this type of child, we might be able to treat the patients with iodine or bismuth with some success. I am, however, not in a position to answer questions about the best treatment of late stages of congenital syphilis, because we have not yet experience with a treatment which would enable me to give some definite suggestions.

ACUTE GENERAL PARESIS.*

By M. HERMAN, M. D., and M. P. ROSENBLUM, M. D.,
New York City.

General paresis is commonly considered a chronic disease that begins insidiously and progresses rather slowly. That an acute onset with a rapidly fatal termination may occur has not been well recognized. Prompt diagnosis in these acute forms is important because of the serious prognosis without the early institution of treatment.

The acute onset manifests itself in several clinical pictures:

A. Fulminating type.
B. Convulsive type.
C. Catatonic type.
D. Acute confusional type.

A. The Fulminating Type.

In this type of case we find a sudden onset and rapid development of symptoms so that it has been frequently in the literature referred to as galloping paresis. The clinical picture usually resembles that of an acute delirium in which there is marked agitation, excitement, and motor restlessness. Hallucinations are prominent and may be intimately associated with grandiose delusions. Physically, the patient shows the neurologic signs of general paresis, but, in addition, he frequently has a febrile course. The symptoms increase in severity, the patients refuse food and become dehydrated. The course may be punctuated by epileptiform and apoplectiform attacks. Death may supervene in one to three weeks. Leverty [1] reports a case with symptoms of a delirious mania that had a fatal termination in four months. The pathology in these cases reveals the typical meningo-encephalitic reaction, but the

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cellular changes are more intense and show a necrotic character.[2] In the fulminating type it would seem that the extent of the lesion and the rate at which it develops are so great that the whole course of the disease is run in a very short space of time.[3] It has been stated[4] that approximately 1 per cent of all cases of general paresis have this type of onset. In an analysis[5] of 335 paretics admitted to the Marburg Clinic there were four cases of the fulminating type.

CASE REPORTS.

C. H., a white male, age 49 years, was admitted to the hospital on July 2, 1937. The patient gave a history of having had a chancre 20 years ago, and some anti-luetic therapy. He has also been a chronic alcoholic. On admission, the patient showed a picture of severe delirium. He was reacting to hallucinations and was completely confused and disoriented. He appeared acutely ill, and his temperature was 101°. The pupils were irregular, unequal, and fixed to light. His speech was dysarthric. The knee-jerks and ankle-jerks were absent. He also had a bi-lateral grasp reflex and sucking reflex. The blood Wassermann was negative. The spinal fluid Wassermann was 4 plus with a colloidal gold curve of 3222100000. The patient became sicker and evidenced difficulty in breathing. The physical signs in the lungs revealed moist rales at the right base. Death occurred two days after admission.

Post-mortem examination showed a secondary patchy atelectasis of the lungs. The coronary arteries showed moderate sclerosis. There was an aortic insufficiency and an aneurism of the ascending aorta. The liver showed portal cirrhosis and the kidneys a moderate arteriosclerosis. The dura was of normal thickness. The basal arteries were thickened and had several placques. The brain weighed 1400 grams and showed a great deal of fluid. Pia-arachnoid was diffusely thickened, especially at the vertex. There was pronounced convolutional atrophy most marked in left frontal lobe and tip of right frontal lobe. The optic nerves were small in diameter. There was slight internal hydrocephalus. There was a softening of the Isle of Reil on the right side which presented a cystic formation with a pale, grey lining. Microscopic examination of the brain showed a fibrous arachnoiditis and a lymphocytic infiltration of the leptomeninges. The arteries were sclerosed. The neurones showed a general decrease and the smaller cells were pyknotic. The spinal cord shows marked meningeal infiltration, mostly lymphocytes and occasional plasma cell. Small blood vessels show proliferation with thickened walls which show degenerated changes. The optic nerves showed infiltration and myelin loss.

The delirium in this case is complicated by alcoholism. But in the literature it has been pointed out by Kraepelin that the delirium in these cases of general paresis resembles very closely the delirium

tremens of the alcoholic. However, to show that a delirium can occur in which there is no question of alcoholism as an etiologic factor, we can quote the following case:[6]

A man aged 35 was admitted to the Royal Naval Hospital, Bermuda, on June 29, 1932, as a case of cerebral syphilis. He gave a history that for about a week prior to admission he had been feeling nervous and shaky; his messmates and others in the ship stated that he had been acting strangely for several weeks. There was a history of syphilis dating from 1922. A Kahn test was positive in June, 1932.

On admission he complained of shakiness and unsteadiness and said that he felt a pricking sensation in the soles of his feet. Temperature was 103.4° F. and pulse rate 90. The patient seemed to be rational, but cerebration was slow and speech halting. The pupils were irregular, the right being longer than the left. Both were sluggish to light but accommodated fairly well. A very coarse tremor of tongue and limbs was present. There was some spasticity in the limbs. Knee-jerks were increased. No rigidity of the neck was present and Kernig's sign was absent. No sensory abnormality was noted. Nothing abnormal was found in the heart or lungs; on the skin there was a dark red papular rash, chiefly centrifugal in distribution.

On June 30 the patient had acute delirium. He was very restless and difficult to manage, repeatedly trying to get out of bed, and refusing food and medicine. He had retention of urine. Lumbar puncture was done and 30 c.c. of cerebrospinal fluid was withdrawn; it was clear and definitely under pressure. The findings in the fluid were: cells 297 per cm.; lymphocytes 60 per cent; globulin increased.

For a few days the patient's condition improved. He became quieter, being only stupid and fatuous instead of delirious and emotional. The skin condition cleared up. This improvement lasted until July 10, when he suddenly became restless. He had periods of delirium and his speech degenerated into a mere mumbling of syllables. Coma, with fecal and urinary incontinence was present on July 13. The patient died early on the 15th—that is on the 17th day after admission to the hospital.

Post-mortem examination of brain only. Dura mater was not adherent to the skull. There were thickening of the dura, arachnoid, and pia, and definite adherence of the pia to the surface of the brain. The surface of the brain showed some flattening of the convolutions and some engorgement of the blood vessels. The pathological report on sections of the brain was: A meningo-encephalitis is present. Meninges are infiltrated with lymphocytes and plasma cells, and there is an infiltration of less degree in the sheath of the vessels in the brain substance. The microscopic appearance is consistent with the diagnosis of acute general paralysis.

B. Convulsive Type.

It is well known[7] that convulsive attacks frequently appear during the course of the usual type of general paresis. Occasionally,

however, they may appear early and may even be the first indication of the disease. In the acute onset with convulsions the usual history given is that the patient had been apparently in good health up to a few weeks of or to the moment of the convulsions. The course may then follow different directions. In some the convulsions increase in severity, the patient becomes increasingly clouded mentally, progresses into a stuporous vegetative state and dies within a short time. In others, following the initial convulsions the patient progresses into the chronic type of the disorder. Sometimes paresis of limbs or of the face may follow a convulsive seizure, but it is characteristic[8] in general paresis that these clear up rapidly in 24 hours to a week without leaving any residuals.

CASE REPORT.

L. B., negress, age 43, was admitted to the hospital on December 26, 1935. There was a history of loss of memory and general weakness for the past two months. She had been attending the H——— Hospital and was receiving anti-luetic therapy. Examination revealed a fairly well-developed middle-aged negress who showed emotional instability and was generally euphoric, exuberant, and boisterous. She was confused and disoriented. The depth of confusion varied. At times she reacted to auditory hallucinations and revealed paranoid persecutory trends against her husband. Physical examination revealed that the pupils were equal and reacted slightly to light and accommodation. The extra-ocular movements were normal in function. Speech showed slurring of test phrases. The heart was not enlarged; the P. M. I. was 9.5 cm. lateral to the mid-sternal line in the fifth interspace. The sounds were regular and of good quality—$A_2 > P_2$. The B. P. was $\frac{184}{124}$. No murmurs were heard. Lungs were clear and resonant throughout. The reflexes were active and equal, no Babinskis or ataxia were present. Serology revealed a blood Wassermann of 4 plus, spinal fluid Wassermann 4 plus, colloidal gold curve was 5555554100, globulin test was negative and there were 36 lymphocytes present. On January 7, the patient suffered from convulsive seizures consisting of spastic and clonic contractions beginning in the left side of the face and followed by flaccidity of the left side of the body. There was also an equivocal right Babinski with spasticity of the right side associated with hyper-reflexia. The next day the patient was acutely ill; there were twitchings of the left side of the face and flaccid hemiparesis of the left side of the body. This was accompanied by a semi-stuporous state during which the patient muttered incoherently and had visual hallucinations. The following day the left-sided weakness had become more marked and the stupor had deepened. She ran a low grade temperature. The convulsive seizures persisted for the next few days with twitchings of the left side of the face and clonic movements of the left side of the body. There were also tonic spasms of the right upper

and lower limbs. The temperature became elevated to 105° and the patient made her exitus on January 13, 1936.

Post-mortem examination revealed enormous thickening of pia-arachnoid over both hemispheres in upper frontal and parietal regions with cortical atrophy. There was very little sclerosis in circle of Willis. Optic nerves were normal in size. There were large, bony placques in falx cerebri. The granular ependymitis was not marked.

Microscopically, the meningeal changes consisted of a fibroblastic reaction as well as the presence of numerous lymphocytes and plasma cells. The cortex showed proliferation of capillaries, distortion of cortical layers, with dropping out of nerve cells and numerous plasma cells in perivascular regions. The neuroglia stains revealed an increase in the number of astrocytes. The diagnosis was syphilitic-meningo-encephalitis.

Examination of the lungs revealed a purulent bronchiolitis with pulmonary infarct.

C. Catatonic Type.

Symptoms of a catatonic type may appear during the course of general paresis [9] or may even be the initial manifestation of the disease. In this condition the patient becomes mute, is oblivious to his surroundings, refuses food, and is negativistic. Frequently, there is also catalepsy and jaw and neck negativism of a type bearing a marked resemblance to the phenomena commonly observed in schizophrenia. When the disease is initiated with such catatonic symptoms a diagnosis may be often extremely difficult before the serology is shown to be positive for syphilis. Even the pupillary changes may not be helpful because fixed pupils may be found under certain conditions in catatonic schizophrenic patients, especially in negroes.[10] The patient with general paresis has a marked mental confusion and frequently extensive aphasia and apraxia. In these catatonic types these symptoms are difficult to demonstrate because of the mutism and negativism. However, it frequently happens that the catatonic phenomena wax and wane, so that the intellectual status becomes more apparent in the relatively free period. The disorientation, memory defects, and the organic type of mental deterioration can be elicited and differentiate the condition from schizophrenia.

When a patient shows catatonic symptoms of general paresis, it may herald an extremely malignant reaction. Death may occur in several days or weeks. In others the symptoms subside and the patient assumes the chronic type of general paresis characterized by dementia.

CASE REPORT.

R. S., a negro male, age 34, was admitted to the hospital on May 4, 1938. The history as given by his wife was that the patient had been losing interest in things for several weeks and then four days before admission had an episode in which he shook violently, became unconscious and remained in a mute state. On admission, he appeared to be in a catatonic state. He was mute and stuporous, resisted movement of his extremities, and refused food and drink, so that it was necessary to tube-feed him. Physical examination showed no signs referrable to heart or lungs. The pupils were regular, equal, and reacted well to light and accommodation. The only positive findings were a bilateral grasp reflex and changing rigidities of the extremities. Within a few days the patient roused from his stupor but remained mute. His mutism suggested a severe motor-aphasia. He also had apraxia and he retained his negativism.

Spinal fluid studies showed a positive globulin reaction (Pandy). There were 8 lymphocytes per cm., total protein of 85 mgs., colloidal gold curve of 5554432210, and a Wassermann which was 4 plus. The blood Wassermann was 4 plus.

On the morning of May 15, the patient suddenly went into stupor again and died within a few minutes.

Post-mortem findings showed that the pathology was confined to the central nervous system. There was a marked milky thickening of the pia-arachnoid over both hemispheres, especially in the frontal and upper parietal regions. There was moderate congestion of the veins over both hemispheres and gross atrophy of the frontal lobe.

Microscopic Report.—The pia-arachnoid showed a pronounced fibroblastic reaction as well as the presence of numerous lymphocytes and plasma cells. The mononuclear infiltration was particularly marked deep down in the sulci, and it appeared also as a perivascular cuffing of blood vessels down in the cerebral tissue. The cortical laminations were considerably disturbed and there was an actual decrease in the number of neurones. Neuroglia stains showed a marked increase in the number of astrocytes as well as a vascular proliferation. The microglia stain showed the typical rod cells of paresis. With the Jahnel stain a few spirochaetes were found scattered through the lower layers of the frontal lobe sections.

D. Acute Confusional Type.

Of all types of acute onset in general paresis the most common is the acute confusional type. In this form of the disease the patient suffers from a rapid mental change with deep clouding of consciousness, and profound disorientation. He walks about in a dazed, confused fashion. As a matter of fact, this type of patient is frequently admitted to a hospital after being found wandering around the streets unable to give any pertinent information. This

confused state may be accompanied by convulsive seizures, but very often is found without such phenomena. Commonly, the acute confused state subsides, and the patient merges into the chronic phase of the disease. Sometimes a patient will have several episodes of acute mental confusion lasting several days or weeks, and, after a latent period of several months or a year, the chronic type of general paresis develops.

CASE REPORT.

J. P., a white male, age 52, was admitted to the hospital on May 5, 1938. He had been found wandering around in the street and no contact could be made with him. He did not talk and appeared to be in a daze. On admission he was confused and semi-stuporous. He fumbled with his clothes, picked at the bed clothes, and was untidy in his habits. He did not recognize his surroundings. He uttered his name but nothing else. He was lethargic, and not at all resistant.

Physical examination showed evidence of luetic heart disease with an aortic insufficiency. His pupils were regular, equal, but reacted sluggishly to light and accommodation. The knee-jerks were equal and active. No pathologic reflexes were elicited. Speech later was shown to be dysarthric.

Spinal fluid had a total protein of 90 mgs., a 4 plus Pandy reaction, a 4 plus Wassermann, and a colloidal gold curve of 5554433310. The blood Wassermann was 4 plus.

A history was obtained from his employer who stated that he had worked as a handy man for her for several years. He was an occasional alcoholic but had not shown any symptoms referrable to a mental disorder prior to this episode of confusion. He never had convulsions. The patient gradually recovered from the acute episode but showed psychiatric evidence of a demented type of general paresis.

On May 16, 1938, he was committed to a state hospital.

Summary and Comment.

The acute onset of general paresis is a dramatic and often catastrophic event in the life of an individual. The initial symptoms have been described as being either a delirium, convulsive seizures, catatonic phenomena, or an acute confusional state. In general they show various grades of stupor and acute clouding of the consciousness. The acute onset carries with it a serious prognosis. The type which begins with a delirium usually runs a fulminating course. The three other types may also run a short and rapidly fatal course, but may progress into the chronic form of general paresis. The catatonic type of case, as has been pointed

out, may be difficult to distinguish from schizophrenia but the serology and the marked organic intellectual disturbances differentiate the case of general paresis.

The pathology in these acute cases shows an extensive and active meningo-encephalitis. The vessels in the meninges, as well as in the cortex, have a pronounced mononuclear perivascular cuffing, and this reaction extends deeply into the cortical tissue. In general, the reaction is more intense than in the usual type of case.[11] In the usual chronic cases, the pathology is more marked in the frontal, parietal and temporal lobes in that order, and microspically, it is typical to find various grades of pathological alterations lying side by side with numerous cells that appear entirely normal. In the acute case one sees all cells of the cortex showing in various degrees indications of the disease process. These cells show Nissl's so-called grave alteration which probably results inevitably in the destruction of the cell. The cortical laminations show the characteristic distortion and a decrease in the number of neurones. Neuroglia stains reveal an increase in astrocytes, and vascular proliferation. Microglia stains show the typical rod cells. In some spirochaetes were demonstrated.

Merritt[12] and his co-workers have shown that the capillaries in brains affected with general paresis are obstructed by swollen endothelial cells and that the hypertrophied endothelium contains phagocytosed spirochaetes and blood pigment. We suggest that the acute symptoms occurring in general paresis may be due to the marked vascular reaction as shown by the intense perivascular infiltration.

The rapidity of the course in many of these acute cases is such that often death may supervene before adequate treatment can be instituted. In an acute case, an emergency can be said to exist. Immediate treatment is imperative. It is true that in a large number of cases the acute symptoms will subside without treatment, but this cannot be predicted in the individual case, for it is difficult to decide which case will proceed to a fatal termination. This problem is in some respects similar to that of acute appendicitis. It requires emergency care and treatment.

Some[13] have recommended immediate institution of fever therapy, malaria or other type. Others[14] have recommended for prompt action the use of a soluble preparation of mercury, such

as the bichloride, succinimide, or red iodide injected intramuscularly daily or every other day in doses varying from $\frac{1}{8}$ to $\frac{1}{2}$ grain.

Solomon [15] and his co-workers have indicated that the preliminary use of tryparsamide injections greatly enhances the probabilities of therapeutic success in the treatment of general paresis by fever, and that the improvement of spinal fluid findings occurs more rapidly following fever if the patient has first been prepared by treatment with tryparsamide. This method appears to be readily applicable to an acute case. Whatever method is used, prompt institution of treatment is indicated in order to avoid a fatal termination.

BIBLIOGRAPHY.

1. Leverty, A. S.: A case of fulminating general paresis with autopsy. Medical Record, Mar. 2, 1912.
2. Schmierer, P.: Acute general paralysis. These de Paris, 1932, No. 52, records 10 Epitome in Brit. Med. Jour., p. 113, June 18, 1932.
3. Nicol, W. D., and Hutton, E. L.: Some clinical aspects of general paralysis, J. Mental Science, 81: 804, Oct. 1935.
4. See 2.
5. Paton, S., and Rusk, G. Y.: Acute paresis with report of a case. The clinical history and pathological findings. Am. J. Psychiat., 59: 457, 1903.
6. Sorly, E. R.: Acute general paralysis. Lancet, 1: 961, May 6, 1933.
7. Solomon, H. C.: General paresis. Oxford Medicine, Vol. 6, chap. 21, p. 591.
8. See 7 and Grinker, R., in Tice: Practice of Med., Vol. 10, p. 103, 1932.
9. Kraepelin, E.: General paresis. Nervous and Mental Disease, Monograph Series No. 14, p. 15.
10. Schilder, P., and Parker, S.: Pupillary disturbances in schizophrenic negroes. Arch. Neurol. and Psychiat., 25: 838, April 1931.
11. See 9.
12. Merrit, H. H., Putnam, T. J., and Campbell, A. C. P.: Pathogenesis of the cortical atrophy observed in dementia paralytica. Arch. Neur. and Psych., 37: 75, Jan. 1937.
13. Schilder, P. Personal Communication.
14. Grinker, R., in Tice: Practice of Medicine, Vol. 10, p. 103, 1932.
15. Solomon, H. C., and Epstein, S. H.: General paresis. Treatment by tryparsamide-induced fever sequence. Am. J. Syph., Gon. and Ven. Dis., 20: 281, May 1936.

THE LONG-TERM PRISONER.*

A Study of Thirty Men Completing Life Sentences.

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On the assumption that a long standing pathogenic process produces pathological manifestations not detectible in the early states of the disease, we undertook the study of a group of long-term inmates in a penal institution to ascertain what effect prolonged incarceration has upon them both in respect to personality and social outlook. At least two factors prompted our investigation: first, that penal servitude is used more widely and for more extended sentences in the United States than in any other country; and second, the current tendency to increase the length of prison sentences. For the purpose of this study only those inmates were selected who had served a minimum of twenty consecutive years and were physically and mentally able to cooperate in the interview. The men are all serving their sentences in the Joliet-Stateville branch of the Illinois State Penitentiary. This would suggest that they are making a satisfactory institutional adjustment, since psychotics or those chronically maladjusted are generally transferred to the Psychiatric Division of the Illinois State Penitentiary, located at Menard.

In the Joliet-Statesville branch there are 49 men who have served a sentence of 20 or more consecutive years. This means that none has been out on parole or otherwise had opportunity during that time to live as an ordinary citizen. Out of the total of 49, 19 were omitted from the study for the following reasons: 14 were rated as mental defectives or borderline defectives, two were blind, two had insufficient knowledge of English, and one gave evidence of senile deterioration. Each of the selected men is making a satisfactory institutional adjustment: 13 out of the 30

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are working on the honor farm, which is considered a position of trust, is outside the prison walls, and has little supervision; three are employed in the hospital; three in a yard gang; three as cell-house workers and eight as odd-job workers, occupations which are considered the better type of institutional employment. Their ages upon admission ranged between 16 and 49 years, while their present ages are between 39 and 73 years. The shortest term served by any one man is 20 years, the longest is 31 years, and the offense in each case was murder. Nineteen were single, six married, three divorced, and two were widowed through their own crime.

The study was conducted by means of a questionary with which every inmate was individually interviewed. The questionary was used only as a guide, since the interview was informal and had the aspect of a friendly conversation. Each subject was called to the mental health office where the psychiatrist, after exchanging a few preliminary pleasantries with the inmate, informed him of the object of the interview and asked him if he would like to assist in a bit of research. He was informed that no benefit would accrue to him for the service. At this explanation some sighed good-humoredly with relief. The interview, which averaged about an hour and a half, was allowed to drift to irrelevant subjects, occasionally interruped with a cigarette and was in general so casual that the inmate did not often differentiate between a crucial and an indifferent question. He was thoroughly assured that he need not reply to questions if he did not wish to.

The study centered around the following five topics:

1. Extent of social severance.
2. Modification of interests.
3. Attitude toward punishment.
4. Interest in freedom.
5. Social attitudes in general.

Extent of Social Severance.

Extramural social or familial connections are of extreme importance to the inmate in that they modify if not actually condition his institutional behavior as well as determine his adjustment when paroled or discharged from the penitentiary. Our inquiry was limited to the number of letters and visits an inmate receives; the amount of help he expects to get towards readjustment when released, and his attitude toward friends and relations

in general, as well as his confidence in the future. At this point certain prison rules should be emphasized such as that letter writing and visits are discouraged or at least limited by the authorities, and that while an inmate may receive all incoming letters, subject to censorship, his outgoing mail is limited to two letters a month. Visitors are limited to not more than two persons for 50 minutes once in two weeks. Under these conditions the extent of extramural contacts an inmate is able to maintain is extremely circumscribed, and as a result there is a progressive diminution in visits and correspondence in every one of the cases. Some of the causes of diminishing contacts with the world are natural ones such as death or migration; but mainly, the inmates were unable to ascribe a specific reason other than the indifference that is fostered by time. Twenty-four of our inmates carry on some form of correspondence and have received at least one letter during the past year. Six have not had any mail for a year or longer. Of the 24, 13 get letters as often as once a month, seven about twice a year and four only once a year. Visits are less frequent than letters, although in individual cases there are those who are visited by friends or relations but do not get letters from them. Only 10 are visited as often as once a month, six see a relative or friend about once a year, and the remaining 14 are rarely visited. Among these 14 are six who no longer expect to have visits. All, with the exception of five, state that if they were released, they would get some help from friends or relations on the outside. Two can get a home, financial support and employment, so that they have no economic apprehension in facing the future. Eleven would receive temporary board and room, but could not expect financial assistance, 12 had no promise of assistance, but believe they would be helped by friends after release, and five would be entirely dependent upon the statutory ten dollars that the state allows a released inmate.

So far as attitudes towards friends and relatives are concerned, all, with the exception of two, expressed good will and praise for the help, generosity and consideration they received from them while serving their sentences. Two felt that their friends were indifferent and had not extended as much help as the inmate believed himself to be entitled to. Curiously, all were optimistic about facing the future, an attitude that would not seem to be supported by the facts or common to inmates in general.

Modification of Interests.

Under this heading the inquiry centered about changes in attitudes toward crime, religious beliefs and subjective mental changes. Eighteen stated that they were never criminally inclined, had always disliked a criminal and hence there had been no change in that respect. Eight gave the rather conventional response that imprisonment taught them greater respect for the law and the seriousness of crime. Four expressed a profound change that indicated the extent of their original criminal attitudes. One of this four, with a great deal of feeling, said that it was a blessing he had been arrested for otherwise he would have perpetrated a crime even graver than the one for which he was arrested. Another said that he seems to have been "crime bent" knowing all the time that he would be caught. Two stated that they had not thought of their criminal acts in terms of morals and that the penitentiary imbued them with that consideration. It was practically impossible to obtain any expression relative to the social significance of a criminal act. The answers were stereotyped, that is, that crime was wrong and their crime would not be repeated. Even those who claimed that their criminal act was unavoidable or committed in self-defense insisted it would not recur under similar circumstances. Responses here might have been influenced by a consideration of the possible effect they would have on the examiner, but we are inclined to feel that repression and defense had been so effective on this point that the subjects were unable to detect an apparent irrelevancy in their stand or to discuss the matter with objectivity.

Eliciting an opinion on religion was difficult, but it appears that religious beliefs were but little modified through imprisonment. Fourteen stated that they attended religious services and expressed a definite religious belief. Fifteen had not attended services for the last two years and appeared indifferent to the offerings of religion. One would not discuss anything pertaining to religion. A crucial question was, "Has religion helped you do your time?" Following are some typical answers.

A. *Those who expressed a religious belief.*

"Naturally a person's hope is built on religion."
"It gave me a better slant on life."

"I feel a whole lot better when I go to church."
"I will say that religion has been a great comfort."
"I don't know, I went to church outside and I still go."
"Yes, I found a lot of solace in religious faith."
"It has given me better hope and kept my mind clean."
"It has helped me to bear up."
"Knowing that if I kept my good health I might see the outside, it gave me the spirit."
"I feel better when I come out of church. I pray morning and night."

B. *Those who were indifferent to, or denied a religious belief.*

"I never went much for religion."
"I have nothing agin religion."
"I can't see where religion would do me much good."
"No, I haven't paid much attention to church."
"No, I was never much on religion."
"I never was against it."
"Well, not much."
"I was never so religious."
"I went the first few years and just let it go."
"Religion ain't hurt me or did me no good."
"I used to attend church but seldom go any more."
"I see so much preaching and doing another, I think it is a pretty bad place to try that."
"After you grow old you drop away from things."
"I never had much of a religious bent."
"I couldn't say that religion helped me to do my time."

Relative to subjective mental changes, no one felt that the long period of imprisonment had had any embittering or deteriorating effect on him. On the contrary, many felt themselves bettered by their experience except for the inevitable encroachment of age. Typical of our subjects' answers to the question of whether these years of imprisonment had effected their mind in any adverse manner are the following:

"Not one bit."
"It has made a wonderful change in me in the way I think of others or property."
"The longer I live the less I think of a thief."
"Not much."
"I have kept on reading of things, taken it easy as I could so I would not go to pieces."
"Well, I don't think so. It hasn't bothered me any."
"I might have gotten a little sluggish that is about all."
"Not that I know of."

"I am better. I think I reason a lot better and have written articles for the prison paper."
"Not that I know of."
"I don't think so. I think I have the same mind as before I came here."
"It has not affected me so far."
"No, sir."
"I don't think so."
"None, I just make the best of it and don't worry."
"It has improved my mind."
"I think along the right things."
"Not that I can see."
"I feel all right and I try to look at everything in the right light."
"I don't think so."
"Not so I know of."
"No, I have always been constructive."
"I read high class books, high class literature and have good thoughts."
"My mind has been improved considerably."
"I have made it a practice not to let it."
"I study grammar and learn anything I could if it is anything to improve it. If I had been on the outside I wouldn't have gotten the chance."
"I think my mind is just as good as the day I came in."
"I think it has broadened my mind."
"I think I do more reasoning before acting."
"They have aged it."
"I can't see why that it would."
"You naturally lead a cleaner life than you do on the street."
"I am considerably older and I don't think my mind is alert."
"I can't remember names so well."

It can be stated that none expressed a reduction in alertness or general interests, except those advanced in years who attributed it to that cause. With the exception of two, they appeared to accept imprisonment placidly and not without some satisfaction. It is felt that to some, imprisonment represented a venture on a new life, they rejected the old emotionally, and accepted the new with considerable gratification.

In summarizing it may be stated that there had been relatively little modification in interests so far as attitudes toward crime, religion or insight is concerned. Objectively, the examiner would be unable to detect "prison stupor" in the cases making up this study.

Attitude Toward Punishment.

Twenty-five inmates stated that they deserved punishment although only 22 felt they were guilty of actual murder. Five insisted

they were being unjustly punished for a crime they did not commit. With one exception, all insisted that the sentence was too long and generally added that a term of 14 years, which in Illinois is the minimum sentence for murder, would have been more just. None would have preferred the death penalty to life imprisonment and none had ever attempted suicide while in the penitentiary. When asked whether they had ever contemplated suicide in preference to the penitentiary they invariably answered with a chuckle to indicate the absurdity of the question.

On the question of deterrence, there was a preponderance of opinion in the negative. Twenty answered that long sentences do not deter lay people from committing a crime; three, that they do deter; and seven that the matter was unsettled for them. Typical answers as to whether long sentences deter from crime are as follows:

A. *Positives.*

"Yes sir, yes sir, it is just a lot of people if they know what sentence it called for would not commit crimes."

"Yes, I think it acts as a deterrent."

"Yes, the certainty, not the severity of punishment would do to keep others from crimes."

B. *Negatives.*

"No, because I know in my case I never gave a thought to what would happen to me."

"I don't think it would because things like that is liable to happen you know."

"I don't because fellows that commit a crime they figure they won't get caught."

"What has? Life sentences and so on never stopped anyone."

"Absolutely no."

"No, I am satisfied to that."

"I don't hardly believe it."

"I don't think anyone who commits a crime like that gives it any thought."

"No, Doc, people never think that."

"It is no deterrent to crime and it has only done injury to my family."

"No, I don't think they even notice it."

C. *Questionable.*

"It might."

As can be deduced from some of the answers, the reply was often determined by the inmate's own crime. If he considered his crime

preventable, then he felt punishment was deterrent; if his crime was of an emotional or what he believed accidental nature, the punishment was not deterrent.

It may be said that the offenders who were the subjects of our study accept punishment for a crime without question, but want it reduced to a minimum. They tend to accept the dictum of punishment abstractly without any regard to its avowed purpose and except for a small number do not consider it a deterrent.

Interest in Freedom.

All wanted their freedom, but with the exception of three said they would not escape if given an opportunity. We already know that 13 are on the honor farm where an escape could at least be attempted without too great a risk. The three who considered themselves not trustworthy had previous criminal records and two had made escape attempts. Some of the men, particularly those on the farm, lead a not too unpleasant existence, but they were all willing to barter it for an uncertain future. It must be stated, however, that only nine showed a strong affective response to this topic. Some, while paying lip service to hope, are resigned to remain prisoners the rest of their lives, and some, the examiner felt, are strongly ambivalent in their aspirations for freedom and with a bit of persuasion would accept the *status quo* as against a choice of freedom. Only half would have been prepared to meet the requirements of parole and a few more had some constructive plans for a life of freedom.

Social Attitudes in General.

The subjects were asked to express opinions about social morality in general, their attitudes toward law enforcing agencies, and prison officials. None seemed to place themselves in opposition to society, or consider society a force or unit detaining or punishing them. They were all warm and friendly to the "outside" and while two expressed animosity towards specific elements in society they did not indict society as a whole. One wit when asked what he thought was the reason society kept him in the penitentiary replied, "Doc, society does not know I am here." Not a single prisoner had formed a close attachment to any other inmate or

official. Replies here were very broad. Some were friendly to and others had preferences for certain persons, but none could point to a single individual as a close friend. There was considerable hostility displayed towards law enforcing agents (guards) by those with criminal records. Hostility was absent, however, among the first offenders.

Discussion and Conclusions.

While imprisonment is currently the accepted method of treating offenders, it cannot be said to be a successful one. Recidivism is a common occurrence and the answer to that has generally been new prisons. It is altogether possible that incarceration should not have been prescribed in the first place either because it is ineffectual or because it might even be harmful. At any rate, the effect of present day imprisonment on men who commit crime is a subject for investigation and should form the basis for an intelligent penal program. Even though it is concluded that imprisonment is inescapable, there is still the need to understand the prisoner so that at the expiration of his sentence he may be helped toward a satisfactory extramural adjustment.

The subjects of this study, namely, 30 murderers, are not to be considered a representative sample of the prison population. They were selected because they had served the longest penitentiary sentences and it was believed that the effects of imprisonment would be most pronounced in them. At least two factors would differentiate our selected group from that of the population in general. From a legal standpoint few of our cases were in conflict with the law before their instant offense and emotionally they are to be considered a comparatively stable group at least so far as prison adjustment might indicate.

In giving our conclusions, we would emphasize that they are to be taken only in the broadest sense and that for more specific findings a thorough individual psychiatric study of each subject would be required.

A study of 30 murderers, incarcerated in a state penitentiary for a period of 20 years or more, reveals the following findings:

1. Imprisonment has been accompanied by progressive social severance.

2. The basic interests of these men with reference to religion, crime and intellectual sphere were but little modified by years of imprisonment.

3. All those who admitted that they were guilty of the crime for which they were sentenced felt that punishment was justified, but that their sentence was too severe.

4. While there is universally, among these inmates, an expressed desire for freedom, it appears that many have become resigned to life imprisonment and without help would probably be unable to make an extramural adjustment.

5. After many years, imprisonment finds nearly all the subjects friendly and without antagonism to society.

DISCUSSION.

DR. KARL BOWMAN (New York City).—I think this paper has several points that are of especial interest. First, we send people to prison just as we place them in mental hospitals, often for a long period of time, and sometimes we lose sight of what is the effect of a long stay of that sort upon the individual. In our mental hospitals, one has stressed many times the deteriorating effect that occurs if we do not have a suitable organization in the hospital. The poorly run hospital we say has more deteriorated cases of schizophrenia. Catatonics are in much higher percentage in such hospitals whereas the hospital that has a well organized occupational therapy department and makes other efforts of that sort has less deterioration. The same with regard to prisons. Few of us, I am sure, have paid much attention to what a prison does to an individual. I think most of us have assumed that an individual becomes embittered, becomes hardened, and that the average individual who spends 20 or 30 years comes out, if anything, more hardened and more embittered; therefore a study of this sort is of great interest to us to see what an environment such as prison does to an individual.

The first point I should like to make is that in this study, as the reader said, the persons examined were carefully selected. It is not a representative sample. It does not tell us what is the general effect upon all prisoners; in fact, it is a selected group of 30 prisoners who are well adjusted at the end of the period. We are told, for example, that none of these prisoners had attempted suicide. One suspects, of course, that the individuals who did, at least some of them, would be classified as psychotic and so placed in another group. If we want answers to such questions as: Does a long-term imprisonment produce psychosis? Does it tend to make an individual wish to commit suicide? This article does not give us the answers. I say that not by way of criticism because the author himself pointed that out.

As to the technique used, it seems to me that the technique was excellent and should be commended to those who are making studies of this sort. We

have grown so accustomed to the questionnaire, which is filled out in a more or less unthinking fashion, that it is refreshing to see an attempt made to get a series of answers to a group of questions of this sort, but to get it not by simply asking questions 1, 2, 3, of a group of prisoners, but carrying it out in this informal fashion, stopping to smoke a cigarette, to chat about the weather or any other topic, and without the prisoner knowing what are the really important things on which the examination is being focussed. So it would be my feeling that a study of this sort is very useful, that the technique given is exactly what should be used for further studies, and that the conclusions of the author seem to be well established and in conformity with what one would perhaps have expected in most cases but not in all.

I was interested in the fact that not a single prisoner had formed a genuine attachment to another prisoner or to an official. I would have imagined that there would have been a certain number of prisoners who would have formed some type of positive attachment to some official over them. The optimistic attitude of the prisoner suggests the possibility that some degree of emotional deterioration may have occurred.

DR. B. R. WEBSTER (Dannemora, N. Y.).—It goes without saying that whether we spend our time in prison or outside during a period of 20 years, our environment will have a profound effect upon us. However, we must bear in mind that this effect is not that of a situation upon a person as a unit, but of a situation upon an individual as a personality; that is to say, the effect of the same environment will vary with the different personalities upon which it plays. As to personalities, Dr. Sukov's group, as he himself has pointed out, is an extremely limited one. In the first place, all of his group have made a very satisfactory adaptation. It would be interesting to note the proportion of those who did not adapt and what were their reactions. In the second place, his group consists entirely of murderers. Because of its tendencies to self-preservation, society has always looked upon murder as an especially heinous offense. From the standpoint of the psychiatrist a murder is in perhaps the majority of cases an emotional reaction to a situation rather than a crime, and while a group of 30 murderers would undoubtedly be an abnormal group the abnormality is not that which generally goes to make up our prison populations.

It would, therefore, be of great interest to note to what extent the personality changes Dr. Sukov finds would be paralleled in a general prison population. One of these personality traits, however, rings a very familiar note, that is his prisoners' attitude toward freedom. I think that the average convict looks forward to freedom in much the same way that the average person looks forward to heaven. The very fact that it is freedom postulates that everything will be satisfactory and therefore will need no planning. The fact that freedom may be accompanied by difficulty and perhaps even by danger never seems to have entered the head of the average convict. At least in my own experience I find that patients even when recovered and looking forward to release in the near future seldom make detailed plans for their freedom.

One might wish that under the heading of social severance Dr. Sukov had investigated what changes there were in the subjects' general interest in current events of the outside world. Perhaps such questions could not well be included in the questionnaire which was necessary to keep the investigation uniform, but I should like to ask Dr. Sukov whether in his general conversation with these men he gained any impression in this regard.

I was rather surprised at the slight degree of undesirable personality changes that the investigation unfolded; however, our own experience at the Dannemora State Hospital would tend to substantiate these findings. In a review of 100 consecutive cases who were serving sentences of 20 years or over I find that 43 per cent were admitted after 18 months or less of prison residence and only 10 per cent after 10 years of prison residence. While this is, of course, a rather crude method of approaching the subject, it would seem that if long continued prison residence had a very deleterious effect upon personality we should find these figures reversed.

The further question arises as to what extent the changes found are due to prison environment and to other causes. I have in mind the fact that a goodly proportion of the prison population is composed of psychopathic individuals and I have reason to believe this would be equally true of a population composed of murderers. While I have never seen the statement in the literature, it has been my personal experience that those traits which are peculiar to the psychopath tend to submerge with the passing of time and that the psychopathic individual over 40 is a much safer unit of society than one under 20.

Dr. Andrew H. Woods (Iowa City, Iowa).—I want to make one comment on this excellent paper to which Dr. Bowman in part referred, the apparent absurdity of intelligent people building a lot of huge buildings and packing into them a lot of human beings with apparently no purpose whatever unless it may be to frighten them from doing evil another time. Dr. Bowman suggested the contrast between our prisons and our hospitals. Imagine a hospital out of which the patients, as a rule, were expected to come worse than they were when they went in. Imagine a hospital so contrived that every prisoner who comes in shall become aware at once possibly of hostility; if not of hostility of marked indifference of the ones who stand in the place of physicians and nurses in a hospital. Imagine a hospital in which the patients when they come in will have free access to other patients who will at once commence to tell them that cure is impossible, that the continuance and increase of this disease is the desirable thing, and will give all aid possible to increase the disease of those persons who have already been admitted.

Now, I do not know when I have been more depressed than by the obvious conclusion which we must take from Dr. Sukov's paper. It was just remarked that possibly these prisoners maintain their personality. I would disagree with that very decidedly. The picture that has been drawn for us out of a proper sampling has been of steady deterioration of all those personality

reactions which make a man fit for living in the world. He has lost his interest in his family; he has lost his interest in society; he has lost his interest in himself; he has lost his interest in religion; has no interest in morals. This isn't because he has vitiated, but is simply a process of atrophy.

If we were to shut up a man with perfectly good vision and to blindfold his eyes so that he could not see, we would expect the retina and the whole optical mechanism to atrophy. The same thing for muscles. The brain is a mechanism that will atrophy if it is not kept active by meeting situations day by day; and the finest qualities of the brain have to do with what we denominate as personality. To be shut up in a prison without friendships, not encouraged to maintain his outside contacts in some way, without games, without occupation, without purpose is a situation very much like that we still sometimes find in our state hospitals; which to that extent may be subject to the same criticisms as our prisons.

I wish the time would come when the prison authorities and society would wake up to the fact that something can be done for the inmates of prisons.

Let us keep in mind the terrible contrast between our good hospitals and these prisons. The prisons can be made just as therapeutic as the hospitals are. We should be satisfied with no lesser objective.

DR. E. WEXBERG (New Orleans, La.).—I feel that the answers given by the prisoners are, among others, determined by one factor that seems to be very essential for the interpretation of this material: by the fact that prisoners are still in prison and that to be in prison does not represent a natural condition of a human being; and, therefore, it would be the crucial experiment for interpretation given here to find out how these identical prisoners would answer these identical questions, or about the same questions, once they are released and are living under normal conditions again. I am almost sure that the changed situation, the fact that they have to face life again, would entirely change their outlook and their response to these questions. It may be that embitterment which seemed to be entirely absent in these answers and which obviously was in their conscious minds would come out once they would have an opportunity to face life again and we would then find out how far they have changed from what is supposed to be a normal human being in a normal society.

DR. MARVIN SUKOV (Chicago, Ill.).—The question as to whether their optimistic attitude would represent deterioration I have not been able to settle. We do know that these prisoners have been out of contact with the outside, with the needs for obtaining employment; we will say they are living in a highly guarded environment. Now, whether that attitude is personal, whether it would represent actual deterioration, or whether, as the last speaker indicated, once they got outside that attitude would change, I am not able to say and for that reason I should not call it deterioration.

They nearly all showed a rather lively interest in current events. They read newspapers and magazines, mostly the picture type of magazine which circulates in the institution rather freely. I suppose it may not be out of place again to emphasize my own clinical opinion that the findings we get here do not represent those that we would get from the population at large. These men all had better-than-average jobs. Now they are on the farm, they do not live in cells, they live in dormitories; they eat better food than the average prisoner and they dress a little better; they are tanned and in more robust health than average prisoners. Necessarily we would be extremely cautious in drawing from these special cases conclusions regarding the population in general.

TUBERCULOSIS PREVENTION AND TREATMENT IN ONTARIO MENTAL HOSPITALS.

By C. A. WICKS, B. A., M. D.

The purpose of this paper is to present to those interested in the clinical and administrative aspects of hospitals for the mentally ill, the experience in tuberculosis control gained in Ontario mental hospitals during the past nine and more particularly during the past six years. A brief review of selected references from the literature will be followed by a chronological account of the development of this work in Ontario, together with an outline of the present policy and a series of tables showing the findings and results obtained.

Tuberculosis among patients and members of staff in mental hospitals has always been a problem to both hospital clinician and administrator. During recent years, considerable interest and discussion has been evident in this subject. Many of the older conceptions regarding incidence, case-finding methods, and control, have been replaced by more modern practices, designed to search objectively for this disease among all patients and staff in such hospitals.

As early as 1900, Harrington [1] recommended "separate provisions for tuberculous patients in state hospitals for the insane." Klopp [2] in 1927 reported, from a questionnaire survey of 106 mental hospitals in the United States, that 59 hospitals possessed specially constructed detached pavilions or buildings for the isolation of tuberculous mentally ill patients. Klopp further reported that of these 106 hospitals, 55 were taking chest X-rays upon suspected cases of tuberculosis, but only one hospital secured chest X-rays for all new patients as well as the entire patient population. The number of beds required for the segregation of tuberculous patients in mental hospitals was estimated at approximately 5 per cent of the total mental hospital population. Greene and Woodall [3] in 1931, following a survey at the Fernald State School by tuberculin testing and X-raying 1681 patients in residence, found 1.5 per cent to have definitely active, and a total of 3.6 per cent with sig-

nificant pulmonary tuberculous lesions. McGhie and Brink [4] in 1933 reported a survey of 1209 resident patients at the Ontario Hospital, London. All patients were tuberculin tested and all positive reactors X-rayed. They found 91.5 per cent positive to tuberculin, 3.7 per cent to have definitely active tuberculosis, and a total of 5 per cent showing significant pulmonary tuberculous lesions requiring isolation. Fellows [5] in 1934, reporting upon a five-year-fluoroscopic and X-ray study of approximately 18,000 individuals applying for work at an average age of 18 years, found slightly higher than 1 per cent of apparently well persons to have definite pulmonary tuberculous disease. He concluded that active tuberculosis occurs in adults more frequently than is generally recognized. Bogen, Tietz and Grace [6] in 1934, following a tuberculin survey of patients at Longview State Hospital, stated that tuberculous infection among mental hospital patients increases according to the length of their stay in institution, and indicated that probably the majority of such patients contract their infection as well as the disease after their admission to mental hospital. Heimbeck [7] in several papers has pointed out the high incidence of pulmonary tuberculosis among nurses. He believes that the nurse entering her hospital training negative to tuberculin, not having had a protective infection with tubercle bacilli, is therefore more likely to develop tuberculous disease than one who enters with a positive reaction. His recent papers report the practice of vaccinating with B. C. G. those nurses who present themselves for hospital training without previous tuberculous infection. Burns [8] in 1936, reported the results of a tuberculin and chest X-ray survey of all patients and staff in the state institutions in Minnesota. In this survey it was found that among 11,517 patients, 82 per cent had positive tuberculin reactions and 9.2 per cent showed evidence of parenchymal infiltration on X-ray. Among 2430 employees, 72 per cent had positive tuberculin reactions and 3.7 per cent were found on X-ray to have parenchymal pulmonary tuberculosis. Harrison and Schein [9] in 1937, report a fluoroscopic and X-ray survey of 2186 mental hospital patients who were ambulatory and not suspected of having tuberculosis. A total of 3.6 per cent of this group were reported by the authors to show X-ray evidence of pulmonary lesions. J. A. Myers, Trach, Diehl and Boynton [10] in 1938, discuss very completely the problem of tuberculosis among

hospital personnel and mention that already legal actions have been taken against hospitals as a result of patients or employees contracting tuberculosis from infection received within the hospital. For the protection of the hospital, it would appear that information should be on file regarding the tuberculin reaction and chest X-ray of all new staff and patients. Leonidoff [11] in 1938, describes what would appear to be a scientific approach to the treatment of tuberculous mentally ill patients but advises against centralization of such patients, and reports a chest X-ray survey at his hospital only among a limited group of patients.

In the Province of Ontario there are 14 widely scattered mental hospitals of various sizes, controlled and operated by the Provincial Government. The total bed capacity is approximately 14,000 with a staff of employees of approximately 2500. A recently commenced building program will increase the capacity within several years to almost 17,000 patients. One hospital accepts only patients with mental deficiency, and another accepts only patients having epilepsy with or without psychosis. The remaining hospitals accept all classes of mentally ill persons.

Prior to 1930, there was no correlated or systematic anti-tuberculosis work being conducted in the Ontario Mental Hospitals. Diagnosis of tuberculosis in most cases was made upon symptoms or physical signs. Unless satisfactory specimens of sputum could be obtained, seldom was there an opportunity to confirm the diagnoses because relatively few of the hospitals possessed X-ray equipment. When the diagnosis of tuberculosis was established, the disease was usually in the far advanced stage and obviously many other patients as well as members of the staff, must have been heavily infected. Such tuberculous patients, usually ill and febrile at the time of diagnosis, were isolated as well as possible. Due to the arrangement of the wards and facilities, however, as well as the frequent lack of cooperation on the part of the patients, such isolation was far from adequate.

In 1930, Dr. B. T. McGhie, now Deputy Minister of Health, and Dr. G. C. Brink, now Director of Tuberculosis Control in Ontario, arranged that an X-ray technician accompanied by a portable X-ray machine, be sent to certain Ontario mental hospitals to secure chest X-rays upon those patients and staff suspected by the medical staffs to have some chest disease. The X-ray films so

taken, were brought for development and interpretation to the office of the Provincial Tuberculosis Prevention Branch. Later, a chest clinician from that office, visited the hospitals to examine those cases where the chest X-ray showed definite or suspicious evidence of disease. In this manner, during that year, small groups of selected patients and staff were X-rayed at several hospitals.

In 1931, the above method was continued and a greater number of the hospitals were visited by the X-ray technician, followed by the visit of the chest clinician. Also, during that year, arrangements were commenced to install permanent X-ray equipment in several hospitals. Toward the end of 1931 as a result of this diagnostic work, small groups of patients in many of the hospitals were requiring isolation because pulmonary tuberculosis had been diagnosed. Such isolation was difficult to maintain. Furthermore, with one small ward or section of a ward at each hospital devoted to this isolation and segregation, there was little or no opportunity for further segregation of the tuberculous patients according to mental condition. As a result of these and other considerations, it was decided to set aside one cottage or building at the centrally placed Ontario Hospital at New Toronto, to accommodate patients with active tuberculosis from all the Ontario Mental Hospitals. Accordingly, late in 1931 the transfer was begun of such cases from the other hospitals to the tuberculosis unit at the Ontario Hospital, New Toronto. One of the physicians on the staff of that hospital was given some special training in chest diseases, and the care of these tuberculous mentally ill patients formed a portion of the duties of this physician.

In 1932, the program of transferring patients with active tuberculosis to the T. B. unit continued. Further visits were made to most of the hospitals by the X-ray technician accompanied by the portable X-ray machine. All student nurses entering their probation period were X-rayed. In that year also, there was conducted a tuberculin survey of all patients and staff at the Ontario Hospital, London, which work was followed by a chest X-ray survey of all positive tuberculin reactors among the patients and staff at that hospital.

In 1933, in view of the increasing work entailed in the extending program of diagnosis and treatment, the appointment was made for this work, of a full time clinician who had already had a

year's experience in a mental hospital, and another year in a tuberculosis sanatorium. With this appointment of a full time physician, the program became enlarged to include regular visits every three to four months with the X-ray equipment, to each hospital. At these visits, all new members of staff were tuberculin tested and X-rayed, previously negative tuberculin reactors retested, and patients with known arrested or inactive tuberculosis as well as new cases referred by the medical staff of the hospitals were X-rayed and reports submitted. An increasing number of the hospitals were supplied with permanent X-ray equipment. Patients found to have active tuberculosis were recommended for transfer to the T. B. unit at New Toronto. Staff found to have active tuberculosis were also transferred for treatment to the T. B. unit where they were placed in a separate section of this unit. The treatment of patients was placed under a regime resembling as nearly as possible that of a sanatorium. Artificial pneumothorax treatments were commenced in selected cases.

In 1934, the program as enlarged in 1933, was continued. A complete circular describing the anti-tuberculosis work with instructions as to the manner of selecting cases for X-ray, as well as the routine procedures expected, was sent to each hospital. Since at that time, facilities and staff were not sufficient, nor were funds available for the X-raying of all patients in all the hospitals, an attempt was made to introduce some objective finding or sign which could be used as a "screen" to select patients who should be X-rayed. The hospitals were therefore requested to conduct, in addition to the already established case-finding methods, temperature surveys of all patients in residence during one week every four months. Temperatures per axilla were taken at 4 p. m. or 8 p. m. as convenient upon all patients and the findings recorded on individual temperature charts. The physicians in charge of the patients reviewed the charts at the end of the survey and brought forward for chest X-ray all patients showing an unexplained axillary temperature of more than 98° on four or more days during the week. This measure resulted in an increased number of cases found to have active pulmonary tuberculosis, and to this degree the plan was useful. Experience showed, however, that although fever was a rough "screen" to detect active tuberculosis, certain infectious cases were missed because if in bed (due

to mental condition) such patients might not exhibit any elevation of temperature. Furthermore, a large number of mentally ill patients showed slight rises of temperature without any evident organic cause, certainly without any abnormality in the parenchyma of the lung fields as visualized in the chest X-ray. It was clear, therefore, that some more delicate and sensitive method was required to assist in the case-finding program.

In 1935, after a preliminary survey by the chest clinician at one of the hospitals, all the Ontario mental hospitals were requested to perform blood sedimentation tests (Westergren Method) upon all resident patients. Those patients showing unexplained blood sedimentation rates greater than 10 per cent in the first hour, were referred for chest X-ray. This plan resulted in a still greater yield of patients showing active tuberculosis, but offered difficulties from the standpoint of time required by the hospital physicians, and lack of complete control of the details of technique as employed at the various hospitals. Furthermore, certain infectious cases might be missed because their disease might be quiescent although they were chronic spreaders of infection. During this period, the number of patients requiring treatment in the T. B. unit greatly increased, requiring an increasing amount of space in an adjoining building at the Ontario Hospital, New Toronto, to be taken over for this purpose.

In 1936, a further attempt was made to find a relatively inexpensive method of selecting patients for X-ray, and thereby develop a routine case-finding system among patients which could be carried out in periodic surveys at each hospital. The chest clinician completed a fluoroscopic X-ray examination of the chests of approximately 1200 mentally ill ambulatory patients in the non-tuberculosis section of the Ontario Hospital, New Toronto. For those patients showing definite or suspicious findings in the fluoroscopic examination as well as for those additional patients who could not stand up for fluoroscopy, chest X-ray films were taken. In all, among 1267 patients not previously known to have tuberculosis, it was found that 2.0 per cent had definitely active and 2.3 per cent showed either X-ray shadows suggesting tuberculosis or tuberculous lesions where activity was questionable, making a total of 4.3 per cent requiring isolation. These findings are even more striking and more significant when it is considered that during the

2 years previous to the fluoroscopic survey at this hospital, temperature surveys had been conducted every 4 months, and just 6 months previously, a blood sedimentation survey had been completed with all cases diagnosed by these methods removed to the T. B. unit. It was found difficult and time-consuming to attempt to fluoroscope many mentally ill patients in a darkened room. Accordingly, a wooden box, specially constructed with a light-proof vent in the top, was attached to the fluoroscopic screen, counterbalancing weights being proportionately adjusted. A black photographic focussing cloth was thrown over the open end of this box and the observer inserted his head into the box. An electric fan directed at the air-vent gave ventilation within the box, and the observer could keep his head within, and his eyes therefore properly accommodated for several hours at a time while nurses and attendants arranged the patients behind the fluoroscopic screen in the lighted room. By this method, cases of minimal pulmonary tuberculosis were detected. Furthermore, an analysis of the findings in a survey at this same hospital by chest X-ray films one year later, would lead one to believe that very few, if any, significant chest lesions were missed by the fluoroscopic examinations. This method, however, represents rather arduous work for the observer, particularly if practiced for many hours each day, as would be necessary to survey large numbers of patients within a reasonable time. The equipment should also be completely shock-proof and thoroughly shielded to prevent danger of electrical shock to the patients and excessive X-ray exposure of the observer. During this same year, a second physician was appointed to care for the increased number of patients in the T. B. unit and to relieve the original clinician to devote a greater part of his time to diagnostic work.

In 1937, the accommodation for tuberculous patients in the T. B. unit at the Ontario Hospital, New Toronto, became taxed to capacity and arrangements were made to establish an annex to this unit at Concord near Toronto, to treat patients from the T. B. unit at New Toronto, who did not require intensive care for their mental or physical conditions. This brought the total bed capacity for active tuberculous patients in the T. B. units at New Toronto and Concord to approximately 300 beds. At the same time, it was

decided to discontinue the treatment of tuberculous staff at the T. B. unit, and those already under treatment were transferred to sanatoria. From that time all established members of staff found to have active tuberculosis have been treated in sanatoria at the expense of the Provincial Government.

Early in 1937, it became evident that although of considerable value as case-finding methods, none of the plans already given fair trial were either sufficiently practical or adequate to detect all cases of tuberculosis and effectively control the spread of this disease within our mental hospitals. At that time, the appropriations were sufficient to cover the expenditure involved, and authority was granted for securing chest X-ray films upon all resident patients in yearly surveys and upon all new patients at the time of admission. All tuberculin positive employees were also to have chest X-ray films in yearly surveys. (Since 1933 all new members of staff had been tuberculin tested and X-rayed, with repeat tuberculin tests every six months on all negative reactors.) An additional clinician well trained in chest diseases as well as two additional X-ray technicians were supplied to undertake the production and interpretation of the large number of chest X-rays required under this scheme.

A comprehensive circular was prepared and distributed to all physicians in the hospitals, outlining in detail the program of tuberculosis prevention as well as the responsibilities of each officer in this program. The following scheme is therefore that in operation at the present time in our Ontario mental hospitals in regard to tuberculosis case-finding among patients and members of staff:

(1) All patients as soon as possible after admission to hospital receive a chest X-ray (usually within the first week).

(2) All staff applicants when applying to fill a definite vacancy receive a chest X-ray.

(3) All new employees in any capacity (including nurses reporting for affiliation in mental hospitals and those returning from prolonged leave of absence) not already X-rayed within the previous month as applicants, receive a chest X-ray within three days of reporting for duty. (All applicants or new staff are X-rayed without preliminary tuberculin testing simply because the travelling chest clinician who does this testing can visit the hospitals only every six months. The individual hospitals have been requested however to perform these routine tuberculin tests for any re-employed staff previously tuberculin-negative.)

(4) All employees in any capacity before resigning, proceeding on prolonged leave of absence, or leaving for affiliation courses, receive a chest X-ray. Where possible, negative tuberculin reactors receive also a repeat tuberculin test before resigning.

(5) Any member of staff in any capacity or any patient who presents symptoms referable to the chest, may be referred for a chest X-ray at any time by any of the hospital physicians.

(6) All patients who are selected to receive insulin shock therapy or metrazol therapy, if not X-rayed within the previous three months, receive a chest X-ray before such treatment is undertaken.

Note.—All the above work is performed by the individual hospitals. Single, safety X-ray films are used unless stereoscopic films are specially requested. Each hospital sends to the office of the chest clinic at the Ontario Hospital, New Toronto, at the end of each week, all such chest X-rays taken during that week. These incoming films are interpreted by the two chest clinicians attached to the T. B. unit, and individual reports are prepared, copies of which are sent to the hospitals for the case records of the patients or the personal files of the employees. In the case of all new staff, and in patients where there is evidence of chest disease, a further copy is forwarded to the Director of Hospitals Division in the Department of Health. Any staff applicant or new member of staff showing evidence of chest disease is considered by a Special Board in the Department of Health, as to acceptability for employment. These not acceptable are so notified and referred to their family physician, with recommendations. New patients showing pulmonary tuberculosis are recommended for transfer to the T. B. unit if the disease appears active, or for isolation and investigation, if the activity of the lesion is questionable. In any case of active tuberculosis discovered in this manner on entering hospital, an attempt is made to secure information regarding contacts, and notification is made to the Ontario Division of Tuberculosis Control.

(7) At intervals of one year, the travelling X-ray technician is sent to each hospital to assist in the taking of chest X-ray films for all patients and all tuberculin positive staff at the hospital at that time. Patients placed in boarding homes are also included in these surveys. (Only at the Ontario Hospital School Orillia for mentally deficient persons, does a tuberculin survey precede the X-ray survey. At this hospital-school, all patients under 25 years of age are tuberculin tested, and only the positive reactors and the remaining patients 25 years of age and over are X-rayed.) When the films have been processed and arranged in order, the traveling chest clinician arrives for his work, and the X-ray technician proceeds to the next hospital to be surveyed. The work of the clinician at this visit, is as follows:

(a) Interpretation of all the chest X-ray films taken in the complete survey. (Individual reports are completed for all staff but

only for those patients showing evidence of chest disease. Records of all findings, negative or positive, are made opposite each name on lists showing alphabetically all patients and staff at the hospital.)

(b) Examination (including blood sedimentation tests where indicated) of all those showing definite abnormalities in the chest X-ray.

(c) Tuberculin testing all members of staff in any capacity, who have entered the service since his last visit.

(d) Repeating tuberculin tests upon all employees previously negative to tuberculin. (When a change in reaction occurs, the employee is X-rayed immediately, and even if this X-ray is normal, he or she is closely watched while on duty and the X-ray repeated in six months.)

(e) Reporting to the Superintendent of the Hospital (copy to the Director of Hospitals Division) the detailed findings and recommendations.

(8) In six months after each complete survey (mid-way between such surveys) a list is prepared and sent from the chest clinic headquarters at New Toronto, to the hospital, indicating those cases among patients and staff who require repeat chest X-rays or reconsideration. The hospital secures these chest X-rays which have been recommended, and the travelling chest clinician visits the hospital, when his work is again similar to that described above.

Note.—In the above manner, two visits are made by this chest clinician to each hospital every year. At each visit, all new staff are tuberculin tested and previously negative reactors retested. Mantoux tuberculin tests are given in all this work. Connaught Laboratory Old Tuberculin is used, and to those reacting negatively to the first test employing 1/20 m.gm., a second test is given using 1.0 m.gm. of Old Tuberculin. All patients found to have definitely active tuberculosis are recommended for transfer to the T. B. unit. (Activity of lesion is determined by a complete consideration of the case, including type and nature of the X-ray shadows, when possible by comparison between films taken at different intervals, blood sedimentation rate, cough and sputum, etc.) Those patients showing suspicious X-ray evidence of tuberculosis or a tuberculous lesion where the activity cannot be determined, are recommended for isolation and segregation, pending further investigation at the hospital. This investigation includes mainly, temperature and weight records, repeated sputum examinations for tubercle bacilli on direct smear and culture, as well as the repeat chest X-ray in six months. (The majority, if not all, of such patients might be better placed in a T. B. unit, but lack of accommodation makes it necessary to accept in the T. B. unit only those cases where the disease is definitely active.) If and when a patient under isolation at any hospital, either awaiting transfer to

the T. B. unit or pending investigation of the chest disease, recovers from his or her mental illness sufficiently to warrant probation or discharge from mental hospital, a chest X-ray is secured at that time and forwarded together with previous X-rays and pertinent information, to the Chest Clinic at New Toronto. The case is reviewed and a report returned to the hospital advising either admission to sanitorium or return to the home. In those cases returning home in this manner, notification is made in the proper manner to the Ontario Division of Tuberculosis Control.

Any established member of staff developing active tuberculosis, is sent to a sanatorium for treatment at the expense of the Provincial Government. For such cases, their salary continues as long as this is permitted according to their length of Government service and as outlined in the regulations respecting the Ontario Civil Service.

(9) No negative tuberculin reactor among the staff, no employee until tuberculin tested or no undergraduate nurse even if a positive reactor, is allowed to work on wards where there are known cases of active tuberculosis or where patients are isolated pending investigation as to chest disease.

(10) A concise but complete lecture course on tuberculosis has been mimeographed and supplied to the hospitals for distribution among the nursing staff.

The following remarks might be made in connection with the treatment of tuberculous patients and staff from the Ontario mental hospitals:

(1) Reference has already been made to the disposal of incoming and established employees who are found to have active tuberculosis. In the case of established members of staff, following discharge from sanatorium, after-care until they are capable of resuming work is provided where necessary by the Ontario Government. During this convalescence, periodic re-examinations are arranged by the chest clinic at New Toronto, and the findings presented to the Board in the Department of Health, who determine when the employee is in a condition to resume duty. Arrangements are made by the hospitals to provide reasonably light work for these ex-sanatorium patients and they are examined every three months for some time following their return to work.

(2) Patients in the Tuberculosis Units at the Ontario Hospital, New Toronto and at Concord, receive so far as possible the routine of a sanatorium. A complete investigation including physical examination, chest X-ray, sputum examinations, blood sedimentation test, etc., is made on admission and again at the end of three months. From that time, the frequency of re-examination (including a synopsis of present symptoms, temperature, weight, etc., as well as chest X-ray, sputum examinations and blood sedimentation test) varies according to the condition of the patient, but usually such re-examinations are

made at intervals of six months and for no patient is the interval greater than one year. After the investigation following admission, each patient is conferenced by the two physicians in charge of these units and also with the other members of the medical staff of the Ontario Hospital, New Toronto. An attempt is made to outline the treatment which would be prescribed for a tuberculous person with a similar condition but mentally well. This treatment is then figuratively applied to the mentally ill, epileptic or mentally deficient patient in question, and those measures used which are applicable. Artificial pneumothorax treatments are given to all cases where the usual indications are present and where the patient would co-operate for these treatments. Phrenic nerve crushing as well as bilateral artificial pneumothorax have been performed in a relatively few selected cases. It is rather doubtful whether any of the other more radical types of chest surgery (with the posible exception of intrapleural pneumolysis) should be attempted in a tuberculosis mental unit. Patients for whom more major operations are indicated should be sufficiently well mentally to receive this further chest surgery in a sanatorium. In January, 1939, of 303 patients under treatment in the T. B. units, 30 or approximately 10 per cent were receiving artificial pneumothorax treatments. All patients receiving pneumothorax are fluoroscoped before each refill. In the treatment of such patients an attempt has been made to visualize and work for the betterment of the individual without emphasizing unduly either the tuberculosis or the mental illness. Bed rest is enforced only to the extent where such enforcement will not produce a response on the part of the patient which would be more harmful than a limited activity. Much can be done, however, by constant persuasion, and if a routine of rest in bed with toilet privileges is established, incoming patients will follow this example to a surprising degree. In January, 1939, of 303 patients under treatment, 6 per cent were on total bed rest, 58 per cent on bed rest with toilet and bath privileges, 23 per cent allowed to sit up by their bedsides, and 13 per cent were allowed up, clothed and assisting with light ward duties and walking outside in suitable weather.

Patients in tuberculosis sanatoria who become mentally ill are admitted directly to the T. B. unit at New Toronto. From 10 to 20 such patients are admitted annually, and those receiving artificial pneumothorax continue to receive these treatments while in the T. B. unit. Many of these patients recover their mental health sufficiently to return to the sanatorium to complete their treatment for tuberculosis.

Excited and acutely disturbed tuberculous patients are regarded as sources of gross infection to the staff and other patients, as well

as harmful to themselves by virtue of their hyperactivity. Because of lack of facilities for continuous bath therapy and the inadequate number of single rooms in our T. B. unit at New Toronto, such patients have necessarily been treated with chemotherapy or packs. Disturbed patients considered to be infectious who persisit in expectorating profusely about the ward without care, have been placed occasionally in bed under restraint with the bed enveloped by a gauze canopy. This method more or less effectively confines the expectorated sputum to the canopy and the bed clothes which can be changed frequently for disinfection while the sputum is moist.

Determination of the progress of the tuberculous disease and the amount of exercise permissible as well as the period when the disease is arrested in a tuberculous psychotic patient, presents considerable difficulty. Comparison between serial chest X-rays and blood sedimentation rates are the main reliable sources of information. Satisfactory specimens of sputum are usually difficult to obtain. Due to the frequent dilution of the specimen by saliva, cultural examination for tubercle bacilli is to be recommended rather than reliance upon the result of direct smears. It has recently been our practice to have patients on full exercise and where possible doing light work for at least six months and usually one year with negative sputum, normal blood sedimentation rates, and a stationary lesion by X-ray films, before they are considered suitable for return to the hospitals from which they came. In moderately and far advanced pulmonary tuberculosis, even with the above findings, it is doubtful if such patients should be returned to wards with non-tuberculous patients due to the great possibility of their disease becoming re-activated and the patient again infective.

The treatment directed toward the mental condition of the patients in the tuberculosis units at New Toronto and Concord is limited by several factors. The unit at New Toronto is relatively a small portion of the entire hospital. The tuberculous patients obviously could not be allowed to participate in the entertainments or other gatherings arranged for the larger number of non-tuberculous patients at that hospital. Due to lack of space in the building constituting the tuberculosis unit, facilities for recreation or amusement are lacking. Treatment has consisted mainly of psychotherapy

and what occupational therapy could be performed at the bedside. The unit at Concord, accommodating patients whose tuberculosis is more quiescent or even apparently arrested, is better situated, but the number of patients is limited to 73 and the building was not designed for its present purpose. It will be evident that many recognized types of treatment are not suitable for mentally ill patients with active or unstable tuberculous disease. Considerable individual attention as well as skill is required, therefore, on the part of the physician and nurses. Hypoglycemia or metrazol therapy have not as yet been applied to patients in the tuberculosis units due to the prevailing opinion that such treatment is contra-indicated by the presence of tuberculosis. It can be reported, however, that one patient with catatonic schizophrenia and moderately advanced pulmonary tuberculosis which became quiescent in the T. B. unit at New Toronto, was transferred to the "insulin ward," where she received a course of 75 hypoglycemic shocks with no exacerbation of the tuberculous disease. Furthermore, a number of patients never in the tuberculosis units, showing x-ray evidence of arrested minimal pulmonary tuberculosis, have been given metrazol therapy after careful consideration. It is intended to review these cases in the near future to determine if such treatment has reactivated an arrested tuberculous lesion.

Dishes and other utensils used in the T. B. units are not removed from these units for use elsewhere. All garbage from these wards is burned in an incinerator. All laundry coming from these units is placed in distinctively marked bags, transported to the laundry in special conveyors, and then sterilized before it is mixed for further operations with the laundry from the non-tuberculous wards.

No undergraduate nurse is employed in the T. B. unit at New Toronto or Concord. All members of staff must have positive tuberculin reactions before being accepted for this work. Chest X-rays are secured for all staff so employed (including the physicians on this service) every four months. Infectious disease technique is used in so far as is practicable. Gowns are donned and gauze masks worn on entering these wards and discarded on leaving. Nurses and attendants employed in the T. B. units have somewhat shorter hours of work and receive slightly higher salaries than do the

nurses and attendants employed on wards with non-tuberculous patients. All staff commencing work on the tuberculosis wards read and sign for their file an instruction sheet, outlining precautions to be used.

Neither the building at the Ontario Hospital, New Toronto, nor the annex building at Concord, were designed for the care and treatment of tuberculous mentally ill patients. The present capacity of these two units (approximately 300 beds) is far below that required to accommodate the number of tuberculous patients discovered in the Ontario mental hospitals by the complete case-finding program now in operation. For these reasons, the Ontario Government has commenced the construction of a hospital at Brampton, for the treatment and care of all tuberculous mentally ill, epileptic and mentally deficient patients in Ontario. This hospital will treat and accommodate only such patients and will be specially designed for this purpose, incorporating features found both in sanatoria and hospitals for the mentally ill. Those in charge of this work in Ontario are convinced that at least in that Province, centralization of such treatment facilities for tuberculous patients from the mental hospitals, offers the only satisfactory solution to the problem. Without such a central tuberculosis mental hospital, small units would be required at each of the 14 mental hospitals scattered throughout the Province. Very few if any of these hospitals at the present time, possess wards of satisfactory design with suitable equipment or service to care for this type of patient. Furthermore, the difficulty of supplying each hospital with a physician and nursing staff trained in the treatment of chest disease is obvious. Mentally ill patients with tuberculosis deserve treatment of a standard which can only be supplied by physicians and nurses who have had at least a definite amount of training in modern sanatorium methods.

TABLE 1.

MORTALITY FROM TUBERCULOSIS (ALL FORMS) AMONG PATIENTS IN ONTARIO MENTAL HOSPITALS.

Year ending.	No. of deaths from tuberculosis (all forms).	Patient population (No. on books at end of year).	Total deaths (all causes).	Tuberculosis death rate (per 100,000 patients).	Per cent of deaths due to tuberculosis.
October 31, 1928.....	60	9,775	690	610	8.7
October 31, 1929.....	79	10,032	741	790	10.7
October 31, 1930.....	66	10,390	725	650	9.1
October 31, 1931.....	39	10,930	655	350	6.0
October 31, 1932.....	68	11,498	813	650	8.4
October 31, 1933.....	67	12,236	687	555	9.8
October 31, 1934.....	73	12,883	695	566	10.5
March 31, 1935.....	29	5,365[1]	281	540	10.3
March 31, 1936.....	70	13,868	770	504	9.1
March 31, 1937.....	65	14,288	829	455	7.8
March 31, 1938.....	61	14,735	818	414	7.5

MORTALITY FROM TUBERCULOSIS (ALL FORMS) AMONG GENERAL POPULATION IN ONTARIO

Year.	No. of deaths from tuberculosis (all forms).	Population.	Total deaths.	Tuberculosis death rate (per 100,000 population.	Per cent of deaths due to tuberculosis
1936.....	1327	3,690,000	37,571	36	3.5

[1] Estimated.

From Table 1, it is to be noted that until the year 1934, there had been no definite downward trend in the tuberculosis death rate in Ontario mental hospitals. From that year there is evident a consistent decrease in both the tuberculosis death rate and the percentage of the total deaths due to tuberculosis. Comparison with the figures for the general population in Ontario, however, shows that for the year 1936, the death rate from tuberculosis in the Ontario Mental Hospitals was 14 times that for the Province as a whole.

TABLE 2.

WORK ACCOMPLISHED BY ONTARIO HOSPITALS CHEST CLINIC.

June, 1933 to June, 1938—5-Year Period.

Type of work.	Patients.	Staff.	Total.
Number of chest X-rays interpreted (some individuals X-rayed more than once)...	23,121	8,098	31,219
Number of tuberculin tests performed (some individuals tested more than once).....	1,305	7,028	8,333
Number of chest examinations performed (some individuals examined more than once).....	2,347	892	3,239
Number of cases advised for treatment because of active tuberculosis.....	674	60	734

Table 2 gives some indication of the amount of work which has been accomplished in the case-finding program from June, 1933, to June, 1938. From this table it may be calculated that one case of definitely active tuberculosis was diagnosed among the patients for every 34 X-ray films taken upon patients during that period.

TABLE 3.

RESULTS OF CHEST X-RAY SURVEY OF PATIENTS IN ONTARIO MENTAL HOSPITALS (1937-38).

(Excluding patients already under treatment in T. B. units at New Toronto and Concord.)

Ontario mental hospitals.	Total number of patients at time of survey.	Pulmonary tuberculosis definitely active		Pulmonary tuberculosis activity undetermined.		Suspect pulmonary tuberculosis		Total showing significant lesions.		Pulmonary tuberculosis definitely or probably arrested.	
		Transfer to T. B. unit.		Isolate and investigate at hospital.		Isolate and investigate at hospital.		Treatment or isolation.		Parenchymal. Not isolated.	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Brockville	1,084	17	1.6	55	5.0	9	0.9	81	7.5	106	9.8
Cobourg	452	4	0.9	22	5.0	18	3.9	44	9.8	25	5.5
Fort William	96	0	0	3	3.1	0	0	3	3.1	1	1.0
Hamilton	1,672	28	1.7	67	4.0	33	2.0	128	7.7	64	3.8
Kingston	1,200	16	1.4	86	7.2	27	2.4	129	11	100	8.3
London	1,613	25	1.6	50	3.1	22	1.4	97	6.1	188	12
New Toronto	1,285	8	0.7	39	3.0	7	0.6	54	4.3	123	11
Orillia (mental deficients)	1,849	10	0.5	34	1.8	7	0.5	51	2.8	99	5.3
Penetang	624	14	2.2	49	8.0	9	0.8	72	11	54	8.6
Toronto	1,128	10	0.9	74	6.5	5	0.4	89	7.8	67	5.9
Whitby	1,662	27	1.6	50	3.0	23	1.4	100	6.0	84	5.0
Woodstock (epileptics)	592	6	1.2	14	2.3	14	2.2	34	5.7	18	3.0
Tor. Psychiatric (reception)	...	...	...	...	...	...	...	...	...	...	...
Total	13,257	165	1.3	543	4.1	174	1.3	882	6.7	929	7.0

From Table 3, it is to be noted that among 13,257 patients in the Ontario mental hospitals, excluding those already under treatment in the T. B. units, 1.3 per cent showed definitely active pulmonary tuberculosis, 4.1 per cent had pulmonary tuberculosis in which the activity of the lesion could not be determined, and 1.3 per cent showed X-ray shadowing suggestive of tuberculosis. Therefore, a total of 6.7 per cent of these patients presented significant pulmonary lesions requiring isolation. In addition to that number, a further 7.0 per cent showed definitely arrested pulmonary tuberculous lesions.

TABLE 4.

RESULTS OF CHEST X-RAY SURVEY OF PATIENTS IN ONTARIO MENTAL HOSPITALS (1937-38).

(Excluding patients already under treatment in T. B. units at New Toronto and Concord.)

Type of lesion.	Male.	Female.	Both sexes.		
	No.	No.	No.	Per cent of significant lesions.	Per cent of total population.
Active tuberculosis (advised for transfer for treatment). Pleurisy with effusion	8	5	13	1.5	0.1
Minimal	31	20	41	4.7	0.3
Moderately advanced	44	35	79	8.9	0.6
Advanced	15	7	22	2.5	0.2
Total active	98	67	165	19	1.3
Suspect tuberculosis or activity undetermined (lesions would not be greater than minimal). (Segregation and investigation at hospital)	389	328	717	81	5.4
Total showing significant pulmonary lesions (requiring isolation)	487	395	882	100	6.7
Total considered:	6,777	6,480	13,257	...	100

From Table 4, it is to be noted that since practically all the "suspect tuberculosis" cases and those with "activity undetermined" would be classed as having lesions not greater than minimal, approximately 11.4 per cent of those requiring treatment or isolation or 0.8 per cent of the total number of patients had moderately or far advanced pulmonary tuberculosis.

TABLE 5.

RESULTS OF CHEST X-RAY SURVEY OF PATIENTS IN ONTARIO MENTAL HOSPITALS (1937-38).

(Excluding patients already under treatment in T. B. units at New Toronto and Concord.)

CHEST FINDINGS ACCORDING TO MENTAL DIAGNOSIS.

Mental diagnosis.	Total number considered (estimated from annual report of hospitals division).	Active tuberculosis. Recommended for immediate transfer to T. B. unit		Suspect Tbc. or Tbc. activity undetermined. Recommended for segregation pending investigation.		Total showing significant lesions requiring isolation.	
		No.	Per cent.	No.	Per cent.	No.	Per cent.
Schizophrenia	5,903	109	1.8	362	6.1	471	7.9
Manic depressive psychosis	1,250	12	1.0	69	5.5	81	6.5
Psychosis with mental deficiency	601	3	0.5	19	3.2	22	3.7
Senile psychosis	435	2	0.5	30	6.9	32	7.4
G.P.I. and cerebral syphilis	456	7	1.5	26	5.7	33	7.2
Psychosis with cerebral arteriosclerosis	311	1	0.3	17	5.5	18	5.8
Involutional melancholia	261	3	1.2	12	4.6	15	5.8
Paranoia and paranoid state	215	1	0.5	11	5.1	12	5.6
Alcoholic psychosis	83	0	...	1	1.2	1	1.2
Alcoholism without psychosis	21	0	...	1	0.5	1	0.5
Epilepsy without psychosis	468	3	0.6	13	2.8	16	3.4
Epileptic psychosis	415	4	1.0	24	5.8	28	6.8
Mental deficiency without psychosis	2,330	17	0.7	107	4.6	124	5.3
All others	509	3	0.6	25	4.9	28	5.5
Totals	13,257	165	1.3	717	5.4	882	6.7

From Table 5, it is to be noted that patients with schizophrenia showed a slightly higher percentage of significant pulmonary tuberculous lesions than those with any other type of mental illness.

TABLE 6.

RESULTS OF CHEST X-RAY SURVEY OF PATIENTS IN ONTARIO MENTAL HOSPITALS (1937-38).

FINDINGS AT O. H. SCHOOL, ORILLIA—FOR MENTALLY DEFICIENTS

Age group in years.	Total number of cases considered.			Positive tuberculin reactors.				Those showing significant lesions.			
	M.	F.	Both.	M.	F.	Both.		M.	F.	Both.	
						No.	Per cent.			No.	Per cent.
0- 4.....	3	3	6	0	0	0	—	0	0	0	—
5- 9.....	50	34	84	14	1	15	18	1	0	1	1.2
10-14.....	173	118	291	34	16	50	17	1	0	1	0.3
15-19.....	239	196	435	83	51	134	30	4	1	5	1.1
20-24.....	150	160	310	107	101	208	67	2	2	4	1.3
25 and over	336	387	723	...	...	...	..	21	19	40	5.5
Total..	951	898	1849	...	...	...	..	29	22	51	2.8

From Table 6, it is to be noted that the incidence of tuberculous infection among these mentally deficient patients rises quite rapidly after 20 years of age. (The incidence of tuberculous infection among these patients is definitely higher than among school children of corresponding age groups in Ontario.) The great majority of significant pulmonary tuberculous lesions in this institution were found among those patients over 25 years of age.

TABLE 7.

RESULTS OF ROUTINE CHEST X-RAYS UPON ALL NEW PATIENT ADMISSIONS TO ONTARIO MENTAL HOSPITALS.

January 1, 1938, to January 1, 1939.

	Male.		Female.		Both.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Total number considered	1,593	100	1,315	100	2,908	100
Cases of active tuberculosis—recommended for transfer to T.B. unit	7	.4	6	.4	13	.45
Cases of suspect or tuberculosis, activity undetermined—recommended for segregation and investigation at the hospital	36	2.2	24	1.8	60	2.1
Total number with significant chest lesions	43	2.6	30	2.2	73	2.5

From Table 7, it is to be noted that among 2908 patients admitted to the Ontario mental hospitals in the calendar year 1938, a total of 2.5 per cent showed pulmonary tuberculous lesions requiring isolation. (This figure is definitely lower than the 6.7 per cent requiring isolation as found among the resident patient population, suggesting that although tuberculous infection can certainly be brought into mental hospitals by new patients, the resident patient population has offered a greater source of infection.)

TABLE 8.

COMPARISON BETWEEN FINDINGS OF FIRST AND SECOND CHEST X-RAY SURVEYS IN ONTARIO MENTAL HOSPITALS.

(For the five hospitals where second survey has been completed by January, 1939.)

Ontario mental hospitals where second chest X-ray survey had been completed by January, 1939.	New cases showing significant lesions. (Advised for the first time for transfer to the T. B. unit or for segregation at the Hospitals.)										
	1937-38 survey.			1938-39 survey.			Findings in new cases.				
							Active Tbc.				
	Male.	Female.	Both.	Male.	Female.	Both.	Pleurisy with effusion.	Minimal.	Moderately advanced.	Advanced.	Suspect tuberculosis.
Toronto	54	35	89	5	8	13	2	4	1	0	6
Cobourg	0	44	44	0	9	9	1	3	0	0	5
Kingston	87	33	120	13	9	22	2	11	1	0	8
Hamilton	82	44	126	16	16	32	2	16	2	2	10
London	46	51	97	10	24	34	1	16	1	3	13
Total	269	207	476	44	67	110	8	50	5	5	42

From Table 8, it is to be noted that in the second chest X-ray survey already completed at five Ontario mental hospitals, the number of new cases of tuberculosis discovered was 110 in comparison with 476 in the first complete survey. The fact that 10 patients showing normal chest X-rays in the first survey had moderately or far advanced pulmonary tuberculosis one year later is sufficient evidence to indicate the necessity for periodic chest X-ray examinations of the hospital patient population.

TABLE 9.

ESTIMATION OF NUMBER OF BEDS REQUIRED FOR NEW TUBERCULOSIS MENTAL HOSPITAL IN ONTARIO—DETERMINED FROM SITUATION AS IT EXISTED IN JANUARY, 1939.

	Male.	Female.	Both.	
			No.	Per cent of total patient population.
1. Already under treatment in T. B. units	147	156	303	2.2
2. Cases of active tuberculosis awaiting transfer to T. B. units	84	32	116	.8
3. Cases under segregation at hospitals pending investigation.	318	286	604	4.3
4. Estimated number of (3) who will require transfer for treatment	159	143	302	2.2
5. Therefore, total number of beds required (1) + (2) + (4)	390	331	721	5.2

From Table 9, it is to be noted that approximately 5.2 per cent of the total mental hospital bed capacity will probably be required to accommodate, segregate and treat tuberculous mentally ill persons in Ontario. (Estimation of future requirements is quite difficult, however, as it is not possible to determine accurately the effect of the relatively recently commenced complete diagnostic program upon the future incidence of this disease.) The figures presented in this table do not include provision for the treatment of non-pulmonary forms of tuberculosis which types, although not large in number in Ontario and not usually infectious, would preferably be treated in such a tuberculosis mental hospital.

TABLE 10.

CLASSIFICATION OF PATIENTS ON ADMISSION TO T. B. UNIT FOR TREATMENT.
(According to period admitted and extent of tuberculous disease.)
(June, 1933 to June, 1938.)

Year ending.	From sanatoria or homes.	From Ontario Mental Hospitals.																		
		Total.	Advanced.				Moderately advanced.				Minimal.				Pleurisy with effusion and non-pulmonary.			Non-tuberculous.		
			M.	F.	Both.		M.	F.	Both.		M.	F.	Both.		M.	F.	Both.	M.	F.	Both.
					No.	Per cent of total.			No.	Per cent of total.			No.	Prr cent of total.						
June, 1934	11	88	4	10	14	16	18	37	55	62	7	7	14	16	2	1	3	0	2	2
June, 1935	9	87	11	5	16	18	25	21	46	53	8	13	21	24	2	1	3	0	1	1
June, 1936	19	96	12	6	18	19	29	23	52	54	12	11	23	24	1	1	2	0	1	1
June, 1937	14	155	19	11	30	19	38	33	71	46	24	18	42	28	5	1	6	5	1	6
June, 1938	8	131	7	7	14	11	30	34	64	49	34	12	46	35	0	3	3	2	2	4
Totals	61	557	53	39	92	16	140	148	288	52	85	61	146	26	10	7	17	7	7	14

From Table 10, it is to be noted that among the 557 patients from other Ontario mental hospitals admitted to the T. B. unit from June, 1933, to June, 1938, 16 per cent had advanced, 52 per cent moderately advanced, and 26 per cent minimal pulmonary tuberculosis. (Admissions to tuberculosis sanatoria in Ontario show a much higher percentage of advanced lesions.) During the last year (1938) a higher percentage of cases with minimal lesions is observed. The non-tuberculous cases include several admitted mainly for investigation, and also a number where the initial diagnosis based on the X-ray appearance was in error (*e. g.*, upper lobar pneumonia, non-tuberculous broncho-pneumonia, etc.).

TABLE 11.

CLASSIFICATION OF PATIENTS FROM ONTARIO MENTAL HOSPITALS ON ADMISSION TO T. B. UNIT FOR TREATMENT (JUNE, 1933 TO JUNE, 1938.)

(According to mental diagnosis, average age, and years in hospital.)

	No.			Average age in years when tuberculosis diagnosed.			Average number of years in hospital before diagnosis of tuberculosis.		
	M.	F.	Both.	M.	F.	Both.	M.	F.	Both.
Schizophrenia	194	158	352	41	46	43	9	12	10
Manic-depressive psychosis	22	19	41	50	50	50	7	6	6
Psychosis with mental deficiency	9	11	20	33	37	33	4	5	5
Senile psychosis	7	6	13	63	69	66	3	5	4
G. P. I. and cerebral syphilis	12	4	16	44	40	43	4	2	3
Psychosis with cerebral arteriosclerosis	1	6	7	..	66	66	..	3	4
Involutional melancholia	1	6	7	..	60	60	..	7	7
Paranoia and paranoid states	4	5	9	52	55	54	2	8	6
Alcoholic psychosis	1	0	1	..	..	..	..	..	..
Alcoholism without psychosis	0	0	0	..	..	..	..	..	..
Epilepsy without psychosis	1	3	4	..	30	30	..	4	5
Epileptic psychosis	4	8	12	37	38	38	8	8	8
Mental deficiency without psychosis	27	26	53	28	31	30	9	6	8
All others	12	10	22	47	40	44	2	5	4
Total	295	262	557	41	45	43	8	10	9

From Table 11, it is to be noted that the average age at which tuberculosis was diagnosed in these mental patients was over 40 years. It is also to be noted that the average length of time in hospital was 9 years before tuberculosis was diagnosed.

TABLE 12.

PRESENT CONDITION (AS OF JANUARY, 1939) OF PATIENTS FROM ONTARIO MENTAL HOSPITALS ADMITTED TO T. B. UNIT FOR TREATMENT.

(According to consecutive groups of 100 patients admitted.)

Commencing June 1/33	Date of end of period.	Died.	Still or again under treatment in T. B. unit.	Returned to Ontario Hospital (chest disease arrested).	Sent to sanatorium (mental illness improved).	Sent home (both chest and mental illness improved).
First 100 admissions.....	July 4/34.....	56	16	19	4	5
Second 100 admissions.....	July 11/35.....	57	27	10	3	3
Third 100 admissions.....	May 20/36.....	55	33	5	5	2
Fourth 100 admissions.....	March 25/37.....	35	51	8	4	2
Fifth 100 admissions.....	August 26/37.....	23	67	5	4	1
500 admissions ending.....	August 26/37.....	226	194	47	20	13
					80	

From Table 12, it is to be noted that of 500 patients admitted to the T. B. unit from June, 1933, to August, 1937, 226 or approximately 45 per cent were dead by January, 1939. During the same period, 80, or 16 per cent, had been discharged from the T. B. unit.

TABLE 13.

RESULTS OF CHEST X-RAY SURVEY OF STAFF EMPLOYED IN ONTARIO MENTAL HOSPITALS (1937-38).

(Excluding those already under treatment.)

CLASSIFIED ACCORDING TO OCCUPATION AND FINDINGS.

Types of occupation.	Total number considered	Active tuberculosis (requiring treatment in sanatorium).		Tuberculosis, arrested (requiring only repeat X-rays while working).		Tuberculous infection but no evidence of tuberculous disease.		No tuberculous infection. Negative tuberculin reactors.	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Graduate nurses	626	3	0.5	19	3.0	492	79	112	18
Undergraduate nurses	186	3	1.6	1	0.5	44	24	138	74
Attendants	784	8	1.0	43	5.4	635	81	98	13
Nurse attendants	48	0	...	3	6.2	29	61	16	33
Cooks, domestics, etc.	383	0	...	3	0.8	303	79	77	20
Office Staff, etc., physicians	132	0	...	13	9.9	90	68	29	22
Artisans	175	0	...	12	6.8	148	85	15	8.6
All others	208	0	...	6	2.9	160	77	42	20
Total	2,542	14	.6	100	3.9	1,901	74	527	21

From Table 13, it is to be noted that among 2542 members of staff in the Ontario mental hospital (excluding those already under treatment for tuberculosis), 0.6 per cent showed evidence of active pulmonary tuberculosis, while an additional 3.9 per cent showed inactive (arrested) lesions. Of the total number of employees, 21 per cent had negative tuberculin reactions, while among the undergraduate nurses, 74 per cent were negative to tuberculin.

TABLE 14.

CLASSIFICATION OF STAFF FROM ONTARIO MENTAL HOSPITALS REQUIRING TREATMENT FOR THE FIRST TIME FOR TUBERCULOSIS (FROM JUNE 1933 TO JUNE 1938).

(Classified according to occupation, age, years of hospital work and tuberculin reaction on entering service.)

Occupation.	No.	Average age at time of diagnosis.	Average years of hospital work before diagnosis.	Tuberculin reaction on entering service.		
				Not known.	Known.	
					Positive.	Negative.
Graduate nurses	24	26	5.7	24	..	..
Undergraduate nurses	20	21	1.5	2	2	16
Attendants	14	37	9.4	11	1	2
Nurse attendants	0	..	...	..	..	..
Cooks, domestics, etc.	2	34	5.0	1	1	0
Office staff, etc.	0	..	...	..	..	..
Artisans	0	..	...	..	..	..
All others	0	..	...	..	..	..
Total	60	27	5.1	38	4	18

From Table 14, it is to be noted that of the 60 employees requiring treatment for tuberculosis, all but two were nurses or attendants. In the case of these two members of staff (one cook and one domestic) it was ascertained that they had been in contact with previously undiagnosed cases of tuberculosis among patients. Of the 60 employees requiring treatment, the tuberculin reaction on entering their employment is known for only 22 cases (the remainder had entered the service before the routine testing of all new employees) but of these 22 cases, the intitial tuberculin reaction was negative in 18.

TABLE 15.

CLASSIFICATION OF STAFF FROM ONTARIO MENTAL HOSPITALS REQUIRING TREATMENT FOR THE FIRST TIME FOR TUBERCULOSIS.

(According to year of diagnosis and extent of disease.)

Period.	Advanced.		Moderately advanced.		Minimal.		Pleurisy with effusion.		Miliary.		Erythema nodosum.		Total.		Totals.
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
June, 1933, to June, 1934...	1	2	1	4	0	6	0	2	0	0	0	0	2	14	16
June, 1934, to June, 1935...	0	0	2	2	0	3	0	1	0	0	0	0	2	6	8
June, 1935, to June, 1936...	0	0	1	0	0	4	0	3	0	0	0	0	1	7	8
June, 1936, to June, 1937...	1	0	1	3	1	5	0	2	0	1	0	1	3	12	15
June, 1937, to June, 1938...	0	0	1	1	6	5	0	0	0	0	0	0	7	6	13
Total.....	2	2	6	10	7	23	0	8	0	1	0	1	15	45	60
Totals....	4		16		30		8		1		1		60		
Per cent of total number.....	7		27		50		14		..		..		100		

From Table 15, it is to be noted that of the 60 cases among the staff requiring treatment for tuberculosis, only four had far advanced disease, and three of this number were discovered in the first year of this five-year period. Of the total number requiring treatment, only one-third had moderately advanced or a greater amount of disease, a condition not found among admissions to sanatoria where approximately 80 per cent have more than minimal disease.

TABLE 16.

PRESENT CONDITION (AS OF JANUARY, 1939) OF STAFF FROM ONTARIO MENTAL HOSPITALS WHO HAVE REQUIRED TREATMENT FOR TUBERCULOSIS.

Period of first admission for treatment.	Total number of cases.	Dead.	Still or again under treatment.	Convalescent at home.	Working.		
					In Ontario hospitals.	Elsewhere.	Total.
June, 1933, to June, 1934..	16	4	2	1	4	5	9
June, 1934, to June, 1935..	8	0	2	1	4	1	5
June, 1935, to June, 1936..	8	0	0	1	7	0	7
June, 1936, to June, 1937..	15	2	6	3	2	2	4
June, 1937, to June, 1938..	13	0	4	5	4	0	4
Total.....	60	6	14	11	21	8	29

From Table 16, it is to be noted that of the six deaths which have occurred among the 60 employees requiring treatment for tuberculosis, four occurred among those diagnosed with advanced tuberculosis in the first year of this five-year period.

TABLE 17.

RESULTS OF ROUTINE CHEST X-RAYS UPON ALL STAFF APPLICANTS OR NEW STAFF AT ONTARIO MENTAL HOSPITALS.
January 1, 1938 to January 1, 1939.

	Male.		Female.		Both.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Total number considered	204	100	635	100	839	100
Number with active chest disease (not accepted)	3	1.5	11	1.7	14	1.7
Number with inactive chest disease or X-ray abnormalities (accepted with reservation)	5	2.4	2	0.3	7	.8
Total with some abnormal chest findings	8	3.9	13	2.0	21	2.5
Total showing no abnormality (accepted without reservations)	196	96	622	98	818	97.5

From Table 17, it is to be noted that of 839 apparently healthy individuals applying for work in the Ontario mental hospital service, 1.7 per cent showed X-ray evidence of pulmonary tuberculosis which was considered to be active or possibly active, making them unsuitable for employment. The majority of these cases were recommended for sanatorium treatment or investigation. An additional 0.8 per cent who showed X-ray evidence of inactive tuberculosis or other abnormalities were accepted but were placed on the permanent staff only if subsequent re-examinations were satisfactory.

The experience gained in tuberculosis prevention and treatment in the Ontario mental hospitals would lead to the following remarks:

(1) Consultation with the state or provincial chief tuberculosis officer would seem to be the first step in the development of any program of this type.

(2) Co-operating with this officer, at least a preliminary or tentative plan should be outlined for case-finding and treatment of tuberculosis among patients and staff in the hospital or group of hospitals concerned.

(3) A physician, well trained in the diagnosis of chest disease (including chest X-ray interpretation) as well as the modern methods of tuberculosis treatment, should be appointed to initiate this work. Preferably, this physician should have had some previous experience in mental hospital procedure or should secure such experience before commencing his duties. As the plan advanced, and as further medical assistance in this work was provided, the original chest clinician would supervise the activities of the chest service.

(4) The program of tuberculosis case-finding should include:

(a) Among patients:

(1) X-ray examination of the chest (preferably by X-ray films) of all patients as soon as possible after admission to hospital.

(2) X-ray examination of the chest (preferably by X-ray films) of all patients in the hospital at yearly intervals.

Note.—For the initial and periodic examinations, where desired, preliminary tuberculin tests could be given and only the positive reactors X-rayed.

(b) Among staff:

(1) Chest X-ray films for all staff applicants or all new staff as soon as possible after employment. This procedure should include any applicant or new employee in any capacity.

(2) Tuberculin tests upon all staff applicants or all new staff as soon as possible after employment.

Note.—If desired, and where the tuberculin tests can be be given immediately before or after employment, only the positive reactors need be X-rayed.

(3) Chest X-ray films at yearly intervals for all tuberculin positive staff employed by the hospital.

(4) Repeat tuberculin tests at intervals of at least one year for all staff previously negative to tuberculin.

(5) Chest X-ray films taken immediately and repeated in six months for all previously negative tuberculin reactors who develop positive reactions. After six months, these individuals are treated as are the positive reactor group.

(6) Negative tuberculin reactors among the staff should not be allowed to work in contact with known cases of tuberculosis under treatment or isolation. Members of staff employed in T. B. units should be protected by all possible precautions and they should receive chest X-ray films at least every six months.

(5) The disposal of patients found to have evidence of tuberculosis which is not definitely or probably arrested, should include isolation or segregation from non-tuberculous patients. Preferably, all active or possibly active cases of tuberculosis should be placed under one service where special provisions can be made for treatment, investigation, infectious disease technique, etc. Whether such a service would necessitate a tuberculosis mental hospital with separate administration, or merely a tuberculosis unit in connection with an already existing mental hospital, would depend upon the number and size of the hospitals in the group and upon the total number of tuberculous patients to be accommodated.

(6) It should be realized by those in charge of mental hospitals that the combination of tuberculosis and mental illness in patients under treatment necessitates a greater proportion of nursing, medical and ward help staff, than on wards where tuberculosis is not a factor.

General Remarks.

Tuberculosis among patients in mental hospitals constitutes a sufficiently serious problem to demand the best efforts and modern concepts in its control and treatment. Without an adequate system of control, mental hospital patients may act as a reservoir of infection which will spill over into the general population. Certainly, the tuberculosis mortality figures for any state or province are adversely affected by the high mortality rate due to this disease among the mental hospital population.

Overcrowding of patients in mental hospital wards increases the dosage of infection and the number infected by an undiagnosed open case of tuberculosis. It is obvious that overcrowding, insufficient sunlight, and other poor hygienic conditions in mental hospitals should be corrected as part of any campaign against tuberculosis in such hospitals.

It is good medical practice and hospital procedure to make every effort to discover and treat the physical ailments among the patients in mental hospitals. Physical examination or other methods short of X-ray examination of the chest will not detect tuberculosis among the majority of mentally ill patients with even far advanced disease. If it is realized that routine X-ray examina-

tions offer the only eventual solution, delay in instituting this procedure only serves to allow the disease to be spread from patient to patient and too frequently from patient to members of the staff.

The treatment of one individual in sanatorium in Ontario for one year costs the Province approximately one thousand dollars ($1000). The length of sanatorium treatment required for a patient with minimal disease is usually one year, whereas the period necessary for advanced disease may be from three years or more. From these considerations it will be evident that the discovery by routine X-ray film of a lesion at the minimal stage rather than far advanced, in 10 members of staff per year, will probably result in a saving to the Province of approximately twenty thousand dollars ($20,000) annually. This figure would almost equal the entire yearly cost of our diagnostic program among patients as well as staff. Economically, therefore, if for no other reason, state or provincial governments will recognize the necessity for such procedures as have been outlined in this paper.

Conclusions.

(1) The tuberculosis mortality rate for patients in the Ontario mental hospitals has shown some tendency to decrease since 1934, yet in 1936, this rate was 14 times as great as that for the Province as a whole.

(2) From May 1937 to May 1938, a survey by chest X-ray films upon more than 13,000 mental hospital patients in Ontario (excluding 1.7 per cent already under treatment for tuberculosis at the beginning of the survey) revealed:

(a) 1.3 per cent—pulmonary tuberculosis, definitely active—requiring immediate treatment.

(b) 4.1 per cent—pulmonary tuberculosis, activity undetermined—requiring isolation pending investigation.

(c) 1.3 per cent—X-ray shadows strongly suggesting tuberculosis—requiring isolation pending investigation.

6.7 per cent—significant pulmonary lesions—requiring isolation

(d) 7.0 per cent—pulmonary tuberculosis, definitely or probably arrested—requiring only casual observation—not isolated.

13.7 per cent—total showing definite or suggestive evidence of parenchymal pulmonary tuberculosis.

Note.—It is estimated that at least one-half of the patients in groups (b) and (c) will prove to require treatment in a tuberculosis mental hospital or unit.

(3) Approximately 2.5 per cent of 2908 patients admitted to the Ontario mental Hospitals during the calendar year 1938, required isolation because of chest X-ray findings.

(4) A second chest X-ray survey already completed one year after the initial survey at five hospitals with 6000 patients, showed 110 patients to require isolation who had previously shown normal chest X-rays. Of that number, ten had moderately or far advanced pulmonary tuberculosis, indicating the necessity for repeated "combing" by chest X-rays of the patient population.

(5) From the tuberculosis situation as it existed in January 1939 (nine years after commencement of some anti-tuberculosis work, and 1½ years after commencement of the present complete program), it was estimated that a central tuberculosis mental hospital would be required to accommodate approximately 5.2 per cent of the patients in Ontario mental hospitals.

(6) A chest X-ray survey of 2542 staff in the Ontario mental hospitals in 1937-38, showed 0.6 per cent to require treatment for tuberculosis. Since 1933, approximately 0.5 per cent of the employees have required treatment for tuberculosis each year. Of all members of staff, 21 per cent were negative to tuberculin, while among the undergraduate nurses 74 per cent were negative tuberculin reactors.

(7) Of 22 members of staff requiring treatment for tuberculosis where the tuberculin reaction on entering their hospital employment was known, 18 had negative reactions which subsequently became positive, followed (in all these cases within six months) by X-ray evidence of the disease.

(8) Approximately 1.7 per cent of 839 apparently healthy staff applicants or new staff in the Ontario mental hospital service during the calendar year 1938, showed X-ray evidence of pulmonary tuberculosis which was active or possibly active, therefore rendering them not acceptable for employment.

(9) It is believed that an increasing interest will be evident at all hospitals in the control of tuberculosis among their patients and personnel. In mental hospitals this work is particularly advisable. Half-way measures are inadequate to cope satisfactorily with the tuberculosis problem in such hospitals. Only by a complete program of routine case-finding by X-ray examinations and modern

treatment of diagnosed cases, can one feel satisfied that the situation is under irreproachable control.

Note.—Appreciation is gratefully acknowledged to Dr. B. T. McGhie and Dr. G. C. Brink, whose continued interest has been a most important factor in the development of the work upon which this paper is based. Acknowledgment is also made for the assistance of Dr. J. J. Weber, Dr. F. T. Fralich, and Dr. O. V. Dent of the T. B. unit at the Ontario Hospital, New Toronto, who interpreted many of the films and gathered the data for certain tables presented. The superintendents of the Ontario mental hospitals have cooperated in this work and to them and their medical staffs, appreciation is expressed.

BIBLIOGRAPHY.

1. Harrington, A. H.: Separate provisions for tuberculous patients in state hospitals for the insane. Transactions American Medico-Psychological Association, 1900, page 203.
2. Klopp, H. I.: The care of tuberculosis patients in mental hospitals. Am. J. Psychiat., Vol. VI, page 641, 1927.
3. Greene, R. H., and Woodall, C. S.: A survey of tuberculosis in a school for feebleminded. Proceedings of American Association for the study of feebleminded, Vol. XXXVI, page 213, 1931.
4. McGhie, B. T., and Brink, G. C.: A tuberculosis survey in mental hospitals. Am. J. Psychiat., Vol. XIII, page 975, 1934.
5. Fellows, H. H.: A study of incidence of pulmonary tuberculosis. Am. Rev. of Tuberculosis, Vol. XXX, page 109, 1934.
6. Bogen, T., Tietz, E. B., and Grace, F.: Tuberculosis and mental disease. Am. Rev. of Tuberculosis, Vol. XXX, page 351, 1934.
7. Heimbech, J.: Tuberculosis in hospital nurses. Tubercle, Vol. XVIII, page 97, 1936.
8. Burns, H. A.: Incidence of tuberculosis in state institutions in Minnesota. Am. Rev. of Tuberculosis, Vol. XXXIII, page 813, 1936.
9. Harrison, D. A., and Schein, G.: Report of tuberculosis survey at Mercy State Hospital. Psychiatric Quart., Vol. XI, page 637, October, 1937.
10. Myers, J. A., Trach, B., Diehl, H. S., and Boynton, R. E.: Tuberculosis in medical and nursing hospital personnel. Annals of Int. Medicine, Vol. II, page 2181, 1938.
11. Leonidoff, A. A.: Control of tuberculosis in the Hudson River State Hospital. Psychiatric Quart., Vol. XII, page 754, 1938.

DISCUSSION.

Dr. Samuel W. Hamilton (New York, N. Y.).—Dr. Wicks has presented an important program in a careful and detailed way.

In my opinion they are doing an excellent piece of work in Ontario with tuberculosis. When they get the new hospital in Brampton, they will be

able to do something still better. Their present unit is in some regards the most cheerful part of the hospital in which it stands, the reason being that in the tuberculosis unit the patients are not crowded and in the rest of the hospital they are.

When one goes about he finds all sorts of standards of examination for tuberculosis and all sorts of ideas about how much tuberculosis there is in the hospitals. I was in a hospital not so long ago with 1400 patients, in which there were five cases of tuberculosis. I suspect that when and if they make an X-ray survey of their patients, they will find at least another five. There are rather careful studies, as you know, and if instead of a little handful of patients we find somewhere from four to five per cent, we are approximating the experience of hospitals that do the thing well and are therefore probably nearer the truth.

I believe thoroughly in this concentration of the patients. They get better treatment that way. In the metropolitan district about New York, a six hundred-bed unit now under construction will serve all the hospitals. There is a little tendency toward concentration near Boston, though Massachusetts hasn't definitely adopted it as a policy. I have no doubt that Pennsylvania, Ohio, Illinois, California, could well make similar moves.

It is hard to get a man with good X-ray experience for a small unit. It is hard to justify his salary, and when you get him, it is hard to justify the amount of time that he spends on a small unit and he will probably be assigned to other duties also.

The reduction in the death rate in the Ontario hospitals can hardly have been fortuitous. I believe that the improved methods of discovery—that is, of diagnosis and of treatment—must have had a great deal to do with it.

HENRY I. KLOPP, M. D.—Presentation of papers before this organization upon tuberculosis is definitely informative. We need such papers to maintain our interest in the subject and to make us check the activities of the individual institution over which we are the responsible administrative heads. Such papers should also stimulate the members of the respective medical staffs as to the importance of this subject. I have been fortunate in being able to read Dr. Wick's paper in its entirety. Time factor has made it necessary for him to condense it. I derived benefit from reading it due to my presentation of the subject of "The Care of Tuberculous Patients in Mental Hospitals" at the meeting of this Association in June, 1926. The thoroughness of the procedure in the examination of all mental patients connected with the Ontario mental hospitals and of all employees connected with these institutions is deserving of special commendation. I am in accord with the idea of centralizing in one or two institutions the care of mental patients suffering from tuberculosis, particularly if these patients are to be cared for on the institution wards and not segregated in buildings especially designed for them. The only disadvantage that I can see is the distance families are required to travel to visit their relatives. Where the individual institution, however, is of sufficient size to warrant having units designed for this purpose, I am disposed toward having same. We, of course,

cannot lose sight of the fact that in Ontario the men in charge of this work are specially trained and therefore, qualified to carry out the examinations as well as the treatment. It is my opinion, however, that in our hospitals approximating 2000 beds and over, that each institution should have special buildings and the equipment necessary to make thorough examinations and at least one or two physicians should be trained and specialize in the treatment of tuberculous patients.

The technique of X-ray examination of the chest is important. Comparatively slight variation in exposure time or kilovoltage may cause differences in densities on the films with resultant possible difference in interpretation. A marked change in weight between successive roentgenograms without change of exposure factors may lead to incorrect readings. Carefully made roentgenograms taken at intervals and interpreted in conjunction with laboratory, clinical and physical findings offer the best procedure for determining the pulmonary status.

In 1922 and 1923 Dr. Hoffman, Clinical Director of the Allentown State Hospital, made fluoroscopic examinations of practically the entire patient population of the Allentown State Hospital. Comparison of these findings with the reports of physical examinations showed a close agreement between fluoroscopic data and the physical examination reports of one physician. Our present practice is to make roentgen chest studies of patients who manifest marked loss in weight as shown by monthly weight records or show suspicious findings on annual physical re-examinations. A careful check is kept that no one is omitted in the annual physical examination. It is important that flat X-ray films be taken of all suspicious patients to determine whether there is a lesion or not; likewise, if for treatment purposes due to loss in weight, the patient is placed in such a pavilion that, before the patient leaves same, there is an x-ray of the chest as a check-up. We have found that upon a preventorium basis, patients are greatly benefitted by being placed in a section of one of our pavilions for tuberculous patients. They invariably gain in weight and are definitely benefitted thereby.

From experience and statistical check-up we know that patients segregated who have definite or suspicious signs of the disease are benefitted by being placed under special modern treatment and above all; it eliminates the danger of contaminating others.

We also do semi-annual fluoroscopics and make flat chest films of the nursing personnel assigned to the buildings for tuberculosis patients. In 27 years we have had a total of eight nurses and employees develop tuberculosis at Allentown.

A study of a ten-year period from the year 1912 to 1922 showed that of 1200 deaths within this period, 221 died of tuberculosis or 18 per cent. It also shows that there were more women than men, 84 men and 137 women. On the percentage basis the deaths for men were 13 per cent and for the women 26 per cent. The reason for this high rating at that time was that we had 740 transfers from other institutions. A study of the second ten-year period, 1922 to 1932, shows a total number of deaths 1341. Those from tuberculosis 164, or 13 per cent of the deaths. Although the total deaths for

men was higher than for women, those for tuberculosis, the men were 77, women 87 and the percentage for the men 10 per cent, women 16 per cent. However, there was a difference of 10 per cent less between the first ten-year period for women and that of the second. The last five-year period, 1933 to 1938, the total deaths were 506. Of this number 56 were from tuberculosis. Out of the total deaths, 273 for men and 233 for women, tuberculosis accounted for 25 men and 31 women, giving the men nine per cent and the women 13 per cent, the total 11 per cent. A study for all Pennsylvania mental hospitals, eight in number, 1937-38, gave the total deaths 1008, of which 115 were from tuberculosis; 48 men and 67 women, a percentage of eight per cent for men and 16 per cent for women or a total of 11 per cent. The original high percentage at Allentown as already stated was due to transfers from other hospitals during the period before the development of pavilions for men and women tuberculous patients.

It seems to us it would be interesting for Dr. Wicks to study the death rate in proportion to total number under treatment and the number of yearly admissions which we are doing at Allentown. For example total deaths showing the percentage of the number of admissions, the percentage of the daily average in the hospital and the percentage of the total number under treatment. In like manner a study of the number of post-mortem examinations giving the total percentage and that of the deaths due to tuberculosis.

Dr. Irving L. Turow (Peoria, Ill.).—At Peoria State Hospital we have initiated, since October, 1937, a program similar to that which Dr. Wicks described, and I might state that the importance of such a survey can be best exemplified by the fact that in October, 1937, we had in our service 42 patients who were known to have tuberculosis—the population of the Peoria State Hospital is about 2500—and as a result of our tuberculosis survey, at the present time we have about 300 known tuberculosis patients. This has been proven by X-ray examination. In other words, about seven per cent of the patient population has tuberculosis, and again this emphasizes the strong need for a survey such as Dr. Wicks has started.

Dr. Joseph R. Blalock (Southwestern State Hospital, Marion, Va.).—We are now in the midst of a comprehensive survey of our patient population for tuberculosis. We have 1250 patients. Of the approximately 1100 ambulant patients who were X-rayed first there was X-ray evidence of tuberculosis in six per cent of the patients. As soon as a patient is suspected of having an active or questionably active tuberculosis from the X-ray standpoint, he or she is isolated and a more accurate evaluation made on the basis of laboratory and clinical study. We are fortunate in having a modern, well-planned tuberculosis pavilion which will provide adequate facilities for segregation and treatment.

This survey has been most surprising and revealing to our staff. We could, I fear, a year ago, almost have qualified as the hospital to which Dr. Hamilton referred. The cases are to be found if one looks for them. I hope that others may profit by and use as an object lesson one of our

experiences. Two patients who were working in the kitchen were found to have tuberculosis. These, it goes without saying, have been promptly segregated. The survey has so far revealed ten patients in whom pneumothorax treatment is indicated.

The State Department of Health has acted in an advisory capacity and has provided an intensive one-week course in tuberculosis for one of our physicians who will carry on this work. This work, it is planned, to be extended to the other Virginia state hospitals.

DR. WALTER A. THOMPSON (Rockland State Hospital, Orangeburg, N. Y.).—We have just completed a series of fluoroscopic tests of five thousand patients, and have checked with X-ray all those that showed anything positive by fluoroscope, and probably have five per cent who have shown tuberculosis. We are also checking up by X-ray at random, throughout the hospital, patients who did not show anything by fluoroscope. There have been no physical examinations or anything in connection with that—just fluoroscope.

DR. WICKS.—I want to thank the discussants. I appreciate getting their opinions. We would appreciate if those who were doing similar work would write us, so that we could get opinions and exchange ideas.

A NOTE ON THE USELESSNESS OF ANALEPTIC DRUGS COMBINED WITH LUMINAL IN THE TREATMENT OF EPILEPSY.*

By T. T. STONE, I. FINKELMAN, AND A. J. ARIEFF,
Chicago, Ill.

Anticonvulsants such as bromides or phenobarbital used in the treatment of epilepsy, at times cause unpleasant effects such as drowsiness and stupor. This is particularly the case if large doses have to be used in patients with a severe convulsive state. It was thought that the addition to the anticonvulsant medication of analeptic drugs such as coramine, benzedrine, metrazol or ephedrine would prevent drowsiness and also make it possible to increase the dosage of the anticonvulsant drug in patients not rendered free of convulsions with the usual dosage. It was also important to determine whether the use of analeptic drugs would precipitate seizures in patients with remissions.

CORAMINE.

(1) In four patients who were free of convulsions under phenobarbital grains 4½ daily, coramine from 45 to 60 minims did not precipitate convulsions.

(2) In four patients in whom 7½ to 9 grains of phenobarbital daily did not prevent seizures and who were very drowsy, coramine in doses up to 3 drams per day did not diminish the drowsiness.

(3) In seven patients in whom phenobarbital grains 7½ to 9 did not prevent seizures the dosage of phenobarbital was increased to the point of drowsiness. The patients continued to have seizures, however, and the administration of coramine did not prevent drowsiness.

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The Minnie Frances Kleman Memorial Fund.

Benzedrine.

In nine patients under phenobarbital medication, who were, however, not free of seizures, the increase of phenobarbital up to 6 to 9 grains daily did not diminish the number of convulsions but caused considerable drowsiness. The addition of 10 to 30 milligrams of benzedrine daily did not diminish the drowsiness during the day although in some cases it caused sleeplessness at night. In two patients it caused great tremulousness and an apprehensive state. One patient who under bromide medication had only petit mal seizures for many months, upon the addition of 30 mg. of benzedrine daily, had two grand mal seizures. On another occasion, however, benzedrine was administered and no grand mal seizures were precipitated. Cohen and Myerson[1] recently reported the value of benzedrine as an adjuvant to phenobarbital in epilepsy. We are unable to confirm their results.

Metrazol.

Metrazol was used in six patients who were receiving 7½ to 12 grains daily of phenobarbital. There was no change in number of seizures or degree of drowsiness. Metrazol was given in doses of 4½ to 9 grains daily (orally).

Ephedrine.

In two patients ephedrine was used in doses of 1½ grains daily as an adjuvant to the phenobarbital therapy (6 grains daily). There was no change in seizures or drowsiness during the day and one patient had insomnia at night.

Conclusions.

None of the drugs studied (benzedrine, coramine, metrazol or ephedrine) was effective in preventing the drowsiness caused by large doses of phenobarbital or bromides in the treatment of epilepsy.

[1] Cohen, Benjamin and Myerson, Abraham: The Effective Use of Pheno barbital and Benzedrine Sulfate (Amphetamine Sulfate) in Treatment of Epilepsy. Am. J. Psychiat., 95: 371, (Sept.) 1938.

PSYCHOSES RESEMBLING SCHIZOPHRENIA OCCURRING WITH EMOTIONAL STRESS AND ENDING IN RECOVERY.*

By HARRY A. PASKIND, M. D., and MEYER BROWN, M. D.,
Chicago, Ill.

The subject of psychoses occurring with emotional stress and ending in recovery is by no means new, indeed, the first description [1] appeared 85 years ago, and since then there have been many contributions on this question. Our reasons for bringing up this topic at this time are threefold. First, there has been recently a paucity of reports on this subject; in the literature of the last 15 years we have been able to find only four papers dealing with this question. Secondly, we believe that this subject is not unrelated to current psychiatric thought; and, finally, we have observed five cases that might prove interesting to report.

Reversible psychoses occurring with mental stress have been reported mainly in persons recently incarcerated in jails and in soldiers at the front. There have been but few reports of such states in civil life.

Our five patients were seen in the Cook County Jail; they were cases studied in the behavior clinic of the Criminal Court of Cook County.

CASE REPORTS.

Case 1.—An attorney and certified public accountant, aged 35, with a family background devoid of nervous or mental disorder was incarcerated in jail after indictment on charges of obtaining $160,000 in fake fees from clients seeking home loans. Since boyhood he had been excessively boastful, irritable, stubborn, suspicious and given to bouts of depression. In December, 1933, shortly after placement in jail, he began to show marked mental changes. He appeared apathetic, dull, indifferent, became careless of his personal appearance, hardly spoke to the other inmates and if allowed by himself would

* From the department of nervous and mental diseases, Northwestern University Medical School and the behavior clinic of the Criminal Court of Cook County.

Read at a meeting of the Chicago Neurological Society, November 16, 1939.

sit in the same place all day long. He took no part in the activities of the tier and often refused to eat. Questioning elicited a profusion of hallucinations and delusions: At night men come into the room and want to kill him, these men look like lions and out of these lions' bodies come two men and two women who jump at him with knives, revolvers and hatchets. These persons say that they are going to shoot him, to cut his head off, to chop his head. At times, 100 men and 100 women come into the jail to see him at night. Since coming to jail he has made $2,000,000. Two thousand women in Chicago have come into his cell at night in groups of two to ten to have sex relations with him because they have heard of the enormous size of his genitals. He owns 50 buildings, has 10 checking accounts, four wives and half a million diamonds. God talks to him and tells him he is a good boy. During the examination his attention was difficult to hold. He was completely disoriented for time and place, on calculation and general knowledge tests he gave irrelevant responses.

During the examination at intervals of about 30 seconds his head would jerk sharply to the left.

Results of neurological examination were negative. Wassermann test on the blood was negative. For five years previous to admission the patient had diabetes for which he had had practically no treatment.

He was sent to the Illinois Security Hospital, and there recovered over a period of about six months. At the end of that period he began working around the hospital and one year after admission to the Illinois Security Hospital he was returned to the Cook County jail and reexamined in the behavior clinic.

Upon reexamination he showed no disorders of conduct, mood, speech or appearance. He was able to give a detailed coherent history, hallucinations and delusions were absent, he was perfectly oriented and on calculation and general knowledge tests he showed superior ability. He showed interest in tier activities, in his plight and in the future.

He remembered being in jail before; he did not remember being transported to the Illinois Security Hospital, and he had no memory for events that transpired the first few months of his stay there. For the hallucinations and delusions present at the time of the first examination he appeared to have no memory at all.

CASE 2.—A white male, aged 43, had had three previous incarcerations in penal institutions, and one in a state hospital for symptoms that could not be determined.

In December, 1933, shortly after admission to the jail on charges of bigamy and burglary, and without having had mental symptoms immediately preceding, he developed mental changes characterized mainly by delusions of grandeur. Although he was a musician he gave his occupation as that of a prizefighter, and among those that he fought he mentioned Jack Johnson, Dempsey, Tunney, Jeffries, Corbett, Fitzsimmons and John L. Sullivan, and that some of his prizefights were attended by 4,000,000 people. He stated that he was worth $20,000,000,000, had a sackful of diamonds, 100 suits of clothes, a dozen fur coats, 200 hats, 10 cars and owned a bank in California.

He had been arrested 1000 times. He had 20 wives and 1000 children, and was such a famous man that when he rode down the street streamers were thrown out of office windows. He had many sexual delusions. He had the biggest penis in the world, was the greatest sexual intercourse artist in the world, he could ejaculate 40 times in 24 hours, and women stand in lines three to four blocks long in order to have intercourse with him.

He was disoriented for year, month, day. He was well oriented for place. On calculation and general knowledge tests he did very poorly. He was indifferent with regard to his plight, the outcome of the case and his future.

Neurological examination yielded negative results; Wassermann test on the blood was negative.

He was sent to the Illinois Security Hospital where he recovered over a period of a few weeks, and he was reexamined in the behavior clinic in October, 1937. In the second examination he was free of delusions, was calm, quiet, cooperative, able to give a complete and relevant history and was well oriented. He did well on calculation and general knowledge tests. Memory for the events that transpired during the period of the psychosis was very poor and for his delusional thought content he appeared to have no memory whatever.

CASE 3.—A white male, aged 18, without nervous or mental disease in the family background and without significant previous illness, was placed in jail in November, 1933, on a charge of burglary. He had always been quiet, uncommunicative, but prior to incarceration he had shown no changes in behavior or personality. Shortly after placement in jail he began to show marked changes. He became indifferent, taciturn, seclusive, spoke very little to the other inmates, took no part in games or work on the tier. He appeared to have no interest in his predicament or about the outcome of his case. He stated that while alone in his cell at night the other inmates yell at him, call him bad names and come in and punch him. The other inmates want to kill him. The devil talks to him and tells him, "You should die." At night other inmates and the devil parade around his cell. He was well oriented for time and place and gave a poor performance on calculation and general knowledge tests. On the fifth day after admission he made an unsuccessful attempt to hang himself.

Neurological examination yielded negative findings; Wassermann test on the blood was negative.

He was committed to the Illinois Security Hospital in November, 1933, and returned to the behavior clinic for reexamination in July, 1937.

Examination at this time disclosed a patient free of any apparent disorders of conduct, mood, speech or appearance. In January, 1934, two months after commitment he began to improve and recovered shortly afterward. He had no memory for the previous arrest and incarceration in jail, no memory for his stay in jail, nor for his trip to the Illinois Security Hospital, and no memory for the first two months of his stay in that hospital—since then his memory has been good. Hallucinations and delusions were absent, he had no recollection of those that he had at the time of the first examination. Interest in his case seemed to be normal.

CASE 4.—A butcher, aged 41, a steady worker of good habits with unimportant family history and who had had neither significant physical disease nor previous mental disorder, was incarcerated in jail in December, 1933, after a dispute in a picket line. Shortly after incarceration he developed mental changes. He heard voices telling him to pray for God, sometimes these voices threatened to kill him. He was indifferent to his predicament, appeared to have no interest in the prosecution or outcome of his case. He was disoriented in all spheres.

He was allowed to return home and was reexamined in January, 1934, one month after the first examination. Reexamination at this time showed a quiet, orderly, cooperative patient, free of the delusions and hallucinations present during the previous examination, with apparent normal interest in his usual interests.

Memory for his stay in jail and the previous psychotic episode was good and he had insight into the impossibility of his previous ideas.

CASE 5.—A clerk, aged 23, was placed in jail in January, 1934, on a charge of attempted burglary. Although he completed one year of high school, he had had difficulty, he showed little interest in learning and was truant a great deal. He had had no significant previous illnesses and no previous arrests. According to the statements of his family his conduct showed no changes previous to incarceration in jail. Examination in the jail shortly after admission disclosed an indifferent, withdrawn, apathetic, seclusive patient, careless of his personal appearance and who answered questions with a considerable degree of retardation. Voices told him to "get out" and to "come in." The voices make him do things against his will, sometimes they tell him to steal and at other times not to steal. The voices threaten to kill him. Sometimes the voices read his mind and repeat his thoughts. They are the voices of strange men, sometimes they follow him around. The voices make fun of him, call him filthy names. Everybody stares at him as if to hypnotize him. He was well oriented in all spheres and made no attempt to respond to questions involving calculation or general knowledge. He showed no interest in his legal predicament nor in the outcome of his case.

Results of neurological examination were negative; Wassermann test on the blood was negative.

He was sent to the Illinois Security Hospital and returned to the behavior clinic for reexamination in May, 1935. A few months after placement in the Security Hospital the "voices" disappeared, he was put at work in the dining room of the hospital and worked there until his return to the behavior clinic. Reexamination led to the conclusion that this patient had recovered. He showed no disorders of conduct, mood, speech, appearance or manner, was free of the hallucinations and delusions present at the time of the first examination and had insight into their nature. He was examined for the third time in March, 1938. No changes had occurred since the second examination of May, 1935.

The salient features of these cases may be summarized as follows:

The onset in all cases was rapid, and occurred directly after placement in jail.

Attention was markedly affected in all cases; the patients appeared to pay no attention to what was going on about them; frequently it was difficult to obtain and to hold the attention during examination. In some the gaze would be directed away from the examiner and they would stare at the same place for long periods of time.

Hallucinations were present mainly in the auditory and visual spheres; one patient had hallucinations of smell. The auditory hallucinations seemed to possess complete sensory distinctness and were referred to the external world and to persons in the immediate environment. The majority were unpleasant, disturbing, often abusive and threatening; in one instance they repeated the patient's thoughts; in another they gave commands. Hallucinations of vision also seemed to have complete sensory distinctness and were often of a terrifying nature. Hallucinations of taste and touch were absent in all our cases.

Orientation for time and place was absent or disturbed in three of the five cases.

Memory, from superficial observation, seemed to be profoundly affected in four of the five patients. It was difficult to determine if this was an actual defect in memory, or whether there was difficulty in recall, or whether responses were conditioned by indifference, evasiveness, irrelevancy or blocking.

Judgment appeared to be gravely impaired. The presence of hallucinations and delusions indicates this. Further, all patients were unconcerned about their plight, showed no interest in their case nor in the trial. They did not appear to be concerned about their legal representation, nor with the outcome of the case.

Delusions were present in all five cases. Three patients had ideas of persecution; two had ideas of exaltation; two had sexual delusions and ideas of reference were present in one case.

Emotional changes were present in all. Mood changes varied from dullness to profound apathy. None reacted with appropriate mood to their hallucinations and delusions most of which were either alarming or terrifying.

Volitional changes were present in all. Volitional impulses were weakened and independent tendencies for work or recreation appeared to be absent in all. They sat about idle, often in the same place and position for hours. All showed an unkempt appearance.

Disorders of self expression were present in all. They were retarded in answering questions and showed diminution of spontaneous speech, and spoke very little to other inmates or attendants.

Mannerisms were present in one case consisting of frequent rotation of the head.

Amnesia for the period of mental derangement occurred in three of the five patients.

Conspicuously absent were the physical stigmata of hysteria.

It may be seen from the above that this psychosis resembles schizophrenia in several important features; in both there are found defects of attention, hallucinations, delusions, impairment of judgment, dullness, apathy, lessening of volitional impulses, diminution of spontaneous speech and mannerisms. It differs from schizophrenia in the constant presence of emotional stress at the time of onset, the invariably rapid onset, the constant occurrence of recovery, the frequent occurrence of disorientation, the common occurrence of amnesia for the period of psychosis.

It is to be expected that writers on this subject should be preoccupied with the nosological position of such a disorder, and a search of the literature discloses several such discussions.

In 1898 [2] and again in 1904 [3] Ganser reported the cases of 20 prisoners who developed mental changes under the stress of incarceration. Amongst his patients he describes the presence of *"vorbeireden," i. e.*, irrational replies to simple questions, disorientation, hallucinations, anesthesias, complete recovery and amnesia for the period of mental change. Ganser advanced the view that such states are closely related to hysteria.

Raecke,[4] who studied such states in four prisoners agreed that they were manifestations of hysteria. Birnbaum [5] in the study of a single patient came to the same conclusion.

In another communication Birnbaum [6] reported the occurrence of such disorders in soldiers. He stated that such patients resemble persons with schizophrenia as far as the symptoms were concerned, but differed from them in the sudden onset in relation to sudden

emotional shock, the presence of frankly hysterical findings such as anesthesias and functional paralyses and in their rapid improvement upon removal from the source of stress. Birnbaum continued his former view that such states are hysterical. Popper [7] commented that many cases diagnosed as schizophrenia among German soldiers were probably of hysterical origin, or belonged to Ganser's syndrome. As a means of differentiating between these two disorders he offers the observation that in the latter condition there is so much mental confusion that the patient is unable to carry out simple arithmetical problems whereas persons with schizophrenia are usually able to do so but may be unwilling to attempt it.

Risch [8] in recently imprisoned persons described states characterized by confusion, stupor, restlessness, apathy, auditory and visual hallucinations, self-accusatory or paranoid ideas, sleeplessness and irritability. He stated that they closely resemble catatonia and do not belong to hysteria, because these mental states unlike catatonia are recoverable, and unlike hysteria they are devoid of hysterical sensory and motor changes.

Bunse,[9] based on a study of such conditions in soldiers arising after bombardment or other causes of emotional shock, believed that such patients belonged to a distinct group, not hysteria but closely related to it. That this disorder is a separate entity, not hysteria and not schizophrenia has also been emphasized by Wetzel,[10] Wetzel and Wilmanns,[11] Kleist,[12] Bonhoeffer,[13] Sträussler,[14] and Lewin.[15] Their studies were made either on soldiers or on recently jailed prisoners. They called such states shock psychosis, fear psychosis, psychogenic psychosis, and situation psychosis, respectively.

In discussing "prison psychoses" Aschaffenburg [16] took the position that a prison psychosis of psychogenic origin has not been proven to exist as a distinct entity. He stated that dementia præcox is often precipitated by stress and that the majority of the cases of prison psychosis are probably instances of dementia præcox.

Review of the literature, therefore, discloses conflicting views as to the nosological position of these psychoses occurring under emotional stress and ending in recovery, some considered them hysteria, others schizophrenia and still others a disease sui generis.

Before presenting our own views regarding the nosological position of the disorders dealt with here we should like to recall their

resemblance to and differences from schizophrenia. The resemblances may we repeat, consist of defects of attention, hallucinations, delusions, impairment of judgment, dullness, apathy, lessening of volitional impulses, diminution of spontaneous speech and mannerisms. Again, the differences from schizophrenia consist in the invariable presence of emotional stress at the time of onset, the constant rapid onset, invariable recovery, frequent occurrence of disorientation and the common occurrence of amnesia for the period of psychosis.

Any theory advanced regarding the nosological position must explain the presence of these similarities to schizophenia as well as the differences.

It seems to us reasonable to believe that this psychosis resembles schizophrenia because they both involve the same mechanisms,* whatever and wherever these mechanisms may be; and that the differences from schizophrenia (and these are related mainly to the course of the illness) may be related to differences in the etiologic agent—in these psychoses emotional shock—in true schizophrenia some other factor.

Although we are unable to adduce proof in support of such a view, still this viewpoint can be bolstered by innumerable precedents. Thus, it is common knowledge that in any given lesion of the nervous system, the mechanism involved controls the nature of the symptoms, the etiologic agent governs the course of the disease. Thus, for example, a tumor or inflammatory lesion in the same location in, let us say, the spinal cord, will produce symptoms very much alike, the course of these illnesses will be vastly different since the etiologic agents are different.

If this theory is tenable these psychoses occurring under emotional stress and ending in recovery do not belong to the category schizophrenia but belong to an independent nosological entity; they then would constitute a disease sui generis.

It is difficult for us to understand why this psychosis has not been seen or recognized more frequently in civil practice. Surely there can be nothing specific etiologically about bars, bayonets

* No reference is made here to "mental" or psychoanalytic mechanisms in the narrow sense. We refer to the anatomico-physiologico-psychologic substratum of the disease.

and bombardments; these must all be reduced to the common denominator of emotional shock. Certainly, civil life cannot always be devoid of situations free from emotional upheavals. Nevertheless, there are few reports of this psychosis occurring in civil life. We have been able to find but two, Raecke [4] reported one case and Flatau [17] described four cases.

It is difficult for us to understand, why, since intense emotional tension is so common in civil life this psychosis should be so rare. However, it is easy for us to suspect that if they do occur they might be considered cases of schizophrenia, and recoveries from such derangements might be considered recoveries or remissions from schizophrenia.

If this be true, statistics regarding remissions in early cases of schizophrenia with or without treatment are in need of revision on account of the inclusion of this psychosis of perhaps different nosological position and of different and more benign natural course.

CONCLUSIONS.

Five cases are described of a psychosis occurring under emotional stress and ending in recovery.

This psychosis resembles schizophrenia at many points and differs from schizophrenia in some aspects, especially in the course.

As a possible clarification of the nosologic position of this psychosis it is suggested that it may involve the same mechanism as does schizophrenia, but is produced by different etiologic factors: this may explain similar symptoms but differing course, and may indicate that these psychoses deserve a separate nosological designation.

We believe it not unlikely that this psychosis is not uncommonly produced by the stresses of civil life but that such cases are thought to be schizophrenia, and that the recognition of such cases may materially alter statistics regarding remissions of treated and untreated patients with schizophrenia.

BIBLIOGRAPHY.

1. Delbruck: Über die unter Straflingen der Strafanstalt zu Halle beobachteten Geisteskranheiten und ihren Zussamenhang mit dem Verbrechen, Allg. Ztschr. f. Psychiat., 11: 57, 1854.

2. Ganser: Über einen eigenartigen hysterischen Dämmerzustand, Arch. f. Psychiat., 30: 633, 1898.
3. ———: Zur Lehre vom hysterischen Dämmerzustand, Arch. f. Psychiat., 38: 34, 1904.
4. Raecke: Beiträge zur Kentniss des hysterischen Dämmerzustandes, Allg. Ztschr. f. Psychiat., 58: 115, 1901.
5. Birnbaum, K.: Zur Lehre von den degenerierten Wahnbildungen, Allg. Ztschr. f. Psychiat., 66: 19, 1909.
6. ———: Psychopathie und Psychosen, in Lewandowsky's Handbuch der Neurology, Erganzungsband, page 1, Berlin, Julius Springer, 1924.
7. Popper, E.: Zur Differentialdiagnose schizophrener und hysterischer Zustandsbilder, Monatsch. f. Psychiat., 46: 362, 1919.
8. Risch, B.: Über die Verkennung psychogener Symptomkomplexe der frischen Haft und ihre Verwechslung mit Katatonie, Monatschr. f. Psychiat., 25: 291, 1909.
9. Bunse, P.: Die reaktiven Dämmersustände und verwandte Störungen in ihrer Bedeutung als Kriegspsychosen, Ztschr. f. d. ges. Neurol. u. Psychiat., 40: 237, 1918.
10. Wetzel, A.: Das Zustandbild der frischen Schockpsychosen im Felde, Dtsch. med. Wchnschr., 9: 288, 1917.
11. ——— and Wilmanns, K.: Über Schockpsychosen, Ztschr. f. d. ges. Neurol. u. Psychiat., 65: 288, 1921.
12. Kleist, K.: Schreckpsychosen, Allg. Ztschr. f. Psychiat., 74: 432, 1918.
13. Bonhoeffer, K.: Zur Frage der Schreckpsychosen, Monatschr. f. Psychiat., 46: 143, 1919.
14. Sträussler, E.: Über Haftpsychosen und deren Beziehungen zur Art des Beobachtungsmaterials, Ztschr. f. d. ges. Neurol. u. Psychiat., 18: 547, 1913.
15. Lewin, J.: Über Situationspsychosen, Ein Beitrag zu den transitorischen insbesonder haftpsychotischen Störungen, Arch. f. Psychiat., 58: 533, 1917.
16. Aschaffenburg, G.: Degenerationspsychosen und Dementia Præcox bei Kriminellen, Ztschr. f. d. ges. Neurol. u. Psychiat., 14: 83, 1913.
17. Flatau, G.: Über den Ganserschen Symptomenkomplex, Ztschr. f. d. ges. Neurol. u. Psychiat., 15: 122, 1913.

BIOELECTRIC RESPONSES IN METRAZOL AND INSULIN SHOCK.*

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This is the third in a series of papers reporting investigations upon the physiological actions of metrazol and insulin when given in convulsive doses. These studies were prompted by the use of these agents in the therapy of schizophrenia. It is hoped that the information gained from them may add to our knowledge of the action of these agents within the body, and eventually to our insight into the psychological improvement of patients under metrazol and insulin therapy.

It is well known that rhythmical electrical potentials accompany the activity of the brain, and that these potentials may be recorded by appropriate means. They change in response to various stimuli and conditions affecting the central nervous system, and for this reason they afford a useful method of studying some of the central effects of drugs and other agents. The present paper deals with the effects of convulsive doses of metrazol and insulin on the electrical activity of the brain in the unanæsthetized rabbit, and the relation of these bioelectric responses to other physiological phenomena associated with metrazol and insulin shock.

Method.

In those experiments in which the cortex was not exposed the brain potentials were led off by means of silver pellet electrodes (Rheinberger and Jasper)[3] placed on the dura through small burr holes in the skull. When the cortex was exposed, wick-silver tube electrodes (Adrian)[4] were placed directly on its surface. The

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† Research Fellows of the Neuro-Psychiatric Institute of the Hartford Retreat.

electrical activity from four different regions was amplified simultaneously by means of a four-channel, push-pull, condenser-coupled amplifier of the electroencephalographic type, and was then recorded on paper with ink-writing oscillographs.

In metrazol shock the recording of brain potentials may be seriously interfered with by the violent convulsive movements (previously described by Lougheed and Hall [5] and others). In many experiments, therefore, curarization (with artificial respiration) was employed to eliminate movement artefacts. It had previously been noted that curare when used alone did not alter the electrical activity of the brain, nor did it appear to affect the response of the brain to a subsequent injection of metrazol.

Local application of metrazol was made by placing a 3½ mm. dia. disc of filter paper, moistened in a 7 per cent solution of the drug, on any specified area of cortex. Such an application was found to be uniformly effective without producing secondary or repeated high frequency discharges. As there were usually no convulsive movements following local application, it was not found necessary to curarize these animals.

Experimental.

Fifty-nine rabbits were used in the present studies. The experiments were divided into the following main groups:

1. Administration of metrazol intravenously (0.3-0.4 cc. of a 4 per cent solution per kilo. of body weight).

2. Application of metrazol locally in minute quantities to discrete areas of the cerebral cortex.

3. Administration of insulin subcutaneously (3 units per kilo.).

Changes in Brain Potentials During Metrazol Shock.

A—Intravenous Metrazol.

The cortical potentials recorded during a typical metrazol shock showed a characteristic series of changes (Fig. 1). For the purpose of description these changes may be divided into four phases. Each phase has been correlated, in non-curarized animals, with the physical signs simultaneously exhibited.

The latent period coincided closely with the time taken by the metrazol to reach the brain, and averaged 6 seconds in the rabbit.

In a number of experiments dye was injected with the metrazol to determine this point. During this latent period the cortical potentials were usually of low amplitude and somewhat higher than normal frequency, a pattern of cortical activity known as the "startle reaction."

The first phase, following immediately upon this startle reaction and lasting about 10 seconds, was characterized by irregular slow

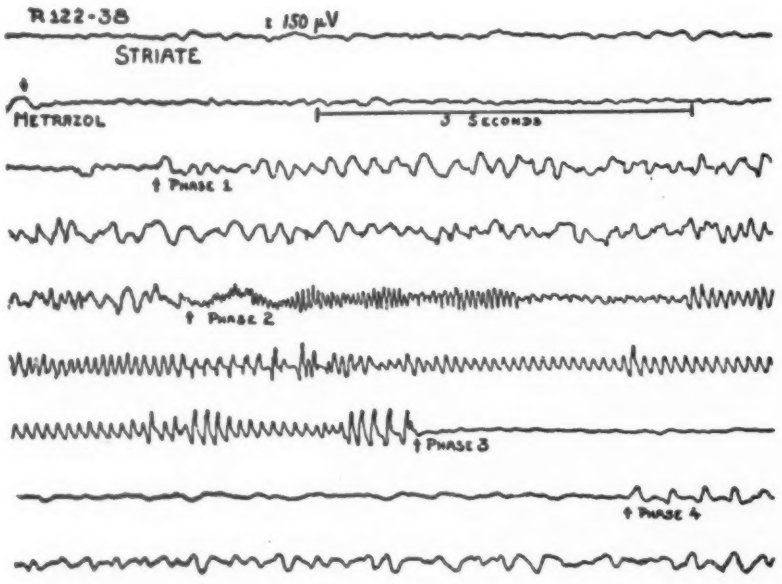


FIG. 1.—Continuous recording of potentials from striate area of rabbit, following intravenous injection of metrazol. The various phases are clearly defined.

waves with occasional spikes. This phase corresponded to the "excited," "first clonic" or "precipitating" stage of the convulsion.

The slow waves of this first phase were replaced in the second phase by a high frequency discharge which commenced with an initial rate of 15-30 per second and then progressively increased in amplitude while it decreased in frequency. This discharge, which coincided with the "tonic" stage of the convulsion, gradually broke up later into small bursts of spike potentials separated by increasing isoelectric intervals. These intermittent bursts cor-

responded to the motor responses of the "clonic" stage of the convulsion. The electrical activity associated with the "tonic" and the "clonic" stages comprised the second phase. It lasted from 30 to 200 seconds.

The clonic discharge then ceased abruptly, leaving the cortex void of all apparent activity (Fig. 2). This stage of profound depression of cortical potentials which ushered in the exhaustion stage of the shock constituted the third phase, which was very variable in duration.

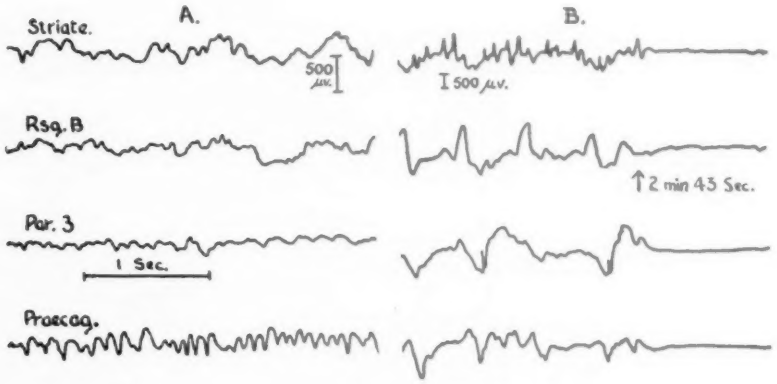


FIG. 2.

A—Simultaneous normal recordings from the striate, retrosplenialis granularis B, parietal 3 and præcentralis agranularis areas of Rose.

B—Same leads, following intravenous injection of metrazol. Figure shows transition from Phase 2 to Phase 3.

The final recovery phase was characterized by an appearance of slow random waves which increased gradually in amplitude and frequency. After 30 minutes or more these merged into the normal pattern of cortical activity. Normal frequencies were at times superimposed on the slow waves during this period of transition.

Variations in the foregoing sequence of events were common, particularly when more than a minimal convulsive dose had been given. These variations took many forms, such as repetition of the second phase in whole or in part (secondary convulsions), or absence of the depressed phase. The time relationships of the various phases also varied widely from one experiment to another.

In order to make an accurate correlation between the cortical discharges and the movements of the animal during the tonic and clonic stages, simultaneous recordings of cortical potentials and of leg movements were taken, the latter being recorded by means of a photocell. The results so obtained showed that the tonic spasm of the animal coincided with the earlier periods of the high frequency discharge, and that the clonic stage appeared when these discharges had commenced to break up into bursts of spikes, each

TABLE I.

CORRELATION BETWEEN THE PHASES OF CORTICAL ACTIVITY AND THE PHYSICAL STAGES OF METRAZOL SHOCK.

Bioelectric phase.	Stage of shock.
LATENT PERIOD.	LATENT PERIOD.
PHASE I: Random slow waves and occasional spikes	"EXCITED," "FIRST CLONIC" or "PRECIPITATING."
PHASE II: (a) High frequency discharge	TONIC STAGE.
(b) Slowed discharge frequency and grouped discharge bursts	CLONIC STAGE.
PHASE III: Extreme cortical depression PHASE IV: Slow waves	"EXHAUSTION" or "RECOVERY" STAGE.

clonic movement eventually being identified with a burst of spike potentials in the cortex (Adrian)[4] (Kornmüller)[7]. (See Table I.)

Further studies were made of the action of intravenous metrazol on the brain by recording from exposed surfaces of cerebrum, thalamus, midbrain and cerebellum, employing the wick type of electrode. (Fig. 3.) The discharges of the brain stem and cerebellum were of smaller amplitude than those of the cerebral cortex, this tendency being noticeable during normal as well as abnormal periods. During metrazol shock the onset, change of phase and cessation of discharge tended to be simultaneous in the regions of the brain under study. In contrast to the relative regularity of the spike potentials in the cortex throughout long periods of the convulsion, the lower centers frequently showed considerable variation in wave form from time to time (*cf.* Korn-

müller). While at first the lower centers and even different areas of the cortex showed considerable independent convulsive activity, towards the end of the convulsion all areas discharged synchronously, the entire brain appearing to act as one unit.

The characteristic synchronicity of discharge in the two cerebral hemispheres tended to be interrupted by a median section through the corpus callosum. Similarly a section isolating an area of cortex neurologically from the remainder of the brain disrupted the

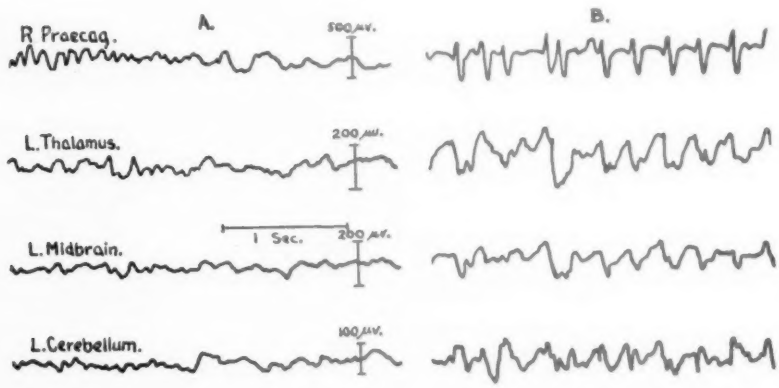


FIG. 3.—Simultaneous monopolar recordings from right cortex (area præcentralis agranularis) and left thalamus, midbrain and cerebellum.
A—Before metrazol. The characteristic 12-15 per second discharge of area præc. agran. may also be seen in thalamic and cerebellar recordings.
B—Following intravenous metrazol. All areas are involved in the abnormal activity.

synchronicity of discharge between the isolated area and other areas of the cortex.

A further series of experiments was performed in an attempt to determine roughly the extent of the central nervous system upon which metrazol exerts its convulsive action. When the brain was separated from the spinal cord by a section through the lower end of the medulla, the same electrical brain response to intravenous metrazol was obtained from the cortex as in the intact animal. In such uncurarized spinal rabbits no convulsive movements were observed, a result which is in agreement with that of Schoen.[8] However, Blume[9] has shown that convulsions can be produced by metrazol in the spinal cat. After a section through

the brain stem just below the inferior colliculi, the animal reacted to metrazol by convulsive movements, and the parts of the brain above the section still showed their typical electrical response. Similar results were obtained following higher sections of the brain stem, and finally, following complete neurological isolation of a portion of the cerebral cortex from the remainder of the brain.

Thus with bioelectric and motor manifestations as criteria, there is conclusive evidence that metrazol acts on cerebral cortex, thalami, midbrain and lower brain stem. Our negative results on the spinal rabbit, while confirming those of Schoen, are inconclusive in that they may be due to the conditions of the experiments. One can at least suggest that the spinal cord is less sensitive to the convulsant action of metrazol than are the higher centers.

B—LOCAL APPLICATION OF METRAZOL TO THE CORTEX.

In order to study the direct effect of metrazol on cortical activity in the absence of the marked systemic physiological and biochemical changes associated with its intravenous administration, minute quantities of the drug were applied directly to the exposed cortex, and the resulting changes in cortical potentials were recorded. A series of 19 rabbits was used in this portion of the work.

The patterns of electrical activity produced by local application differed somewhat although not fundamentally from those induced by intravenous metrazol. Unless the cortex had been sensitized by a previous application, the latent period was approximately twice as long. Only occasionally was the first phase of irregular slow waves present. The second or high frequency phase was indistinguishable from that following intravenous metrazol. The third or depression phase was of frequent but not constant occurrence. The final recovery phase of slow wave activity was of shorter duration, lasting about 15 minutes as compared to the 30 minutes or more following intraveneous metrazol.

The convulsive discharge spread far beyond the limits of diffusion of the locally applied metrazol. The spike-like potentials appearing in distant regions increased in amplitude much more slowly than at the site of application (Fig. 4). These potentials were evidence of facilitation of cortical activity comparable to that due to repetitive cortical stimulation by faradic current

(Adrian)[4]. Facilitation occurred not only within the limits of a cytoarchitectonic area but frequently spread to other regions (for example, from area striata to regio parietalis or regio præcentralis agranularis (Rose)[10] sometimes involving the entire hemisphere or even extending to the opposite side (Fig. 5)). It was found

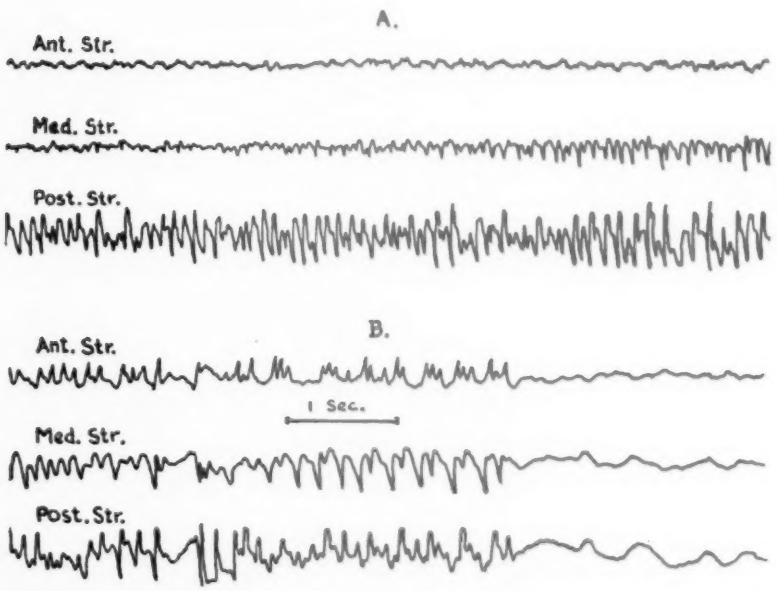


FIG. 4.—Spread of abnormal cortical activity in striate area of rabbit, following local application of minute quantity of metrazol to posterior part of striate area.

A—Approx. 1 min. after application.
B—Approx. 3 min. after application.

Note simultaneous cessation of phase 2 throughout the striatum, and absence of phase 3. Monopolar recording.

that if neurological connections to other areas were severed, the activity did not spread beyond the limits of the isolated area. In "cerveau isolé" preparations (Bremer)[11] in which transections of the brain stem were made at the midbrain or lower medulla, local metrazol applications produced the customary cortical discharges.

These experiments on the isolated brain and on isolated areas of cortex corroborate the findings in similar preparations after intravenous metrazol, which show that metrazol has a local action on

the cortex and does not depend on association or projection pathways for its effect on cortical potentials. Furthermore, it is apparent that this local action of metrazol on the cortex, resembling as it does the effect of direct electrical stimulation, is not in any way dependent on physiological changes occurring elsewhere in the body.

In contrast to the intravenous injection experiments, where all areas of the cortex appeared equally able to participate in the shock

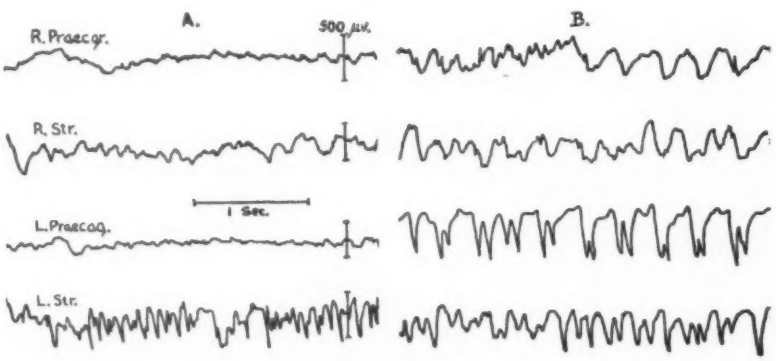


FIG. 5.—Electrical activity following application of minute quantity of metrazol to left striatum of rabbit.

A—20 sec. after application, showing abnormal discharge in L. striate area. B—Showing spread to areas left præcentralis agranularis and to right striate and præcentralis agranularis approximately 30 sec. later. Monopolar recordings.

disturbance, it appeared from the local application experiments that certain areas would respond more readily than others. The sensory cortex (area striata, regio parietalis, etc.) of the rabbit was more sensitive to the action of local metrazol than was the motor cortex (præcentralis agranularis). Applications of metrazol to the thalamus and midbrain on removal of the ipsilateral cerebral hemisphere did not elicit any bioelectric response, nor did injections of small quantities (0.1 cc.) of 10 per cent metrazol into the substance of thalamus, hypothalamus and midbrain. In one experiment, metrazol applied locally to the motor cortex produced unmistakable spike potentials in the thalamus as well. In general, however, only intravenous administration of a larger than minimal

convulsive dose was found to be effective in such preparations. In view of the fact that Kornmüller [7] has induced more or less typical bioelectric responses in the thalamus by injection of metrazol into this portion of the brain, one must assume that all these higher centers may be capable of initiating to a greater or lesser degree the typical response to a convulsive drug.

Cortical Potentials in Insulin Shock.

(This work has been reported briefly in preliminary papers [1, 2])

The study of insulin shock in the rabbit by means of the cortical potentials was not as productive as in the case of metrazol. As far as could be determined insulin has no direct action on the brain potentials; its effect is apparently an indirect one associated with the systemic hypoglycemic response. Furthermore, the bioelectric changes in the cortex are not specific for insulin. They resemble, and indeed may be identical with the responses to many other agencies which cause a decrease in functional activity.

Insulin was administered subcutaneously to rabbits which had fasted for 24 hours. The dosage was 3 units of insulin per kilo. of body weight. The effect on the cortical potentials was to cause a gradual increase in the slow wave activity as the hypoglycemia progressed. Slow wave activity was well established when the blood sugar (true glucose)* had decreased to approximately 50 mg. per cent. Occasionally, short bursts of localized low potential spikes were recorded from the animal's cortex, during this pre-convulsive period.

Onset of the actual convulsion was not preceded by any significant cortical potential changes similar to those seen, for example, preceding a clinical Jacksonian epileptic attack, or a metrazol convulsion. During the convulsion the recordings appeared to be almost entirely artefactual. It was found that curare could not be used to immobilize the preparations during the convulsion stage. This drug inhibited the hypoglycemic action of insulin, causing an increase in the blood sugar level.

Immediately following the convulsion there was a short period of depression of cortical potentials, usually lasting less than a

* Determined on an 0.3 cc. blood sample by the method of Herbert and Bourne, using a modified Shafer-Hartman reagent (Harding and Downs).

minute. Slow waves similar to those seen prior to the convulsion then appeared, persisting until another convulsion took place. This cycle of events could recur several times following a single injection of insulin. This we attributed to variations in the blood sugar level which we observed, due probably to a release of glucose from the liver following the sympathetic activity associated with the convulsions. If at any time sufficient glucose were injected intravenously to raise the blood sugar to above 50 mg. per cent, the abnormal waves were replaced by normal frequencies as long as that level of blood sugar was maintained.

The studies made to date on the bioelectric activity of the brain associated with insulin shock are very incomplete. Further investigations must be undertaken to determine the reaction of the various parts of the central nervous system to insulin hypoglycemia, especially during the period of convulsive activity. As is apparent, difficulties are inherent in the study of the effects of insulin hypoglycemia on the brain in the absence of systemic changes, a procedure which is possible with metrazol by means of local application.

Discussion.

The bioelectric responses of the brain to metrazol are very similar to those resulting from experimental neuronal stimulation by other convulsant drugs, or by faradic current. Ordinarily such bioelectric activity occurs only in certain pathological stages, such as grand mal epileptic attacks. However, the study of such activity is not without importance, since, as Adrian [4] observes, "the (abnormal electrical) reactions illustrate properties of the cortical neurones which may play some part in the normal working of the brain and must certainly do so in pathological conditions." If the abnormal electrical potential patterns resulting from metrazol reflect the nature of the cerebral response just as they do under other experimental conditions, then an interpretation of these responses may be attempted in the light of earlier studies of normal and abnormal neuronal activity.

According to Hildebrandt,[12] metrazol is classified with the central convulsant drugs. Although a camphor derivative, it bears but slight pharmacological relationship to camphor, since it has no central or peripheral paralyzing action. In this respect it re-

sembles picrotoxin and, like it, can produce almost pure excitation. Experimental evidence would indicate that metrazol has a widespread action on the central nervous system, the spinal cord being less sensitive to its convulsant action than the higher centres. Although denied by Hildebrandt, it has been suggested by Dean [6] that afferent impulses enhance its action. The effect of intravenous metrazol might be compared to that of widespread electrical stimulation of the brain.

The significance of the bioelectric responses seen in the various phases of metrazol shock can best be discussed phase by phase. The "startle" reaction, which appears before the drug has reached the brain, is not a specific response to metrazol; it is due to handling of the animal and may result from frightening or startling by any means. It is comparable to suppression of alpha activity in the human by afferent stimulation, and is probably a desynchronization of neuronal activity.

Phase I.—This phase corresponds to the precipitating stage of the physical metrazol convulsion. It commences with the arrival of the metrazol at the brain. The mode of production of the irregular slow waves occurring in this phase is uncertain, but they may be electrotonic swings of potential resulting from a preconvulsive action of the drug.[14] The random small and large spikes frequently superimposed on the slow waves suggest that synchronous discharge of small groups of neurones has already commenced before summation and facilitation have had time to produce the widespread and orderly synchronicity of the second phase.

Phase II.—This coincides with the tonic and later with the clonic stages of the physical convulsion. In the preceding phase priming by summation has, no doubt begun on an extensive scale, so that now in this second phase the neurones fire off repeatedly in unison. The fundamental processes involved are more evident in records taken when metrazol is applied locally to limited areas of cortex. Due to facilitation of neighboring neurones the discharge occurs in increasingly large numbers of cells. The activity of the neighboring neurones in turn influences that in the initiating area, resulting in great regularity and synchronicity of the spike potentials. In such records the spread of the activity to distant areas, and the slow regular increment in the amplitude of the

spikes can easily be seen (Fig. 4). These phenomena appear to be identical with those which occur as a result of local faradization of the cortex (*vide* Adrian). With intravenous metrazol a spreading facilitation is not so apparent, since the drug can act on all parts of the central nervous system almost at once, although evidence of facilitation can usually be recognized at any given point. Later in the second phase the spike potentials tend to reduce in frequency and finally to break up into isolated bursts, which resemble strikingly the after-discharges seen following cessation of faradic stimulation of the cortex. Adrian stresses the importance of the previous repetitive activity of the neurones as being a major factor in the formation of these highly synchronous discharges. The quiescent periods between the bursts appear to be evidence of a depression of excitability, such as is known to occur following repetitive stimulation of neurones (Fulton).[13] As would be expected following intravenous convulsants, this depression of excitability (seen in our records as an isoelectric period) is widespread. When it passes off, the first activity at any point fires off large areas of cortex. Such alternate periods of depression and of large spike potentials frequently recur with great regularity.

Despite the similarity of the responses, there exists a fundamental difference between electrical and chemical stimulation of the brain. When the former is employed the stimulus may be withdrawn at any time, although reaction may persist as an after-discharge following this withdrawal. On the other hand, a convulsant drug cannot be withdrawn, but rather must remain in some unknown (albeit undoubtedly reduced) concentration in the perineuronal tissues even after cessation of convulsive activity. Just how its continued presence influences the duration and character of the various phases is not yet known, but it may be assumed that it does so to some degree. The occasional appearance of secondary or repeated shocks even after several minutes of depression is probably due in part to residual metrazol in the brain tissue.

Phase III.—With final disappearance of the large spike potentials there usually appears a brief stage of depressed activity, probably due to depressed excitability. This constitutes the third phase,

and corresponds to the early part of the rest or exhaustion stage of the physical shock.

Phase IV.—Gradually the slow waves of the fourth phase appear. Their mode of production is uncertain, although they may be electrotonic in nature. The slow waves present during sleep, ether anesthesia, hypoglycemia or following compression of cortical tissue are very similar, and are thought to be indicative of depressed neurones or cells which can do no useful work (Walter).[14] Before the period of slow waves is over, evidence of returning normal function of some neurones is apparent. Normal frequencies are occasionally seen in this transition period, superimposed on the slow waves. It seems probable that the normal frequencies are produced by cells which had contributed earlier to the slow wave activity, but have recovered at a slightly more rapid rate.

Although the administration of metrazol causes profound physiological and biochemical changes in the body, as was shown in the studies of Lougheed and Hall, the bioelectric responses of the brain do not depend primarily on these changes. These bioelectric responses retain their characteristic form in the curarized preparation, where the chemical state of the blood tends to remain more constant during the shock, and also in the case of local irritation of the cortex by metrazol, when systemic changes cannot be observed. It would seem, then, that the somatic and visceral changes which follow intravenous metrazol in the intact animal are the result, rather than the cause, of the brain's abnormal activity. Just as the convulsive movements are somatic manifestations of the cerebral activity, so the associated visceral changes may be but autonomic manifestations of the same phenomenon, due to direct or indirect involvement of central autonomic mechanisms. One may assume that these visceral and other changes modify the bioelectric responses of the brain to some extent, even if the method of such interaction is not yet known.

The experiments of Leibel and Hall[2] show that the so-called autonomic drugs exert a profound effect on the action of metrazol. For example, adrenalin, in pharmacological doses, not only lowers the convulsive threshold of the central nervous system to the drug, but also converts a normal convulsive dose into a fatal one. On the other hand atropinization, while it alters the convulsive

threshold very little, permits injection of normally lethal doses into animals without fatal results. Although these experiments might be interpreted to indicate the importance of the autonomic system in metrazol shock, it is not yet clear what rôle it plays either in the shock itself or in the therapeutic value of the shock treatment (See Gellhorn).[17]

In spite of the fact that the brain can exhibit but a limited number of bioelectric responses to an almost unlimited number of widely different agents, the pharmacological characteristics of any agent do determine to a very great degree the nature of the response. Insulin is widely different in its action from metrazol. The former produces hypoglycemia (in contrast to the hyperglycemia due to metrazol) and this apparently deprives the brain of fuel for its normal oxidative processes. The convulsant action of metrazol, on the other hand, probably raises the metabolic needs of the neurones far beyond their possible immediate fulfilment. While these are two fundamentally different methods of interfering with neuronal metabolism, it is of possible interest that both methods eventually produce neuronal depression, with its attendant slow wave activity. In the former case it may be a direct result of hypoglycemia, whereas in the latter it is probably an end-result of the intense neuronal discharge.

The site of origin of the convulsive activity which frequently occurs in insulin shock has not yet been definitely established. The intense cortical activity during metrazol shock would indicate that here the cortex is an important factor in causing a typical seizure. With insulin, however, there is no unusual cortical activity immediately before a convulsion, other than the slow waves which one associates with neuronal depression or coma. It must be admitted that the entire brain is in a uniformly low sugar environment, and that under these conditions it might explode into abnormal activity in a widespread manner. In this event a convulsion might not be preceded by significant cortical frequencies. There is a possibility, however, that the insulin convulsion might be caused by a purely sub-cortical disturbance. Experimental and clinical evidence of other workers would tend to bear this out.[18] It is known that these lower centres may initiate a convulsion, as when metrazol is injected into a thalamic preparation. If this is the case, the cortex need take no part in the actual

convulsion except to the extent that it is no longer in a condition to exert an inhibiting influence on the lower centres. We believe that this aspect of the problem warrants future study.

Summary and Conclusions.

(1) The bioelectric potentials from the brain of the rabbit have been studied during metrazol and insulin shock. Insulin, administered subcutaneously, causes a marked increase in the slow wave activity during the coma stage. Cortical potentials during the convulsions are masked by the potential artefacts caused by the violent movements. Except for a few cases of localized spike potentials recorded from the cortex during the pre-convulsive stage, no characteristic cortical potentials are seen immediately preceding the convulsion.

(2) The potentials recorded from the cortex following a shock injection of metrazol may be divided into four phases. These phases are correlated with the various stages of the physical shock.

(3) Thalamus, midbrain and cerebellum show abnormal electrical activity during metrazol shock.

(4) Sections at various levels in the central nervous system show that the electrical response in the cortex due to the action of metrazol, is not primarily dependent on the severed pathways. Characteristic shock potential sequences may even be recorded from small areas of neurologically isolated cortical tissue. The physical shock, on the other hand, is altered profoundly by these neurological sections.

(5) Minute quantities of metrazol, applied locally to discrete areas of the cortex of the rabbit, initiate a bioelectric response which, although much more localized, resembles closely that seen following intravenous injection. This activity may spread to other cortical or even sub-cortical areas, presumably by facilitation.

(6) An attempt is made to explain something of the action of metrazol on the brain, on the basis of what is already known regarding the effects of faradic stimulation of the cortex.

BIBLIOGRAPHY.

1. Goodwin, J. E., Lloyd, D. P. C., and Hall, G. E.: Proc. Soc. Exp. Biol. and Med., 38: 897, 1938.
2. Hall, G. E.: Am. J. Psychiat., 95: 553, 1938.

3. Rheinberger, M. B., and Jasper, H. H.: Am. J. Physiol., 119: 186, 1937.
4. Adrian, E. D.: J. Physiol., 88: 127, 1937.
5. Lougheed, D. W., and Hall, G. E.: Am. J. Psychiat., 96: 657, 1939.
6. Dean S. R.: J. Conn. State Med. Soc., 2: No. 7, 1938.
7. Kornmüller, A. E.: Fort. Neur. Psychiat., 7: 1-30, 1935.
8. Shoen, R.: Arch. f. exper. Path., 113: 257, 1926.
9. Blume, W.: (Cited by Hildebrandt.)
10. Rose, M.: Jour. f. Psychol. u. Neurol., 43: 353, 1931.
11. Bremer, F.: C. R. Soc. Biol., Paris, 118: 1235, 1935.
12. Hildebrandt, F.: Handbuch der experimentale Pharmakologie, 5: 151-183, 1937. (Edit. A. Heffter-Publ., Julius Springer, Berlin, 1937.)
13. Fulton, J. F.: Physiology of the nervous system. Oxford University Press, 1938.
14. Walter, W. G.: J. Neurol. Psychiat., London, 1: 358, 1938.
15. Penfield, W., von Santha, K., and Cipriani, A.: J. Neurophysiol., 2: 257, 1939.
16. Dubner, H. H., and Gerard, R. W.: J. Neurophysiol., 2: 142, 1939.
17. Gellhorn, E.: Arch. Neurol. Psychiat., Chicago, 40: 125, 1938.
18. Parfitt, D. N.: Proc. Roy. Soc. Med., 31: 137, 1937.

THE EFFECT OF METRAZOL ADMINISTRATION ON PROLONGED REFUSAL OF FOOD IN THE PSYCHOSES.*

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At the present stage of metrazol therapy its modus operandi is still essentially unknown. Von Meduna believes [1] "it is possible to theorize that the medullary irritation with resultant respiratory, vasomotor and autonomic responses is the basis of the metrazol convulsive therapy." However, in the same article another aspect of the treatment is mentioned: "The anxiety of the patient against the treatment is frequently mentioned by various workers; the reasons are numerous. As inner causes there are the feelings of impending death and sudden annihilation during the phase of the aura." And although Von Meduna and Friedman [1] feel that such expressions as "to frighten a patient to his senses" or "to scare the devil out of him," ought not to be employed in scientific discussions" of the action of metrazol, they are not in complete accord with many workers who are attempting to eliminate the significance of the aura, admitting that "it is not known what particular phase of this therapeutic convulsive syndrome plays the important rôle in producing remissive changes in the psychotic organism. It is not altogether excluded that this anxiety and fear might possibly be just as important as other phases of the convulsion. In line with this are the experiences of one of us with camphor routine. Beneficial effects with this drug have been noted without actual motor phenomena and with the predominant features of anxiety mounting to panic." And again: "psychiatrists have repeatedly stated that if a patient is threatened with death and annihilation, all the 'imaginary' symptoms will disappear."

In my own experience with this method of treatment, I was greatly impressed with the features of fear, anxiety and apprehension in a great number of my patients. Arguing against the treatment, begging to be excused, hiding under beds and in closets, threatening and actual physical resistance were common, and in

* From the Spring Grove Hospital, Catonsville, Md.

most instances fear was given as the reason. We recorded the pulse of the patients both while on the ward, not anticipating the treatment, and later when they saw the preparations for the injection, and a rapid rise ranging from 10 to 32 beats per minute was often noted. Several of my patients promised to go to work and do other things they had refused to do, and to accede to certain prohibitions if the treatment were discontinued. A schizophrenic, mute for several years, talked every time he was taken to the treatment room, begging and promising to talk if the treatment was omitted, only to lapse into mutism when back on the ward. From these experiences originated the idea of inducing or compelling self-starving patients to break their fast and resume eating through the administration of metrazol.

CASE REPORTS.

CASE 1.—A. S., age 53, cashier, married, admitted November 27, 1935, after eight months in a private sanitarium. He was agitated, depressed, unable to work, cried a great deal, thought that other men were better than he; he was impotent during the past year, could not sleep, heard his wife's and daughter's voices; he was indecisive, poorly nourished with food phobia and could not "understand this eating business." He believed he offended or harmed his family or the physician or sinned when he ate, and refused all food for long periods, necessitating tube feeding, but at times he would steal food when no one was looking. He made several serious attempts at suicide. He was given a course of insulin in June, 1937, with no improvement. In February, 1938, a course of antuitrin-S was given intramuscularly with no improvement. Patient refused to eat for two months, and we began the administration of metrazol in May, 1938, with 4 c.c., completing it on July 2, 1938, with 6½ c.c., obtaining one slight and nine severe reactions. After the first reaction the patient remained in bed with backache for over a week, he began to eat, cleared up mentally and was paroled. He has a clear insight into his illness, has secured a position, and can be considered as completely recovered. He has been wearing a corset for the past year and an X-ray a year after treatment shows a compression fracture of the eighth thoracic vertebra. For lack of space I shall not give the results of our orthopedic and neurologic examination which I shall present fully in another communication. Our original aim in giving metrazol to this patient was to break his fast and induce him to resume eating, and this proved successful.

CASE 2.—F. C., age 23, single, high school graduate, farmer, admitted May 21, 1938, when he was picked up in a confused state with a quantity of silverware in his possession, and could not give an account of himself except to say that he was from South Carolina. It was later learned that he had been a patient in a South Carolina State Hospital for a year in 1932.

On admission he was disoriented, negativistic, grimacing, seclusive, uncommunicative, mute for long periods. He refused food, was spoon-fed with difficulty for some time, and it became necessary to start tube feedings July 8, 1938. He lost weight rapidly. On August 20 we began metrazol treatment, ending September 5, obtaining five reactions in 12 treatments. The patient struggled each time against the administration of the drug, and finally he promised to eat if the treatment were discontinued. However, it was necessary for the first several days thereafter for the physician to come to the dining room with a loaded syringe, otherwise the patient would not eat. He then began to eat ravenously, and after gaining considerable weight and strength, eloped, arriving safely home in South Carolina. There was slight improvement in his mental state. The aim in forcing the patient to eat by the administration of metrazol, after his physical condition became alarming, was successfully achieved. In March, 1939, the patient was again found wandering through Maryland. His mental condition had declined in the past six months. He stated that he greatly feared and hated both the treatments and the tube feeding, but that he could have continued with the tube feedings, but that the fear of the metrazol treatments compelled him to resume eating.

CASE 3.—G. N., age 40, married, steel worker, admitted April 3, 1936, claimed that he had had a "nervous stomach" for two years, that his work was too much for him; he was worried, thought he was inferior, believed his fellow workers hated him, had visual hallucinations, was afraid of people, and kept a gun under his pillow. He was vague, apathetic, preoccupied and stuporous. He was given a course of insulin in 1937 with no results. In February, 1938, he refused to eat, and metrazol was administered, 17 reactions resulting. The patient then began to eat, gained weight and eloped to his sister's home in Virginia in April, 1938. In August, 1938, he was admitted to Eastern State Hospital, and when he was returned to us he was refusing food and was tube fed until December. Metrazol was then once more resorted to, and he started eating after six injections and continued to eat for three weeks, again refusing food but resuming eating after two more injections. In February, 1939, he stopped eating, but after two injections resumed and continued eating ravenously, gaining in weight and strength, and again eloping in April. He stopped eating at home in June, and was again returned to the hospital. This patient expressed a great dislike and fear for the drug, and would eat in order to avoid its administration. In this case five times within a year metrazol proved successful in inducing the patient to eat after prolonged refusal of all food and water.

CASE 4.—A. C., age 44, single, peddler, admitted July 6, 1938, with paranoid delusions against his family and friends, believing that he was being persecuted, that his people wanted to ruin him because of his religious beliefs, that his mother wanted to poison him. He was preoccupied, assumed the same position for hours, entertained auditory hallucinations. He refused to eat, giving various reasons at different times: because the food was not kosher, because it did not agree with him, because it was not his own, and

because the "hospital voice" told him not to eat. In October, 1938, he refused to eat, lost considerable weight, and tube feeding was resorted to. Twelve metrazol injections were given in December, 1938, obtaining eight reactions, and the patient resumed eating for a month; again refused to eat, resumed eating after one injection of metrazol, and continued to eat for three weeks. Upon again refusing to eat he was given two injections, and resumed eating for two months. In May, 1939, it was necessary to administer metrazol with seven reactions resulting, and he has been eating for over two months. When he was compelled to eat by the administration of metrazol, he did not object that the food was not kosher. He expressed fear of the treatment on several occasions. When asked why he ate after the treatment, he replied: "The 'hospital voice' tells me not to eat, but when I get the medicine I hear the doctor's voice which is stronger than the 'hospital voice', the doctor's voice tells me to eat." In this case four times in one year metrazol administration was successful in inducing the patient to resume eating after he had refused to eat and had lost much weight and strength despite tube feeding.

COMMENT.

Four cases are presented in which during the course of long standing psychoses there had been refusal of food in any form with considerable loss of weight and of strength leading to a serious nutritional condition. Spoon feeding and tube feeding did not relieve the condition, and metrazol therapy was resorted to. The treatment proved successful in all four cases, in one permanently and the others temporarily. In the one case of involution melancholia in which the patient completely recovered, there has been no return to fasting for over two years since discharge; in the remaining three cases of schizophrenia where slight or no mental improvement occurred, the cure of the fasting was only temporary, the patients relapsing repeatedly into refusal of food, necessitating repetition of the treatment. The success of the treatment was apparently due to the element of fear, anxiety, apprehension and subsequent submission, judging from the statements and overt behavior of the patients. Three of the patients showed great fear, and stated they would rather eat than go through with the treatment, the fourth attributed the successful outcome to his mental improvement as much as to the fear. Apparently the aura, light reactions or equivalents with great fear and anxiety, mounting to panic proportions, were as potent in inducing the patients to break the fast as the reactions with actual convulsions. When metrazol was administered, the patient was impressively and repeatedly told

that the treatment was to help him to eat, and that as soon as he ate, the treatment would stop. Case 4 stated that "the doctor's voice told him to eat, and that was stronger than the 'hospital voice' which told him not to eat." Case 2 promised to eat when confronted with the treatment, but when the threat was removed, he again refused. Again it is emphasized that in the majority of the cases presented, the effect of the treatment, although pronounced, was only transient, the patients lapsing into their original condition of refusing food, and a renewal of the treatment became necessary.

SUMMARY.

1. Four cases are presented in which metrazol therapy was successful in inducing a resumption of eating after prolonged refusal of food.

2. Fear, anxiety and apprehension play a determining rôle in the treatment with metrazol of obstinate refusal of food in the psychoses.

BIBLIOGRAPHY.

1. Meduna, Laszlo, and Friedman, Emerick: The convulsive irritative therapy of the psychoses. J. A. M. A., 112: 501-509, Feb. 11, 1939.

UNSUCCESSFUL SEX ADJUSTMENT IN MARRIAGE.*

MODERATOR DR. S. BERNARD WORTIS, NEW YORK CITY.

DR. S. BERNARD WORTIS, opening the discussions said: I have purposely arranged to have representatives of many fields of thought give us their opinions concerning these problems. We will hear tonight from the endocrinologist, the gynecologist, the urologist, the anthropologist, the marriage counsellor and the psychiatrist. I had invited to this meeting representatives of the various religions, but unfortunately they were not able to attend.

The more one deals with problems of sexual maladjustment, the more one realizes that these are not the province of any one specialist alone. In this field, certainly, we need the united efforts of many branches of the biological and the psychological sciences.

The first speaker, representing the endocrinologists, is Dr. Allen T. Kenyon who is working in the department of medicine of the University of Chicago. Dr. Kenyon has done outstanding work in the endocrinological aspects of sexuality and has especially been concerned with the androgens and estrogens.

DR. ALLEN T. KENYON: Since my own thoughts have largely been concerned with the properties of androgenic hormones in the male, I must take the liberty of discussing only a minute phase of your very broad subject. Certainly this group would insist at the outset, and I would agree, that endocrinological defects are not the sole cause of deficiency in sexual desire and potency in the male. Indeed we have all seen eunuchoids with very imperfectly developed secondary sex characters, small genitalia and practically no prostate who have maintained an active and apparently satisfactory marital life. The question of real importance here is whether endocrine defect has anything at all to do with that vast group of men who appear perfectly normal and yet fail in sexual performance. I shall be so bold as to say that I believe that this point is insufficiently explored. When once well established, hair growth and voice are no longer sensitive indicators of the amounts of circulating androgens and prostatic size means little if the previous mass of this organ in the patient is unknown. A decline in testicular secretion below the level to which the organism has been accustomed may therefore be of importance and be easily overlooked. Assays

* Report of a round table discussion held at the ninety-fifth annual meeting of The American Psychiatric Association, Chicago, Illinois, May 8-12, 1939.

of urine for androgenic materials have not as yet been widely applied in this problem, and their potential usefulness hence not defined. This it seems to me is the next step.

In the eunuchoid, testosterone propionate, in addition to increasing the size of the penis and of the prostate, causing growth of body hair and deepening the voice, has oftentimes a powerful effect in increasing sexual desire and frequency of erections. The degree of these latter responses seems to be greatly qualified by individual differences in threshold which we may assume are partly determined by the individual nervous system. While normal men, whose natural load of androgens is already high, seem less sensitive to these erotogenic properties of testosterone propionate, it does not follow that no such response may ever be elicited. Indeed we have recently found that the nitrogen, phosphorus and salt retaining properties of testosterone propionate can be well observed in normal men. Accordingly I should expect that granted a suitably sensitive nervous system even endocrinologically sound men may well respond to added androgens by increased sexual desire and potency and that certain psychological handicaps to healthy sexual function may be overborne. Our own data, collected with the collaboration of Dr. Carmichael of your association, together with that of others, is as yet too meager to warrant a statement on this point. Thus far normal appearing men with impaired sexual desire and capacity for intercourse may or may not be benefited by androgen treatment. Whether the successes are due to suggestion or the failures to inadequate dosage cannot now be answered.

We have thus outlined only one possible course through the labyrinth of uncertainties of a very small phase of your problem. I shall therefore close, the evening's first example of the "Ariadne complex."

DR. WORTIS: Dr. Earl T. Engle is research director of the National Committee on Maternal Health in New York City. He has been in large part responsible for the planning, on a wide, careful and sensible foundation, of the research work in problems of human sterility and fertility that this committee has been interested in. He is also an endocrinologist and an anatomist connected with the Columbia University College of Physicians and Surgeons in New York City.

DR. EARL T. ENGLE: To confirm the last speaker, I wish to emphasize the need of research in this field as we are totally and absolutely ignorant of the rôle of estrogens and androgens and their effects on the personality structure. All we know is that hormones are essential to reproduction.

DR. WORTIS: In this country the man who has devoted his life to the elucidation of problems of sex has been Dr. Robert L. Dickinson of New York City. He is a gynecologist of note whose Atlas

of Human Sex Anatomy is known to all of you. In his unavoidable absence I will read a short abstract of his views.

DR. ROBERT L. DICKINSON: I wish to emphasize the necessity of including a careful physical study in any research program on sex. In five of the six main studies of sex life so far published, as well as in the great mass of Freudian evidence, deliberate refusal or evasion to secure anatomical findings has been conspicuous.

The answer to sweeping denials of any considerable proportion of physical maladjustments is my Atlas. Here history with human sex anatomy are correlated with the drawing to scale of the vulva or the elasticity of the hymen tied to the record. Here the otherwise unbelievable disproportion of piston and passage are shown. A single diagram suffices to make clear that a third part of the contents of a bone encased cavity must make room for swift thrusting, then for sudden collapse.

Happily, I have been able to instigate sense of proportion into three of the present sex studies. George Henry's homosexual inquiry has x-rays of skull, chest and pelvis and full physical and genital examination of the men and women. The valuable Landis study has D'Esopo's pelvic records. The important Kelly follow-up has James Raglan Mill as counsellor on gynecologic findings.

It goes without saying that physical findings are so easily brought out that undue stress is likely to be laid on mere structure. The complicated and difficult analysis of psychological factors is of major import in almost any problem. But avoidance of complete study is not excusable.

DR. WORTIS: Dr. Max Mayer of New York City has been devoting his efforts for many years to a study of the problems of frigidity, sterility and dyspareunia at the Mt. Sinai Hospital in New York City. His unusually broad training in obstetrics and gynecology and in psychoanalysis have particularly equipped him to deal with problems in this field.

DR. MAX MAYER: A good percentage of cases of marital maladjustment come to the gynecologists. The barriers against discussing such matters are down and gynecological symptoms are often projected to the genital zone. My own experience has been that only a minority of patients approach the problem directly. The complaints may take the form of conversion symptoms, of anxiety states, or the over-evaluation of existing gynecological conditions used consciously or unconsciously as an excuse.

The gynecologist must be willing to realize that the presenting symptom is not of necessity the most important. He must be willing to listen, to evaluate and to obtain a history that can be reconstructed into a dynamic biography.

In the diagnosis there must be:

1. A diagnosis of the situation.

2. A diagnosis of the individual. Are we dealing with a factual situation or with a deep-seated neurosis or a psychosis.

3. A diagnosis of the partner.

4. A diagnosis of therapeutic resources.

In the management of the cases we must evaluate the situation as a whole, organize the problem and organize our objectives. Above all, the doctor must be extremely modest in his estimate of what can be accomplished. The primary consideration is care not to do harm under any circumstances. Great patience is necessary in the reservation of judgment and all hurry must be avoided.

In a simple situation involving the use of gynecological technique and common sense, the gynecologist may be in an extremely advantageous position. This is particularly true if in addition he has some training in this line of work, a knowledge of his own limitations and a willingness to cooperate with specialists in psychotherapy as well as a recognition of the importance of anxiety as the root of many problems.

DR. WORTIS: Dr. Robert Hotchkiss of New York University is a urologist who has done much research in human sperm biology.

DR. ROBERT HOTCHKISS: The otherwise well adjusted male who develops impotence is most apt to consult first his family physician or a urologist. The first confidant has certain advantages that are often difficult to confer to subsequent consultants.

The rôle of an organic lesion of the genital apparatus as it influences the sexual function, is likely to be under-emphasized by the neurologist and psychiatrist and and exaggerated by the urologist. All will agree that certain congenital and endocrinal faults seriously impair the sexual function. These defects are quite obvious, and as a rule cause no controversy. Other hidden abnormalities produce lesions which are apparent only upon detailed urological examination. For example, a urethral stricture may exist which is not appreciated until an instrument is passed into that canal. Posterior to this obstruction there may be marked inflammation of urethra and prostate. Definite prostatitis and seminal vesiculitis may be disclosed only by rectal digital palpation and microscopic examination of the expressed secretions. The complaint of premature ejaculation is sometimes found to be associated with old inflammatory lesions or new growths about the verumontanum. It becomes the objective of the urologist to determine what part these lesions have, or now play, in the development of the sexual disorder. If the urologist acts as a consultant and finds no demonstrable abnormalities in the genital structures, his participation in the case should then cease, but being the first man consulted, the whole problem may be presented to him for solution. It is here that he runs the greatest risk of mismanagement. Psychotherapy is essentially foreign to his routine practices; whereas, it is the routine work of a psychiatrist. The urologist may not recognize fundamental and serious psychoses of which impotence is but a presenting symptom. However, certain situations can be handled with encouragement and the

correction of misconceptions and faulty knowledge on the part of the patient. In dealing with this subject we are concerned with the question, who is best qualified to treat? As a urologist I ask whether a psychiatrist may do harm by unfolding or disclosing a complex mental disturbance to an otherwise well adjusted man complaining only of impotence; and finally, do too many consultants destroy the confidential relationship between patient and physician?

DR. WORTIS: Dr. Ernest W. Burgess, Professor of Sociology of Chicago University, needs no introduction to any psychiatric or sociologic group.

DR. ERNEST W. BURGESS: Terman's study "Psychological Factors in Marital Adjustment" attempted to analyze the factors making for happiness in marriage as revealed through the study of several hundred married couples in California. The most important factors were (1) conflicts in childhood; (2) over-attachment to a parent; (3) discipline in childhood; (4) cultural differences between the married partners. These factors all loom larger than the actual sexual adjustment itself. The part played by economics is a relative one—not the amount of money, but the attitude toward money which the couple has is most important, and finally it is the woman rather than the man who makes the adjustment.

I indicated in my studies made in 1931 on predicting adjustment in marriage of couples living in the State of Illinois who had been married one to six years, economically in the middle class group, and half of whom had college educations, the following important factors:

1. The happiness of the parents in marriage was a most significant factor, indicating the possibility of a happy marriage for the persons involved.

2. The more closely young people were attached to their parents, the more likely they were to have success in marriage later in life.

3. Children belonging to families of more than four children had more success in marriage.

4. Marriages of oldest children or youngest children were likely to be less successful.

5. There was a factor of the impress of the cultural background that seemed important. The more similar the backgrounds, the more opportunity there was for success in any particular marriage.

6. The more education people had, the greater possibility they had for success in marriage.

7. Those couples who were married in church or in synagogue had a greater success than those who were married without religious ritual.

8. Economic factors were of some importance. Those couples with moderate incomes had the most success in marriage. A preceding regular work record was a favorable sign.

There were many other factors that I evaluated, including the relation of occupation and career to marriage success, length of acquaintance of

the couples before marriage, the question of parental approval, and the relative ages of the individuals contracting marriage. Marital compatibility depends in large part on the relationship of the individual to his parents and to siblings, which relationship is formed in early life. Cultural and psychogenic factors are most important and it must be stressed that both the psychiatrist and the sociologist are necessary for the achievement of the best research work in this field. Cooperation is an absolute necessity in this work.

DR. WORTIS: Dr. Jacob Conn of Baltimore is a psychiatrist who has been working at the Johns Hopkins Hospital in the pediatric division, where he has been investigating sex awareness in children. He has made particular use of the play technique.

DR. JACOB CONN: In contrast to the attitude of earlier students of human sexual behavior, none of the previous speakers has referred to the existence of a sexual instinct. They have preferred to speak of sets of facts (endocrine, personal or sociological) and have considered it unnecessary to reintroduce the concept of an hypothetical sex instinct. The present-day psychologist, sexologist and psychiatrist prefer to think of the sexual impulse and sexual drive in terms of an unformed, unorganized, blind urge yet to be molded and directed by the social process.

In going over our own material, I prefer to think of the gradual development of sexual awareness and sexual attitudes in children as part of the more general process of growth. We are apparently dealing with one phase of a directional process, during which the infant, who began as a dependent, helpless unit, and must be served in order to exist, is being directed and influenced by those about him to become an independent, helpful adult who is ready to serve others.

It is not very helpful to speak of this process of growth and social differentiation in terms of conditioning. The present tendency is to humanize our concepts of behavior and to think in terms of plastic human processes rather than in terms of rigid laboratory experiments. The development of human sexual behavior is not so much one of conditioning in its proper laboratory sense, but rather one of direction. The question therefore may be raised as to what factors maintain and what factors hinder this directional process, which goes hand-in-hand with that of maturation. Our own observations as well as those of Terman and Miles ("Sex and Personality") indicate that certain personal-situational constellations may influence future sex adjustments. The overprotected child who is kept from learning boys' games and who is urged by his oversolicitous mother to engage in sedentary activities, may in this manner be made uncomfortable in the presence of his coevals and become dissatisfied with his sex status. In this manner he also may become less oriented in sexual matters than his fellows, who are discussing sexual topics on the street corners and gradually turning their sex imaginations and sex interests in the direction of heterosexuality. We may begin to wonder if the formal sex instruction, which is being offered in

some of our schools, is actually more helpful than these street-corner opportunities for the exchange of sexual facts and fancies, coming as they do, at the age when the average child is interested in this topic.

In our culture it is expected of the male to take the lead. His sex interests and sex pace are more readily initiated. He is still considered the protector and despite all that is said about career women and the equality of the sexes, the woman in our culture must be able to respect the man with whom she lives in order to get along with him harmoniously. The inadequate, timid male may be expected to have more difficulty in making a satisfactory sex adjustment than his relatively more forceful, self-assertive brother.

In our experiments we have been able to redirect the play interests of timid boys who have been overprotected. In a relatively short time these children, who had wanted to be girls and play girls' games, were able to develop interests in active, competitive sports after they had learned that they had been scaring themselves by constantly anticipating being hurt whenever they thought of engaging in boys' sports. The acceptance of the full realization that it was not the game itself, but their own timidity, which was responsible for their desire to avoid active rivalry, was essential in the process of redirection. This material indicates that the problems of sexual adjustment are contained in the larger setting of personality difficulties. Perhaps with our particular scientific and cultural interest we are emphasizing too much the topic of sexual maladjustment and losing sight of its larger frame of reference, namely, that of its personality setting.

Finally, it may be reemphasized that in considering sexual behavior, we must become more and more aware that we are thinking in more complex terms than those of hormones and structural-physiological data. Our colleagues who deal with these factors have been very modest in their statements. Let us also be discreet and discuss our own findings in terms of personal relationships. Our contribution as child psychiatrists may best consist of studying those factors which help to direct these human relationships toward mature, adult patterns of behavior. It is my belief that in such future studies there will be found a greater degree of plasticity in the formation and direction of these early relationships than we have been led to believe by the dogmatic statements of the past.

DR. WORTIS: The two outstanding European medical men who helped to throw light on the problems of sex were Sigmund Freud of Vienna and Havelock Ellis of England. The next speaker, Dr. Sandor Lorand of New York City, is a psychoanalyst who has been especially interested in problems of sexual impotence.

DR. SANDOR LORAND: Sexual maladjustment of marriage partners may be the cause of many difficulties which may not only lead to the breaking up of the marriage but may also create various neurotic difficulties. The ability to have a happy sexual life is parallel to a happy outlook and adjustment to reality situations.

Patients who seek psychiatric or psycho-analytical treatment for sexual difficulties—whether man or woman—seem to fall into two groups. First, those who developed difficulties in marriage. This group is easier to help and treat. Second, those patients whose sexual difficulties are deeper and more fundamental and are directly related to an earlier period of their life. Their maladjustment concerning sexual matters was already present from puberty and even earlier childhood.

In the first group, the difficulties acquired in marriage may be due to a number of causes. I will briefly mention a few. A common example is that of a husband who has had premarital sexual experience and who fails to take into consideration the wife's inexperience or ignorance concerning the whole problem of sex. The bride who has to learn to adjust herself and to enjoy this new sexual relationship in the early months of marriage, may not be given a chance by the over eager or aggressive husband. He is mostly concerned with himself and his own gratification. Or perhaps he does not know how to handle the wife and does not realize that under the best circumstances sex relationship carries a traumatic charge to her.

The second group, those whose sexual difficulties lead back to early childhood experiences, find that the difficulties are chiefly caused by the fixation of a strong early childhood attachment to the parents which is known as "Œdipus complex." This complex is a result of the strong attachment of the little boy to mother and is helped very often by the over protective mother who hampers the independence of the little boy; or in the absence of the father this fixation is nurtured by the development of a strong bond between the boy and mother. Or in the little girl an over strong attachment in early childhood to the father may be carried over unconsciously to adult life and may create sexual disturbances in marriage: the husband is always somewhat a father to the wife and the wife is, to an extent, a mother to the husband (at least in the unconscious).

Early childhood intensive masturbation carries to a degree a sense of guilt since it represents revolt against adult authority. And this sense of guilt is carried over to the whole problem of sex and pleasure in adult life. This too may interfere with the proper sexual adjustment.

In general, impotence and frigidity can be looked upon as conversion symptoms where mental and emotional conflicts are creating bodily symptoms. As such they can be favorably treated and influenced by psychotherapy, particularly psychoanalysis.

DR. WORTIS: Representing the marriage counsellors are Dr. Alexandra Adler of Boston, Dr. Beatrice Bishop Berle of New York, Dr. Grace Baker of Johns Hopkins and Mrs. Stuart Mudd of Philadelphia.

DR. ALEXANDRA ADLER: To begin with, it is rather difficult to make a definite statement whether two people are well or poorly adjusted sexually. It seems to be rather a matter of opinion and the opinion of the two people concerned may be contradictory to that of other persons. To give an ex-

treme example, I had an opportunity to examine a transvestite, a young man 30 years of age, a clerk, who wore hidden under his coat a long braid of his hair. He also wore women's underwear. He had been educated as if he were a girl by a mother who very much regretted that her only child was a boy and not a girl. After the death of his mother he married a woman 15 years older than himself who apparently replaced the mother for him. They slept in the same bed, kissed each other, but never came to sexual intercourse. Still, both were perfectly happy and felt very well adjusted sexually. His perversion was discovered by chance and, after having lost his job, he tried to commit suicide and was put under observation. They were adjusted to each other though not to the community.

It has been stressed during this discussion that sexual adjustment in marriage has to be taken as an expression of the personality development and not as a primary factor. Every marriage counsellor ought to keep this in mind, and I feel urged to point out that still many mistakes are being made in this respect. Even psychotic patients are sometimes expected to benefit by marital relations. One case occurs to me where the parents of a young evidently schizophrenic girl were advised by their physician that marital sexual activities might save the girl. They bought her a husband, a young man who was in financial straits. No wonder that this girl did not derive any kind of satisfaction from this new relationship. She developed vaginism which made sexual relations painful and almost impossible. Her psychosis progressed rapidly. One cannot expect a patient with progressive psychosis to make a satisfactory adjustment. In her hallucinatory experiences her husband became distorted, without arms, his eyes removed from his face. The only activity of her husband was finally that of a male nurse to a deteriorating schizophrenic.

Also, neurotics are still being strongly advised by psychotherapists to masturbate if they do not derive sexual satisfaction from their marital partner. However, some kind of sexual maladjustment is almost the rule in neurotics and is in direct connection with their personality. Masturbation, the sexual activity of the isolated individual, is the appropriate symptom of the neurotic, but not the way out of the neurosis. Still, in innumerable cases, this advice is given by the psychotherapist. I know of a severe case of compulsion neurosis. She tried to force her husband into adopting ceremonials and rites in their relations in the same way that she used them in her daily life. As will be suspected, she was frigid. Her psychotherapist threatened that he would not see her any more unless she masturbated. Faithfully she did it, but her neurosis did not improve upon this advice and her relations to her husband became unbearable. The same advice is still being given to sexual perverts. I have seen at the best no effects and frequently a change to the worse.

It has been stressed in this discussion by experts in endocrinology that we can only be sure that the sex glands are responsible for reproduction, while we have no definite knowledge about the relation of sex glands and sexual adjustment or maladjustment. Whenever sexual perverts succeeded in their marital relationship,—and there are many cases known—this took

place after a profound change of personality and not after any technical advice for sexual activities.

Finally, I would like to point out that we can expect some kind of sexual maladjustment frequently at the beginning of marriage. Under normal conditions, these will be straightened out by the efforts of the partners themselves. Normal people will not need a great deal of marriage counselling.

DR. BEATRICE BERLE: I have been talking with college audiences both men and women for the past ten years about marriage and family problems. As I look back over the discussions which we have held and the questions which young people have asked, it seems to me that we have gone through a certain evolution. Ten years ago sex was news to college students; this might be called the age of innocence when students as well as educators thought that knowing the facts of life would be a sufficient remedy for all evils. This merged into the age of Freud. Then all our ills were due to a father fixation, and quarreling at breakfast was a symptom of lack of orgasm. Today I hope we approach closer to the age of reason, and whereas we realize the importance of knowing the facts of life, clinical case studies bear out the truth of many of Terman's statements, and the fact that sex problems cannot be considered apart from problems of the total personality.

DR. GRACE BAKER: Dr. Wortis, in the arrangement of this evening's program, has been indicating what are some of the needs and what should be the essential equipment of a competent marriage counselor. It would appear that a marriage counselor should be able to correlate, integrate and utilize the contributions from the fields of endocrinology, anthropology, the social sciences, psychology, psychiatry, psychoanalysis, religion, philosophy, urology and gynecology.

1. Since it is obvious that no one human being can be a specialist in all these fields, it would seem that the marriage counselor's special contribution might be one of integration.

2. It would seem well to remember that, since marriage concerns two individuals, both persons should be given careful consideration. New problems can frequently arise if only one party to the marriage receives counsel.

3. Careful attention should be paid to the complaint. There seems to be a tendency to take the complaint too much at its face value. The patient may object to some apparently trivial difficulty of the partner's, such as an irritating mannerism, some problem of personal hygiene or to apparently more serious problems about methods or technique. Should we feel that the problem is solved when we acquaint the partner with the objections, suggest better habits of cleanliness and give directions as to improved methods of technique, or should we ask ourselves what is the real significance of the complaint? Does it perhaps represent a deep-seated fear or repugnance of the sex relationship or of the partner? What does a complaint *mean* to the patient? I feel increasingly suspicious of "quick" cures.

4. Indications that we have failed to really solve the individual's difficulties are to be found in the variation of the complaints against the partner or the relationship; difficulty in grasping the information given or in reactions towards the counselor that the information given was unclear or inadequate and in repeated requests for more information. We should never lose sight of the difference between intellectual understanding (thinking) and emotional acceptance (believing).

5. I would like to call attention to the importance of the counselor's own emotional adjustment.

MRS. STUART MUDD: The principal work of the Philadelphia Marriage Counsel is preventive; it is not a psychiatric clinic, but rather works in cooperation with psychiatrists. One-third of the total intake are pre-marital cases. In the contacts with this group fear and insecurity are almost always obvious, even in that classification which we term "normal," that is, those who do not exhibit signs of personality disorder.

The majority of clients who come for interviews before marriage are already engaged, and come at the suggestion of ministers, doctors or friends, to have their questions answered about the physiology and psychology of sex adjustment, and to obtain information about contraception and pre-marital examination. 80 per cent of the pre-marital cases fall into the group of so-called "normal" young people. Short-term service is often all that they require. Over half of them are given pamphlets or books to supplement the interview. A few need to think through the relationship of their religious background to their plans for family limitation. Others ask for help in deciding what pattern of behavior they will follow before marriage, especially when a long engagement is necessary. But for the most part this group is able to accept facts and go into marriage with many fears dispelled.

The remaining 20 per cent of those seen before marriage is made up of two types of clients: those who come on account of a specific situation, or those who are so disturbed that their questions about marriage seem to imply a total personality maladjustment. Of the 14 per cent who have a specific problem, several have come because of fear of the inheritance of mental disease, others with questions about the fiancé's relationship to members of his or her family, and a few with fears about venereal disease. Some of these doubts and fears can be removed by facts so that these individuals are able at least to crystallize a plan of action. A few who apparently come with a definite question turn out to be suffering from deep-seated anxiety and are not fundamentally helped by a short contact with a counselling service, but need more far reaching psychotherapy. They are referred to psychiatrists whenever possible.

DR. ROBERT H. FELIX (U. S. Public Health Service, Lexington, Ky.): It would be misleading to stress any one aspect of the marital state above all others as the principal factor contributing to sexual maladjustment. Probably no one cause operates alone to produce marital discord, but,

rather, several act together, each influenced by the others. We can do no more than mention some of these.

Let us first think a moment of many young people of today, with little or no insight into the emotional or physical requirements for successful marriage. They look upon marriage as a refuge or as a means to an end. Thus they marry for security, because of social ambitions, obstinacy, spite, or to secure freedom from parental rule. These motives are all self-centered and lacking in the altruistic motives which are essential to a successful adjustment. These individuals are usually emotionally immature to begin with and hence are not prepared for marriage.

Speaking of the immature individual brings to mind the psycho-sexually arrested man or woman who has never attained full heterosexual maturity. Successful marriage in these cases is dependent upon the ability of both partners to realize the difficulty without too much emotional turmoil and to adjust to it as far as is possible. Certainly psychotherapy is badly needed, but lacking this, common sense and a sympathetic attitude on the part of the marital partner has frequently worked wonders.

Many couples appreciate the obligations of marriage so keenly that they postpone entering into the relationship until a later period in life in an effort to be more adequately prepared. This occurs frequently among college students. Marriage is often deferred in these cases until the latter twenties or even older. These years set the individual more deeply in bachelor ways and make adjustments more difficult. Further, the sexual drive, is, to a great extent, sublimated unless promiscuity or autoeroticism is indulged in. In any case, a satisfactory sexual adjustment is made more difficult and in some cases is impossible.

One sees all too many cases of marital discord after several years of apparently good adjustment. It is felt that much of this is due to a growing disregard for the æsthetic side of marriage as the years go by. This is usually due to personal carelessness. The man or woman who forgets that after marriage pleasant stimulation of the senses, including the sense of sight, continues to be a necessary part of the technique of successful love making is in danger of destroying a satisfactory relationship, since not only is erotic stimulation difficult in the presence of conditions which offend the senses, but also the end result is very likely to be a feeling of disgust which will eventually, at the very least, change love to a feeling of tolerance. In other cases the everlasting dailiness of marriage is killing to the erotic drive. To see the same face at bed and board, to see it getting up, being prepared for bed or being made up day after day may destroy the charm of the finished product and become more than one can endure unless supplemented by mutual interests and a mature outlook.

Another cause should be mentioned, which, if properly handled, can be avoided. One encounters cases in which there is a disproportionate rate of cultural, physical and emotional development. One partner continues to grow in stature while the other grows stagnant. This may be due to gross disproportions in intellectual capacity, lack of opportunity for development due to work or children, illness, or a waning of interest in improvement.

In some cases the interest of the husband and wife are along such divergent lines that they can find no common ground. Disaffection gradually develops, together with a desire of one for intellectual and cultural stimulation which leads to outside interests and contacts. In some cases these contacts may cause such a sense of guilt that the breech becomes even wider because of inability to face the marital partner.

Time prevents more than mention of those who feel unable to rear a family for one reason or another and thus inhibit free expression of their physical desires through dread of pregnancy; and also those who use contraceptives much to the dislike of one or both or contrary to their moral teachings. Mention should also be made of the man or woman who is preoccupied with worry or tired out from work. If this becomes a chronic condition not shared by both, trouble may result.

From the standpoint of the psychiatrist, many of these problems can be handled and corrected by proper guidance and council, and until this can be provided in adequate amounts, we can expect to meet these and other problems in our clinics.

DR. THEODORE WATTERS (Associate Professor of Psychiatry at Tulane University, New Orleans, La.): I wonder whether we realize that more is expected of a psychiatrist than can reasonably be demanded of any human being. We are supposed to be able to solve all problems in all fields. It behooves us to be humble. Let us remember that many things make a marriage. Old-fashioned selfishness spoils more than one matrimonial venture. Often we see people who know nothing about finances and cannot budget, and in my part of the world religious differences are frequent sources of conflict. The whole attitude towards life and the standard of values of both partners must be considered. There are minor matters such as halitosis which must not be disregarded. As a last word to the psychiatrist, however, let me emphasize that sex in our civilization, in our scientific text books, as well as in our advertising has been oversold.

DR. WALTER BROMBERG (Psychiatrist to the Court of General Sessions, New York City): I have been asked to comment on some of the sexual problems in marriage which one encounters in a court practice. The number of such difficulties is surprisingly large. First, we find in the bigamy group, contrary to the popular notion, that many of these cases have serious inadequacies in their sexual marital histories. The "Bluebeard type" commonly expected among bigamists is lacking in our material. More often we deal with a severely inadequate individual who starts extra-marital contacts through his dependency-need and moves into a bigamous marriage from that point. Another type is the Don Juan, whose flashy exterior covers unconscious psycho-sexual difficulty. This individual often uses bigamy as a swindling technique. In this, he utilizes his sexual powers, charm and social attractiveness to get a wife who will give him money. Underneath this ease of "seduction," we have found evidence of an immature psycho-

sexual make-up, with marital break-ups and thinly covered sexual maladjustments.

I have said that in general there is a high correlation between adult delinquency and sex maladjustment. In the history of many larcenists can be found broken marriages. There are, of course, external factors to be considered, such as the impossibilities of reconciling a legitimate career with a marital adjustment. The other factors are internal ones which have to do with the personality immaturity of the offender. This immaturity is expressed in two ways: one, the acceptance of a social ideology which leads them to use crime in the normal expression of their social feelings, and the other, the sexual disturbances which force them into extra-marital relations and consequent marital disharmony. Very often the lack of tenderness to the wife which characterizes marriage of these offenders, is a personality hardness which covers up a deeper inner insecurity. Especially is this so among the adolescent or young adult offenders who marry early up to the age of 25. Here sexual inadequacy, premature ejaculation, disinterest in satisfying the partner, crudeness in sex relations and a utilitarian attitude toward women as sexual partners is frequent. In connection with young rapists, I speak now of statutory rape with consent of both parties although under age, where marriage is forced following the rape, difficulties often arise on a sexual basis. Social agencies, religious groups, legal institutions insist that legitimatization of a child has prior rights over any other consideration and marriages are consummated without the slightest interest in the conflicting or incompatible personalities. A personality analysis of the principles involved would prevent possibly, so many sexual incompatibilities and marital disharmonies which arise in such situations.

The third group where sexual difficulties arise includes the sex criminals. This cannot be discussed at this time, but a study is being prepared in the clinic which will appear in the course of the next year. Here a large number of sexual neurotics are found in contrast to the popular view that sex offenders are bloodthirsty sadistic monsters. I may quote one case of a 34 year old man who was arrested for sodomy on a boy. He had become impotent some years before for neurotic reasons and his wife forced him into divorce for this reason. Subsequently he turned to inverted sex activities with young boys.

DR. WORTIS: The man in this country who has always insisted on the happy correlation of psychological and somatic studies is Dr. Adolf Meyer of Baltimore, Professor of Psychiatry, Johns Hopkins University Medical School.

DR. ADOLF MEYER: We have been talking a great deal about sex and marriage this evening. I do not think we can answer any of these questions unless we consider what is the purpose of sex and marriage. The statistical evenness on which the physical scientist depends is much displaced if not annulled when we come to man. Hunger has to do with the preservation of life, but in sex there is always the element of choice. I am a pluralist

by conviction and by necessity. We must make a compound picture of a human situation realizing that psychiatry can only offer a solution patched up in the direction that people want. We must develop a sound sex concept with an understanding of what it means in the total situation. Finally, life involves compromise, concessions and adaptations.

CLOSING REMARKS BY DR. WORTIS: This meeting has emphasized the necessity for a pluralistic viewpoint in studying sexual behavior and sexual problems. The sexologist must be qualified and willing to integrate advances in endocrinologic, gynecologic, urologic, physiologic, sociologic, anthropologic and psychologic knowledge. Disturbance in sexual function may arise from one or several causes. As regards the rôle of the marriage counsellor, he or she must be a good listener and himself or herself have no problems or pet ideas to work off on the patient seeking advice. The counsellor must not adopt a coercive attitude and should be ever mindful that there are no dogmatic "laws" of sexual behavior. People's methods for obtaining sexual satisfaction vary. Any happily, mutually acceptable, satisfaction-giving method, is a good one.

The physician dealing with these problems should not only be informed in the related branches of medical, sociologic and psychologic learning—but in addition must mix with his scientific knowledge the essential quality of human sympathetic understanding.

In this field, especially, the patient's confidence in his physician is the most important therapeutic instrument.

THE MECHANISM OF FRACTURES OF THE VERTEBRAL BODIES.

Fractures of the Mid-Thoracic Area Complicating Metrazol Therapy—Prevention and Treatment.*

By A. M. RECHTMAN, M. D., F. A. C. S.

A review of the anatomy, together with a practical consideration of the usual sites of vertebral body fractures, aids in the further consideration of similar injuries in the more unusual site at the mid-thoracic area. Fractures of the vertebral bodies in the mid-dorsal spine have been observed and reported during convulsions. This paper is written in an endeavor to explain these fractures in the mid-thoracic spine.

Anatomy and Practical Consideration of the More Usual Spinal Injuries.

Despite the anatomical construction of the spine, structurally designed to withstand trauma, injuries of the bony parts and soft structures do occur. A study of the spine shows that there are 135 joints, that the vertebral bodies articulate with each other through the intervertebral discs, and that the compression of the nucleus pulposus of the disc causes the action of the articulation to simulate that of a ball and socket joint. Further, in the thoracic region there are articular, demiarticular and postarticular facets at the junctions of the ribs with the spine. In the thoracic area, in addition to the intervertebral discs, the vertebrae have approximately ten synovial-lined cavities. Despite all these many anatomical factors tending to provide motion, the spine is not very flexible. Actually there is but little movement in any of the three areas of the spine (cervical, dorsal and lumbar). Flexion is most marked in the lumbar spine, and there is least motion in the dorsal region. The accessory movements between the head and neck, and between the pelvis and hips, are responsible for the apparent freedom of spinal motion.

* From the orthopedic service of the Jewish Hospital, Philadelphia, Pa.

For purposes of balance, particularly in the fully upright adult posture, the spine has a number of physiological curves (Fig. 1). The thoracic and pelvic curves of the spine [1] are convex backward;



FIG. 1.

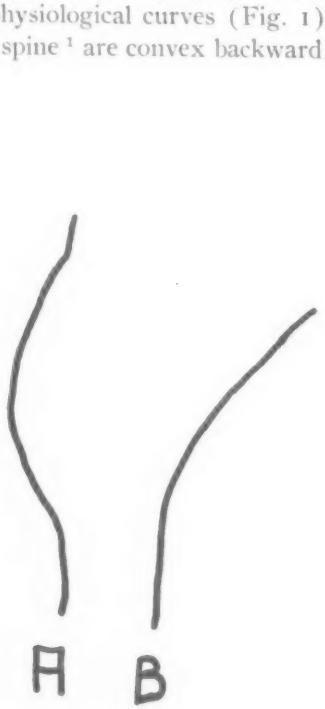


FIG. 2.

FIG. 1. (After Love).—Shows a schematic sagittal section of the spine indicating the vertebral bodies and discs, the physiological curves, the fourth to the sixth thoracic area, the usual site of fractures after metrazol injections.

FIG. 2. (After Jones and Lovett).—"A" is a line drawing indicating the physiological curves of the spine formed by the vertebral bodies and vertebral discs. "B" is a line indicating the attitude of the spine when the discs have been removed.

while the cervical and lumbar curves are concave backward. The dorsal spine which forms a part of the thoracic wall has a primary curve, which is present at birth and is due chiefly to the shape of

[1] Piersol, George A.: Human Anatomy, Ed. I. (Practical Considerations of the Spine), pp. 143-148. Philadelphia, J. B. Lippincott Co., 1907.

the vertebral bodies. The cervical and lumbar curves are formed after birth [2] and are, therefore, secondary curves. The shape of these curves depends mainly on the discs. (Fig. 2.) Also, there is greater mobility in these areas. The spinal curves are in part produced and maintained by the 23 intervertebral discs. The discs increase in size and elasticity in the lower portion of the spine where, too, the vertebral bodies are larger. In the thoracic region of the spine the discs are wider posteriorly, while, conversely, the bodies of the vertebrae are wider in front. The function of the discs is to give shape to the spine, to help form the physiological curves, to assist in motion between the vertebral bodies by compression of the nucleus pulposus, and to act as buffers in minimizing the effects of shock and jarring and to help protect the vertebral bodies from fractures. With dehydration occurring in old age, the discs lose some of their elasticity and shape. This accounts for the round back attitude and loss of stature seen in older individuals. The vertebral bodies are smallest in the cervical and largest in the lower (lumbar) region of the spine. The cancellous tissue of the bodies is present in direct proportion to their size. The vertebral bodies, together with the discs, bear the chief strain of shocks.

The spongy nature of the vertebral bodies and their comparative vascularity is such that when softening occurs, as a result of decalcification from whatever cause, the effects of superincumbent weight and muscular action cause disintegration with wedging. In the thoracic region, wedging tends to an increase of the physiological attitude and the resulting deformity is a kyphosis.

Being more exposed to the effects of injury, and due to the absence of support and fixation which the dorsal spine receives by being a part of the thoracic cage, the cervical and lumbar areas of the spine, because of their greater mobility are more prone to the effects of trauma. Since the junctions of the fixed and movable areas of the body are potentially weak, spinal injuries are more common at the first and second cervical areas, the cervico-thoracic, dorso-lumbar and lumbo-sacral areas.

The cervical spine is a frequent site of the effects of trauma because the bodies are small and the area is relatively exposed.

[2] Jones, Sir Robert, and Lovett, Robert W.: Orthopedic Surgery, Vol. 1, (Anatomy of the Spine), pp. 311-14, New York. Wm. Wood & Co., 1923.

The lower dorsal (11 and 12) vertebræ and the junction with the lumbar area are also prone to the effects of injury because this region is situated more nearly in the center of the spine, the leverage is greater, and it is the static level of stress and the two lower dorsal vertebræ are smaller than the adjacent lumbar vertebræ and carry almost as much of the superincumbent weight. Further, this site is the most concave part of the lumbar curve and the circumference of the trunk is smallest. In the lumbar region, there is also powerful leverage of the muscles attaching to the transverse processes and coursing between the ribs and the pelvis. All these factors contribute to the frequency and the usual location of spinal injuries.

The extent and nature of spinal injury is dependent, among other factors, upon the amount of force received. A force of certain amount may cause a sprain and, if the force continues to act or is of greater intensity, a fracture, dislocation or fracture dislocation of the spine may result. A combination of flexion and torsion, in tissues which for some reason are vulnerable, may cause a rupture of the nucleus pulposus. Then, if and when the posterior limiting membrane is stretched or broken, protrusion of the cartilaginous bodies toward and into the neural canal may obtain and pressure of the nerve roots may result in symptoms which depend upon the degree of pressure, the local reaction to a foreign body and the area of involvement.

Reactions to Metrazol Injections.

A group of patients was observed while receiving metrazol injections and during the immediate subsequent reactions. From 5 to 20 seconds after the injection, a tonic spasm or convulsion occurred, which at times was preceded by a slight cough. Occasionally the first spasm was of the clonic type, the tonic spasm being entirely omitted; and in some instances no convulsions occurred, presumably because of inadequate dosage. During the tonic spasm the trunk was in an attitude of mild hyperextension, about 15 degrees, and this attitude persisted for a period of from 5 to 35 seconds. Following a brief period of relaxation, the clonic spasms occurred, attended with a tremendous pounding and impacting muscular force with the trunk flexed 15 to 20 degrees, certainly

far short of the acutely flexed or so-called "jack-knifing" attitude associated with the usual history of vertebral body fracture. The initial clonic spasm was held from a few to about 30 seconds and was followed by as few as three spasms lasting a few seconds to many spasms, the total time of which was as long as a minute. Short intervals of relaxation intervened, with marked muscular contractions until the convulsions ceased. According to the theory of Radmann [3] the force of a jarring or bending fracture of the spine is not transmitted simply from one vertebral body to another, but its horizontal and vertical components are resisted by a third force, a reflex contraction occurring in muscles fixed at a point anterior to the spine. The contraction of these muscles compress the anterior surface of the bodies in a mechanism similar to the tetanic curving of the dorsal spine anteriorly. The convulsions following metrazol therapy have been likened to those associated with tetanus. As a result of the physical and muscular work and energy expended, owing to the one and one-half minutes or less duration of the convulsive stage rather than to any other action of the drug *per se*, the patients were usually exhausted for about 8 to 24 hours.

Fractures confined to the vertebral bodies of the dorsal spine, usually between the fourth and sixth and occasionally the seventh, have been reported following metrazol therapy. Although fractures also occur in this region as a result of the more usual types of traumatism associated with fractures of the bodies of the vertebræ, nevertheless they are unusual. All the spinal fractures complicating metrazol therapy have, however, been reported and shown by roentgen studies to occur in the mid-dorsal spine. Roentgen evidence of such fractures was reported [4] in about half the patients who complained of backache following metrazol injections. Tenderness over the spinous processes of the affected vertebræ was another sign. Thus far, however, disability resulting from these injuries has not been reported.

Fractures were detected in a patient who received two injections of metrazol and in another patient after 15 injections. There

[3] Radmann: Quoted by Roberg.

[4] Bennett, B. T., and Fitzpatrick, C. P.: Fractures of the Spine Complicating Metrazol Therapy. Jour. A.M.A. CXII: 2240 (June 3), 1939.

would appear to be no relation between the degree of compression (wedging) of the vertebral body and in the number of bodies injured as compared to the number of metrazol injections given. The severity of the individual convulsions and the strength of the spine appears to be the primary factors determining the occurrence of fracture. The force of the muscular contractions in the convulsions cannot be estimated nor the resistance of the vertebral bodies to that force. Messerer [5] found that a weight of 440 to 540 pounds was necessary to alter the shape of the adult thoracic vertebral bodies. Gocke [6] found that weights up to 1000 pounds did not alter their form, but that the resistance to compression was decreased and the brittleness increased. Other predisposing causes for the injury have not been observed. Some of the causes considered were demineralization associated with parathyroid or other disturbances resulting in a changed calcium-phosphorus ratio, as osteomalacia, osteoporosis secondary to dietary disturbances, localized inflammatory lesions with a hyperemic decalcification of the vertebræ and bone atrophy from whatever cause. Roberg [7] reported four cases of fresh fractures of the vertebral bodies following tetanic convulsions which were demonstrated anatomically. The type of convulsion due to tetanus has been likened to that caused by metrazol injections. He stated that apart from the mechanical disturbance of structure the vertebræ were normal.

The question regarding the future effects of these injuries is pertinent. Will further wedging of the vertebral bodies, with continued narrowing of the intervertebral discs, occur? Will nerve root pain ensue? May a traumatic arthritis result? These are all possibilities and time and further study can best supply the answers. The wedging observed and reported, however, has not been great and the changes are most manifest in the anterior portion of the vertebral bodies. Therefore, it would seem that the possibilities were not too remote for injury to occur in the articular facets, the nerve roots, intercostal nerves or the spinal cord. Will disc injury cause a rupture of the nucleus pulposus with a protrusion of the cartilaginous fragments into the neural canal?

[5] Messerer: Quoted by Roberg.

[6] Gocke: Quoted by Roberg.

[7] Roberg, O. T.: Spinal Deformity Following Tetanus. Jour. B. & J. Surg. 19: 603, July 1937.

Although protrusion of the neucleus in the fifth and sixth thoracic interspace has been reported, the lesion is rare. Again, during a symposium on fractures of the spine at the January, 1938, meeting of the American Academy of Orthopedic Surgeons, it was stated by the essayists—and there were no dissenting opinions—that fracture of the bodies of the vertebræ was not a known etiological factor for protrusion of the nucleus pulposus into the neural canal. Injury to the disc and its contained nucleus is probably of frequent occurrence, and the products of the disc are displaced, not infrequently, into the fractured centrum. When the injury is sufficiently severe to rupture also the posterior limiting membrane, then the cord lesion associated with the fracture *per se* may overshadow the relatively minimal signs and symptoms of the displaced nucleus.

Anatomy and Practical Considerations for Fractures of the Bodies of the Mid-Dorsal Vertebræ.

The facts previously discussed pertaining to the dorsal spine are pertinent factors in the explanation. A study of Fig. 1, a schematic drawing of a vertical hemisection of the spinal column, shows that the usual area of fracture following metrazol injections is in the mid-dorsal spine, about midway between the cervical and lumbar curves. The dorsal spine is but a slightly flexible column so that with the usual application of force, it moves more or less as a unit, and fractures are, therefore, more usual at the cervico-thoracic or dorso-lumbar areas and are more likely associated with acute hyperflexion, or the so-called "jack-knifing" attitude.

The application of force for the clonic convulsions following metrazol injections differs from that force usually associated with fractures of the spine, in that there is repeated traumatism from a terrific impacting, pounding and compacting due to forcible muscular contractions. The greater strength of the flexor muscle chains, the leverage of many of which are well forward from the spine itself, results in a maximum application of force which occurs with the trunk in an attitude of slight flexion. In the dorsal spine the vertebral bodies are broader anteriorly and the discs are more narrow anteriorly. This again suggests why the fractures have been observed as a wedging or crushing downward of the anterior part of the body of the vertebræ. Further, the fractures occurred

in the mid-dorsal region because the vulnerable area of a slightly flexible column may well be its center; this area is the dynamic center of the flexor muscles of the trunk; and according to Fick the fifth dorsal vertebra is the weakest in the entire spine. Conversely, the lower thoracic and lumbar spine is not injured because the degree of flexion is not sufficient, the vertebræ are larger and stronger, and the vertebral discs are larger and more flexible. Again, when fracture occurs higher in the vertebral column it probably breaks the effects of the force being applied or transmitted. Another indication that the fifth and sixth areas may be the site of undue strain in the thoracic spine is that the only cases of ruptured nucleus pulposus with protrusion [8] occurred here, other than at its lower extremity.

Prophylaxis for Spinal and Lower Extremity Injuries.

The problem of preventing vertebral body injuries, as well as other injuries to the bones and joints, is of great interest and importance, if the use of metrazol therapy is to continue unchallenged because of attending complications to those parts of the anatomy not previously affected by the patient's original disease or disability. It may not be in the province of the orthopedic surgeon to discuss the merits of metrazol injections in the treatment of mental diseases. Suffice it to say that these treatments enjoy wide usage, and benefits have been observed and remissions reported.

After observing the convulsions in a number of patients receiving metrazol, it may definitely be stated that no type of mechanical fixation can prevent injury to the vertebral bodies and be otherwise practical. The degree of flexion and extension associated with the spasms is certainly not sufficient to be the primary factor in causing the fractures, which are due to forcible muscular contractions acting upon an area which is anatomically weak and which is exposed to the type of stress and strain which is peculiar to the convulsions following metrazol injections. It is impossible to immobilize the trunk so that no motion (flexion or extension) of the deeply seated spine is possible. Again, muscle action continues even though the origin and insertion of muscles do not approach

[8] Walsh, N. M., and Love, J. G.: The Syndrome of the Protruded Intervertebral Disc. Proc. Staff Meet. Mayo Cl. 14: 230 (Apr. 12), 1939.

each other. (A well known example of this is the ability to make the patella taut by contracting the quadriceps muscle with the leg fully extended.) Even if an apparatus could be applied so efficiently as to prevent flexion or extension in the vertebral column, muscular contractions still could not be controlled; and because of the tremendous strength of the muscular action, a muscle, ligament, disc or bone would in all probability be injured. Potentially, the resulting trouble might be more severe, since the fractures in the vertebral body which now occur may relieve the tension or effects of the force upon other structures and protect them from injury. Again, disabling results have not been reported as a result of fractures in the bodies of the vertebræ following metrazol injections.

Hamsa and Bennett [9] have suggested and used spinal anesthesia, given one-half to one hour prior to the metrazol injections, as a means of causing a complete though transient motor and sensory paralysis of all the thoracic and lumbar segments, so that the convulsive shock ensues without contractions of the anesthetized areas. It is their opinion, and that expressed under "Current Comment" [10] in the *Journal of the American Medical Association,* that prophylactic treatment by spinal anesthesia offers no serious drawback and that this treatment may aid to prevent fractures of the spine and lower extremities. It then only becomes necessary to control the head to prevent a dislocation of the jaw and to control the upper extremities by keeping them adducted during the convulsive stage of metrazol therapy, in order to prevent bony injury. An apparatus to act as an aid in preventing shoulder dislocations [11] and other upper extremity disabilities has previously been described.

Orthopedic Treatment for Fractured Vertebral Bodies.

If fractures occur, fixation of the trunk in a plaster jacket, with the patient ambulatory, for a period of six months will probably

[9] Hamsa, W. R., and Bennett, A. E.: Traumatic Complications of Convulsive Shock Therapy. Jour. A.M.A. CXII: 2244 (June 3), 1939.

[10] "Current Comment." Fractures of the Spine During Metrazol Therapy for Dementia Præcox. Jour. A.M.A. CXII: 2293, (Jun. 3), 1939.

[11] Rechtman, A. M., and Winkelman, N. W.: The Prevention of Shoulder Dislocations During Convulsive Therapy in Schizophrenia. Penna. Medical Journal, p. 39, Vol. 43, No. 1, October, 1939.

result in healing. If wedging of the fractured vertebral body or bodies is present, an attempt to correct the deformity by hyperextending the trunk (Davis) should first be practiced. Certainly a fracture of the vetebral body, mild in degree, in the midthoracic area, and amenable to treatment, is easier to treat and of less serious moment than the psychotic state for which metrazol is now being used.

Patients complaining of backache should be carefully studied, clinically and roentgenologically, for evidence of fracture. If a fracture occurs, at the first or with subsequent injections, then repeated or continued convulsive therapy may tend to aggravate the original injury. A traumatic hyperemia with decalcification occurs. Then unprotected weight bearing, superincumbent weight and muscular action tend to increase the wedging of the vertebral bodies with a resulting increase in the deformity (kyphos). If fracture occur during the course of treatment, then the patient may be kept on a convex stretcher frame, or fixation may be maintained by the use of a plaster, leather or celluloid corset. Such support should be removed when giving subsequent metrazol treatments. When the course of treatments has been completed the application of a plaster jacket, to be worn until healing is completed, may protect the patient from an increase in the wedging of the fractured vertebral bodies. Spinal anesthesia as a preliminary to the remaining metrazol injections is naturally indicated and recommended. The neurologist should also decide if metrazol therapy should be stopped. Complete recovery, orthopedically, with healing of the fractured vertebræ should obtain when adequate treatment is instituted.

Summary and Conclusions.

1. A study of the anatomy and of the practical considerations of the spine shows that fractures of the vertebral bodies are usual at those areas of the spine that are mechanically weak or exposed to the effects of traumatism.

2. Metrazol acts to cause convulsions attendant with very forcible muscular contractions sufficient to fracture the bodies of the vertebræ in the mid-dorsal region which is anatomically and dynamically vulnerable. The localization differs from the more usual sites of fracture because of a modification of the type of

stress and strain peculiar to the muscular contractions caused by metrazol injections and their effects on the anatomy of the parts affected. The degree of injury varies directly with the force of the muscular contractions and inversely with the resistance offered by, or the strength of, the vertebral bodies. The force of the muscular contractions and the energy expended is so great that a convulsion continuing for 90 seconds may exhaust the patient for 8 to 24 hours.

3. The mid-dorsal area of the spine is anatomically vulnerable and fractures occur at this site following metrazol therapy because the site is midway between the forward physiological curves of the cervical area above and the lumbar below. The degree of flexion occurs through an arc sufficiently small to make the mid-dorsal spine the recipient of its greatest effects. The vertebral bodies are here wider anteriorly while the discs are smaller, less resilient and more narrow anteriorly; and the dynamic center of the flexor contracting force of the trunk muscles is at this level.

4. The number of metrazol injections has no relation to the number of vertebral bodies fractured or to the degree of wedging. The extent and degree of injury are rather dependent upon the intensity of the muscular contractions, due to any one particular convulsive treatment, and the strength of the spine to withstand the shock. Undoubtedly if a fracture occurs early in the course of treatment and if treatments continue without the prophylactic measures recommended, increased wedging of the vertebræ may obtain.

5. Rupture of the nucleus pulposus with protrusion into the neural canal is most unusual following fractures of the vertebral body.

6. No practical type of mechanical fixation can prevent injury to the vertebral bodies.

7. Prophylaxis is possible, and the use of spinal anesthesia given prior to the metrazol injections as suggested by Hamsa and Bennett is recommended.

8. If fracture occur, immobilization of the trunk between treatments and reduction of the deformity, if it is present, with fixation in a plaster jacket for six months after the treatments are completed should result in entire healing of the fractures.

MUTISM AND RESISTANCE BEHAVIOR IN PSYCHOTIC PATIENTS.

A Physiologic Study.*

By CHESTER W. DARROW, Ph. D.,
AND
ALFRED P. SOLOMON, M. D.†

Psychologic resistance frequently characterizes psychotic behavior. The various manifestations of resistant attitudes are familiar to all psychiatrists. When psychotic patients are presented with a question pertinent to their emotional conflicts they may avoid evaluation of the idea by evasive replies, ambivalent responses, perplexity, or confusion; they may attack the examiner with argumentativeness, hypercritical attitudes, and overt anger; or they may successfully avoid discussion by sullenness, obstinacy, or even mutism. In a previous publication [1] we demonstrated that in some cases effective pharmacologic treatment resulted in the change of resistant attitudes to attitudes of cooperation permitting frank discussion of the patients' problems. It was shown that this was attended by significant changes in autonomic response to ideational stimuli. In the present paper we confirm our previous conclusion by demonstrating similar differences between the autonomic responses of resistant untreated and cooperative untreated patients, and in addition we present similar data differentiating patients presenting various types of mutism.

In Fig. 1, we present sample physiologic records of the type on which this presentation is based. B. P. indicates blood pressure,

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† From the Psychiatric Institute, the University of Illinois, and the Institute for Juvenile Research, Chicago, Series C, No. 309.

We are indebted to Drs. H. Douglas Singer and Paul L. Schroeder for rendering the facilities respectively of the Illinois Psychiatric Institute and the Institute for Juvenile Research available for this research. We are also indebted to the Illinois Department of Public Welfare, to members of the staff who gave assistance, and to Mrs. M. Wright under W.P.A. for stenographic aid.

R. P. the record of galvanic (sweating) responses of the right palm, R. B. is from the back of the same hand, and Resp. is respiration. The stimuli are briefly indicated. Vertical lines represent second marks. The three records illustrate responses by three clinical types in our series. The record labeled "improved" shows good reactivity of the galvanic skin reflex and blood pressure. The one labeled resistance mutism shows poor reactivity of the galvanic response except after a sensory stimulus and fair reaction of blood pressure. The record labeled indifference mutism shows little response to crucial ideational stimuli by either galvanic or blood pressure mechanisms. A sensory stimulus was again effective.

In Fig. 2 we represent the reactions of a group of overtly hostile resistant patients by circles and of cooperative improved patients by crosses on the autonomogram.[1] The blood pressure changes are represented on the ordinates and the galvanic reactions on the abscissa. The circles and crosses falling within the respective squares have been arbitrarily grouped within the squares to assist visual differentiation. It will be noted that the circles fall toward the left showing small or zero galvanic responses whereas the reactions of improved or cooperative patients fall toward the right with predominately moderate or large galvanic responses. Blood pressure reactions are about the same in the two groups. This is almost identically the same difference in response reported previously, before and after successful pharmacologic treatment of psychotic patients when there was a change from resistant hostile attitudes to attitudes of cooperation and readiness to discuss and understand their problems.

Fig. 3 deals with that group of resistant patients, who, when questioned about their emotional conflicts, gave replies (indicated by the circles), without hostility yet with evasiveness, ambivalence, perplexity or confusion or momentary periods of silence. We regard such verbal responses as indicative of a deep resistance to the consideration of their conflicts. It will be noted that the general trend toward decreased physiologic response is not as great as in the overtly hostile group of the preceding slide yet this tendency is evident. In a previous publication [1] dealing with more confused patients the reactions before pharmacologic treatment were similar to those of the overtly hostile group.

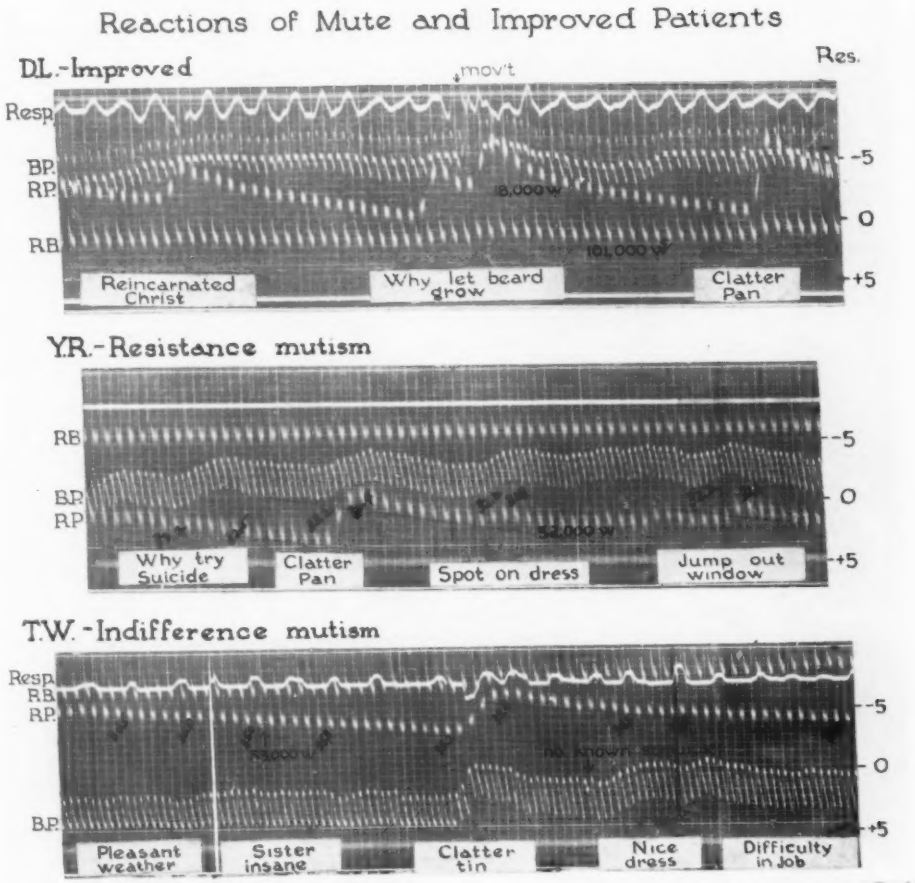

Reactions of Mute and Improved Patients
D.L.-Improved
Res.
mov't
Resp.
B.P.
R.P.
R.B.
18,000 w
101,000 w
-5
0
+5
Reincarnated Christ
Why let beard grow
Clatter Pan
Y.R.-Resistance mutism
R.B.
B.P.
R.P.
32,000 w
Why try Suicide
Clatter Pan
Spot on dress
Jump out window
T.W.-Indifference mutism
Resp.
R.B.
R.P.
B.P.
no known
Pleasant weather
Sister insane
Clatter tin
Nice dress
Difficulty in job

FIG. 1.


Blood Pressure Change

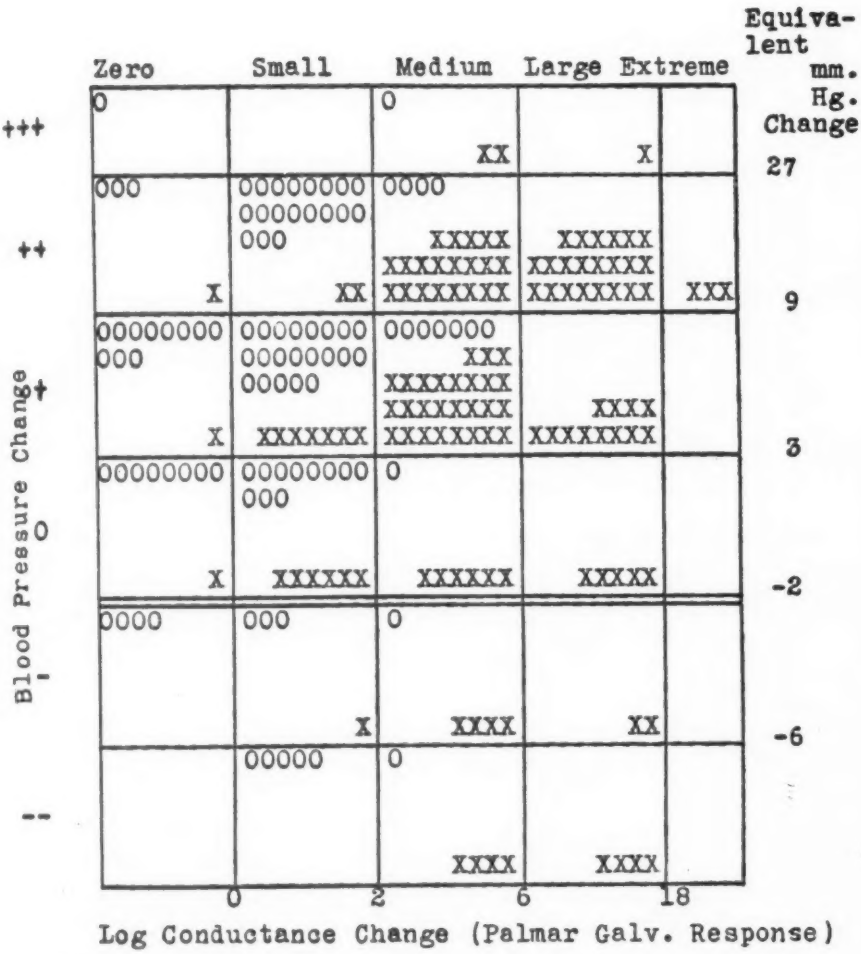


FIG. 2.—Hostile Resistant Patients Contrasted with Improved Cooperative Patients, No Pharmacologic Treatment.

Reactions to crucial ideational stimuli.

O = Reactions of 5 resistant non-paranoid patients.
X = Reactions of 10 improved cooperative patients.

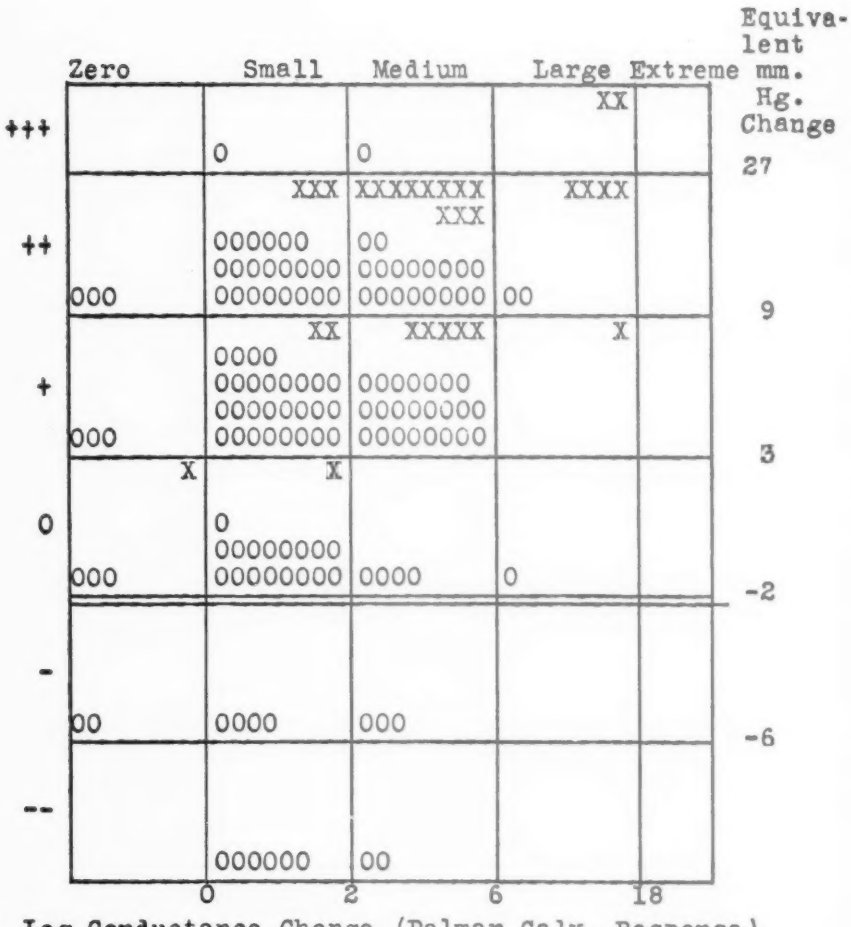


FIG. 3.—Untreated Patients with Resistance Perplexity Contrasted with Paranoid Patients.

Reactions to crucial ideational stimuli.

O = Resistance perplexity—9 patients.
X = Paranoid—3 patients.

We have contrasted the reactions of this type of patient, in whom there is apparently a blocking to consideration of their problems, with the reactions (indicated by crosses) of three paranoid patients who presented an absence of blocking to discussion of their problems and who were aggressive, uninhibited and outgoing in their expression. Their anger was not due to resistance toward the discussion of their problems, or to hostility toward the examiner, but was directed toward the objects of their projections. We have observed similar physiologic reactions in certain manic patients. Apparently the differential point is the presence of uninhibited behavior. It will be noted that the X's tend to fall toward the upper right hand corner with large blood pressure and large galvanic responses. While these observations characterize these three patients, obviously we do not make this as a characterization of paranoid patients in general.

Resistance Mutism.

It is common experience in the treatment of psychoneurotics that resistance may take the form of silence. In psychotic patients mutism is often associated with other manifestations of negativism. In the preceding communicative case groups ample criteria were available in the verbal responses for the recognition of psychologic resistance. Obviously such criteria are lacking in the mute cases although we did have evidence that these patients were or were not showing affect in their motor behavior and facial expression. Further confirmatory evidence was afforded from the study of their behavior in the ward.* From these observations we were able to divide our 25 mute cases into four groups: (1) Resistance mutism without tension or anxiety; (2) resistance mutism with tension or anxiety; (3) mutism associated with passive submissive attitudes; and (4) mutism as a manifestation of indifference.

In the mute resistant group such overt signs of resistance as scowling, biting of lips, clenching of teeth, turning the head away, closing eyes, blinking, suppressed laughter, swallowing movements, grunts, groans, and sighing, were observed in response to our

* The groups could not be differentiated by the presence of selfishness, stinginess, neatness, orderliness, seclusiveness, or obstinacy in childhood, as instances of these traits were found in all groups.

questions. In addition, all of these patients resisted, in varying degrees, coming to the laboratory, lying down on the examining couch, or the application of the apparatus. Their psychiatric history revealed a lack of cooperation in ward routine, and combative-

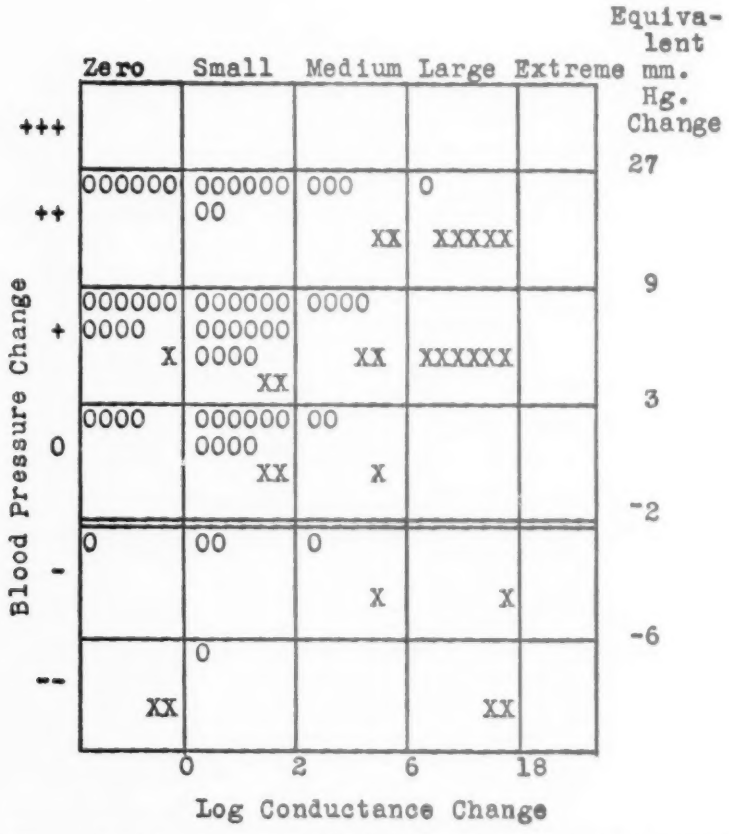


FIG. 4.—Reactions to Crucial Ideational Stimuli by Mute Resistant and Mute Passive Patients.

O = Reaction of 9 mute, resistant patients.
X = Reaction of 3 mute, passive, cooperative patients.

ness,—active resistance to nursing care and feeding. During periods when they were communicative they manifested verbal anger and irritability. The autonomic responses of these patients will be seen by Fig. 4 to be practically identical with those of the com-

municative resistant groups shown in Fig. 2, with the reactions (indicated by the circles) toward the left of the autonomogram.

These reactions of resistant mute patients are contrasted in this figure (4) with a group of mute patients having an absence of this resistant behavior who are characterized as *passive*. They were acquiescent and submissive to the test procedure. In one patient there was complete relaxation of the musculature approaching cataplexy, and in another there was true waxy flexibility with typical catatonic posture. Common to all three patients to a marked degree was the presence of passive feminine masochistic traits in their prepsychotic personality and the absence of aggressive behavior on the ward during the psychoses. The reactions of these patients (indicated by the X's) are to be noted on the right side of the autonomogram indicating good physiological reactivity to the questions. These are practically identical in distribution with those of passive communicative psychotic patients given pharmacologic treatment in our previous study.

Fig. 5 shows reactions of patients with resistance mutism as in the preceding group but with the complicating factor of tension or anxiety. These patients showed the signs of overt resistance to the test procedure as in the preceding resistant group but to a lesser degree. The presence of anxiety was determined by the existence in their facial expression of the apparent affect of fear, pleading, sorrow, and depression, by tension of their long muscles, absence of spontaneous muscular activity, clenching of hands, and profuse palmar perspiration. These symptoms were often accentuated by the crucial questions. Psychomotor retardation, feelings of guilt, self condemnation and anticipation of punishment were evident in all but one of these patients during periods of communicativeness. It will be noted that reactions of this group, indicated by the circles, are slightly larger than in the case of the more inhibited, more resistant patients of the preceding figure. Three of these patients were given sodium amytal during the test. Evidence of tension was then reduced and the patients became communicative and relaxed.

The reactions of patients under tension are to be contrasted with those of indifferent patients. The reactions of these patients indicated by the diagonals show predominatingly zero blood pressure

change and small galvanic responses. The indifferent group showed also by their overt behavior that they were not responding to the ideational stimuli. They appeared to be completely out of contact

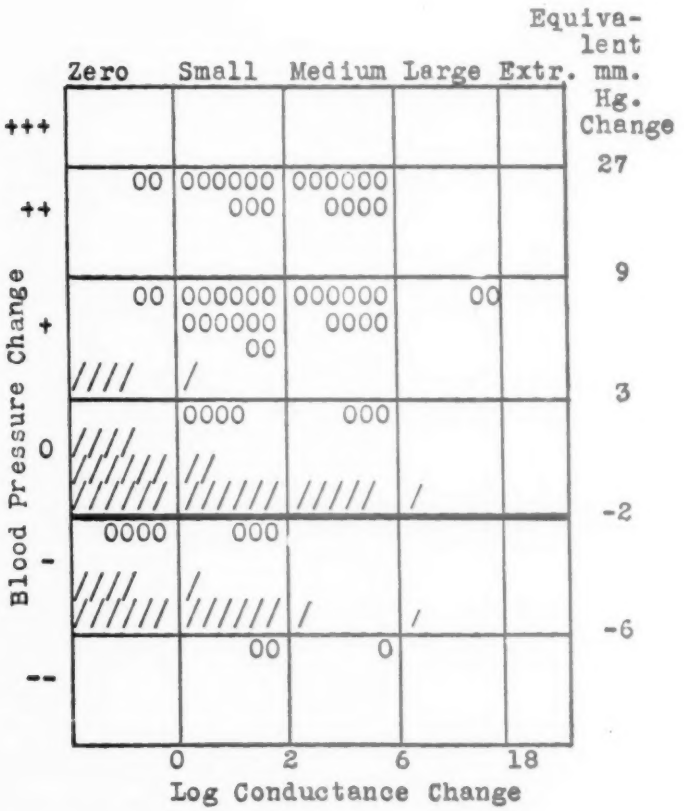


FIG. 5.—Contrasting Reactions of Patients Having Mutism with Indifference and Mutism with Tension.

Reactions to crucial ideational stimuli.

/ = Reactions of 7 mute patients with "indifference."
O = Reactions of 6 mute patients with "tension."

save for occasional responses to sensory stimuli. The absence of observable affect and measurable physiologic responses indicates that stimuli were ineffective. We designate this *indifference mutism.*

Table 1 provides an opportunity for statistical evaluation of the data presented in the preceding figures. It may be seen that the reactions of the improved patients compared with those of the resistant communicative patients show a difference, which when

TABLE 1.

AVERAGE POSITIVE BLOOD PRESSURE AND GALVANIC REACTIONS.

(Figures Represent Numerical Values of Cells in Autonomograms. Log Conductance and mm. Hg. Equivalents Are Indicated.)

Group.	N	X Ave. galv. resp.	Equiv. log cond. change.	Y Ave. B. P.	Equiv. mm. Hg. change.
1. Improved	117	2.20 ± 1.59	(4.5)	1.34 ± 1.05	(8.0)
2. Resistant to Examiner	87	0.89 ± 0.73	(0.9)	1.12 ± 0.91	(6.7)
Difference		1.31 ± 0.17		0.22 ± 0.14	
Critical ratio		7.7		1.6	
3. Paranoid	30	1.97 ± 1.46	(3.6)	1.67 ± 1.22	(12.1)
4. Resistance perplexity	126	1.34 ± 1.02	(1.7)	1.11 ± 0.96	(6.1)
Difference		0.63 ± 0.21		0.56 ± 0.23	
Critical ratio		3.0		2.4	
5. Mute passive ...	21	2.24 ± 1.40	(4.8)	1.19 ± 0.92	(7.1)
6. Mute resistant ..	64	0.63 ± 1.04	(0.7)	1.03 ± 0.75	(6.2)
Difference		1.51 ± 0.34		0.16 ± 0.24	
Critical ratio		4.45		0.7	
7. Mute with tension	56	1.40 ± 1.05	(1.8)	1.25 ± 1.02	(7.5)
8. Mute with indifference ...	30	0.63 ± 0.53	(0.7)	0.14 ± 0.24	(3.8)
Difference		0.77 ± 0.76		1.11 ± 0.14	
Critical ratio		1.0		7.8	

divided by the probable error of the difference gives a critical ratio of 7.7 in the case of the galvanic responses, and of only 1.6 in the case of the blood pressure. The comparison of the reactions of the paranoid with those of the perplexed resistant patients gives a critical ratio of 3.0 for the galvanic and 2.4 for the blood pressure

reactions. Comparison of the mute passive with the mute resistant responses gives a critical ratio of 4.4 in the case of the galvanic responses and practically no difference for blood pressure. In the case of the mute (resistant) tense group and the mute indifferent group there was little difference, for galvanic responses of both groups are small. Blood pressure reactions were, however, larger in the tense than in the indifferent group, the critical ratio being 7.8. Other comparisons might also be made showing differences between the reactions of the resistant, non resistant, and indifferent groups but these would only present further elaborations of the type already shown.

Fig. 6 shows, plotted on a single autonomogram, circles representing the average reactions of the various groups.* The small galvanic and blood pressure reactions of the indifferent group are shown to the lower left. The reactions of the resistant groups 2, 4, 6 and 7 are shown by circles in that part of the figure representing small galvanic and moderate blood pressure rise. The various non-resistant groups 1, 3 and 5, including improved, passive, and paranoid patients are shown by circles grouped farther toward the right with larger galvanic responses. The two squares on the autonomogram represent data from a previous study on pharmacologically treated resistant patients who became cooperative following treatment. Again reactions are distributed toward the left in the presence of resistance behavior and toward the right after improvement or recovery. As shown by Table 2 the difference between the average of individual scores by 33 resistant patients, whose reactions are represented by circles 2, 4, 6 and 7, and the scores of 17 individuals whose reactions are represented by circles 1, 3 and 5 gives a critical ratio of 12.7 for the galvanic and 4.8 for the blood pressure changes.

Discussion.

This paper is one of a series in which we present the concept that resistance behavior can be observed in psychotic patients and, furthermore that it can be correlated with concomitant physiologic

*Averages are determined after elimination of reactions involving a negative B. P. change as explained in preceding communication.[1]

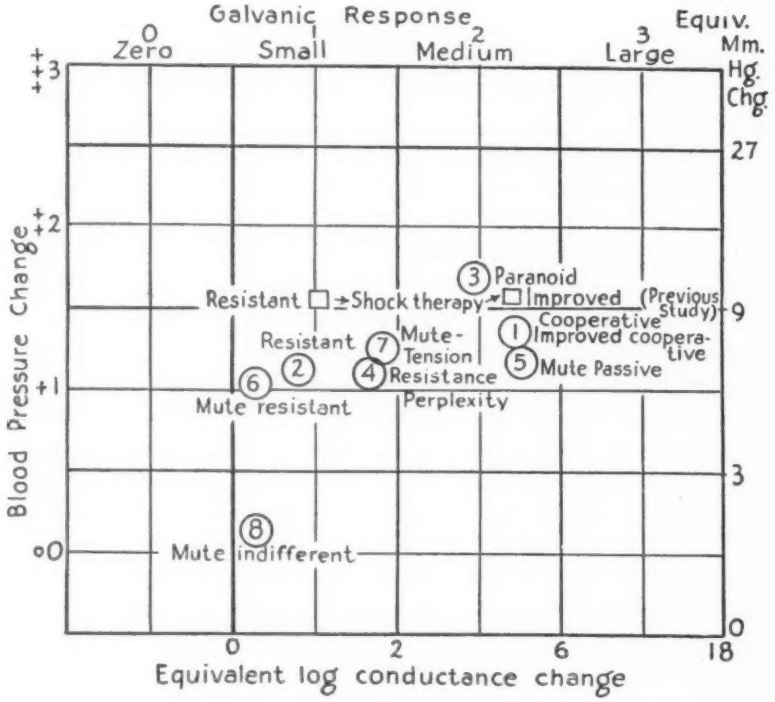


FIG. 6.—Average Galvanic and Blood Pressure Response of Different Groups of Patients.

TABLE 2.

VARIABILITY OF INDIVIDUAL AVERAGES OF RESISTANT AND OTHER PATIENTS.

(Reactions with Minus B. P. Omitted.)

	Galvanic response.	Blood pressure response.
33 resistant individuals, groups 2, 4, 6 and 7	1.04 ± 0.262	1.05 ± 0.269
17 individuals, groups 1, 3 and 5	2.11 ± 0.288	1.41 ± 0.233
Difference	1.07 ± 0.084	0.36 ± 0.075
Critical ratio	12.7	4.8

measures. Our earlier study read at this society last year offered evidence that when patients were studied during the manifestation of their resistance behavior and again after the disappearance of this behavior following pharmacologic treatment there was a significant change in the galvanic and blood pressure responses. It was hypothecated that the reduction of the galvanic responses in the presence of the rises in blood pressure was evidence of inhibition of parasympathetic or cholinergic activity associated with decrease of sympathetic activity. In the development of this theory it was pointed out that these inhibitory effects may be either neural or humoral. For example, adrenalin secreted by the resistant hostile patients may result in inhibitory effects on the autonomic system through the action of homeostatic, especially carotid sinus, mechanisms. Hoskins and Rowley,[2] and Hsu and Chu [3] have shown inhibitory effects of adrenalin on circulatory mechanisms. Physiologic evidence of the inhibitory effect of adrenalin on other autonomic mechanisms, both sympathetic and parasympathetic, has been obtained in recent animal experimentation in association with Dr. E. Gellhorn of the University of Illinois College of Medicine.[4] Marazzi has also recently reported inhibition of nerve impulses in the sympathetic ganglion by adrenalin.[5] Darling [6] recently has offered evidence that autonomic activity and behavior of children in a behavior clinic are related in a manner quite similar to that here shown in psychotic patients.

Summary.

This paper presents a psychiatric and physiologic study of eight groups of patients. Four groups of communicative patients offer confirmation of the difference in physiologic response associated with resistant and nonresistant behavior.

In addition we offer data on mute patients where the study of the behavior during the laboratory procedure and in the ward enabled us to differentiate four groups, resistance mutism, resistance mutism with tension or anxiety, mutism associated with passive submissive attitudes, and indifference mutism.

The problem of determining the presence of psychologic resistance in the absence of verbal replies has been outlined. Experience has shown that it is possible to gain an impression of the patient's

attitude not, however, infallible, from his affective reactions to the examiner. When such observations are evaluated it is believed to be possible to distinguish resistance, passive masochistic submission, and indifference.

The resistant mute cases, with overt affect at the time the questions were asked, clearly indicated that these patients were emotionally reacting to our stimuli. The hostile nature of the resistance was evidenced by the character of the overtly manifested affect and the patients' antagonism to the test procedure. The indifferent patients appeared to be wholly out of emotional contact except for isolated instances in which they reacted selectively to certain ideational or some sensory stimuli. It may be inferred that the indifferent patients were either (1) completely absorbed in their own autistic thinking or (2) that the capacity for physiologic response was impaired or (3) that perceptual mechanisms were not functioning.

The physiologic study of these four groups of mute patients shows that with the resistant cases there is a predominance of smaller galvanic responses in the presence of definite blood pressure reactions suggestive of neural or humoral inhibition of autonomic responses; with the passive submissive patients there are strong reactions by both the galvanic and blood pressure mechanisms suggestive of uninhibited autonomic response, and with indifference mutism both responses tend to be small, suggestive of diminished autonomic activity.

BIBLIOGRAPHY.

1. Solomon, A. P., Darrow, C. W., and Blaurock, M.: Blood pressure and palmar sweat (galvanic) responses of psychotic patients before and after insulin and metrazol therapy. Psychosomatic Med., 1: 118-137, 1939.
2. Hoskins, R. G., and Rowley, W. N.: The effect of epinephrin infusion on vasomotor instability. Amer. J. Psychol., 37: 471, 1915.
3. Chu, L. W., and Hsu, F. Y.: The effect of adrenalin on vasomotor reflexes. Quart. J. Exp. Physiol., 27: 307, 1938.
4. Darrow, C. W., and Gellhorn, E.: Inhibitory effects of adrenalin on autonomic function. Proc. Soc. Exper. Biol. and Med., 40: 487-488, 1939.

———: Antagonism of adrenalin to the autonomic effects of metrazol. Proc. Amer. Physiol. Soc. (Amer. J. Physiol., 126: 474, 1939).

———: The effects of adrenalin on the reflex excitability of the autonomic nervous system. Amer. J. Physiol., 127: 243-251, 1939.
5. Marrazzi, A. S. Inhibition at a sympathetic synapse. Proc. Amer. Physiol. Soc., 1939 (Amer. J. Physiol., 126: 1939).
6. Darling, R. P., Autonomic action in relation to personality traits of children. J. Abn. and Soc. Psych. 35: 246-260.

DISCUSSION.

Dr. Oskar Diethelm (New York).—In this paper we have an attempt to single out special reactions instead of being guided by large disease entities. The authors use the term, "resistance," in a well-defined way. Throughout the paper, definite and concrete language is used so that one is able to understand clearly what was done and why conclusions are drawn. This allows the listener to arrive at his own conclusions which may be different from those of the authors. I do not think, for example, that I can agree with the final interpretations.

In previous physiological studies, *i. e.*, until about ten years ago, studies were carried out on large "disease entities." It was accepted that one dealt with definite types of illnesses, whereas in this study, the reactions as such are investigated wherever they may occur.

A third point I want to stress is the continuation of various physiological methods. Through Dr. Darrow, an apparatus is available which allows us to take permanent blood pressure readings, and to register at the same time respiration and skin responses. This possibility of attacking a psychobiological problem from various points physiologically is an important step forward in physiological studies in the field of psychiatry.

The paper was a most interesting one, and I was happy to be invited to discuss it because it shows careful work described in psychological terms which one can understand, and formulated so clearly that we all can draw our own critical conclusions.

Dr. Alfred P. Solomon.—We are indebted to Dr. Diethelm for his discussion. I am glad he has emphasized the futility of attempting to find correlation between physiological changes and psychiatric "disease entities." For quite a number of years we have recognized the necessity for an adequate description of the behavior as well as of the physical state of the patient at the time of the test. It is obvious that physiological changes in the patient's body are taking place from moment to moment and hour to hour along with changes in his attitudes and that this must be taken into consideration in the interpretation of physiological reactions to stimulation. It is my conviction that what we need in psychophysiological research is more attention to identification and description of the dominating attitudes of the patient as the time of his physiological response to stimulation.

METRAZOL AS A DIAGNOSTIC AID IN EPILEPSY.*

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Epilepsy is readily diagnosed in cases where the history is definite as to occurrence of a seizure of the grand mal type; however, the question of the diagnosis of epilepsy where no history of convulsive seizures is available has made the use of diagnostic tests to determine the presence or absence of convulsive tendencies a distinct advantage depending on the reliability of such tests. Hans Werner Jans [1] in a recent investigation of the methods of testing epilepsy used to date indicated that Foerster's hyperventilation test could induce seizures in only 10 per cent of true epileptics. Muck's spraying of the radial or carotid arteries with ethyl chloride failed to produce seizures in one of ninety verified cases of epilepsy. Using three to six cubic centimeter doses, Jans was able to induce a seizure in 18 of 33 cases injected intravenously with metrazol; however, in 38 controls he obtained 14 seizures. Using McQuarrie's fluid retention test, he obtained seizures in 29 of 69 known epileptics and none in 40 non-epileptic controls. He concluded that the water retention test was a definite aid in the diagnosis of epilepsy.

Many articles have been written on the diagnostic applicability of metrazol injections. Kruger [2] stated that doses non-convulsant for schizophrenic patients (one to one and five tenths cubic centimeters) may be found helpful in differentiating hysteria and epilepsy. Several other investigators have used intravenous injections of metrazol as a diagnostic test. Schönmehl,[3] Duensing,[4] Grubel,[5] Hoyer,[6] Langeluddeke,[7] Muyle,[8] have varying opinions as to the diagnostic applicability of an intravenous injection of metrazol but all apparently feel that it has some value.

In a study of the effects of subcutaneous injections of metrazol as a sympathetic stimulant, it occurred to us that some of the data

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obtained could be applied to an investigation of the value of metrazol as a diagnostic test for epilepsy.

Method of Study.

A group of non-epileptic patients were selected as controls and were given a 10 per cent solution of metrazol subcutaneously in the inner aspect of the thigh and distributed so as to permit ready absorption.

Nine patients diagnosed as having a schizophrenic psychosis were given eight cubic centimeters of metrazol per 100-pound body weight (45.5 kilograms). In nine other patients the dosage was reduced to six cubic centimeters. Eight of these patients were diagnosed as hebephrenic dementia præcox and one as a paranoid dementia præcox. Eighty-four patients were given five cubic centimeters of metrazol per 100-pound body weight (45.5 kilograms). The diagnoses as made by the staff of the institution were, hebephrenic dementia præcox, 61, catatonic dementia præcox, 13, paranoid dementia præcox, seven, manic-depressive psychoses manic phase, one, undiagnosed, one.

The procedure of giving a subcutaneous injection of 10 per cent metrazol was repeated on a group of epileptic patients with the dosage being kept at five cubic centimeters per 100-pound body weight (45.5 kilograms). A total of 34 such cases were treated of whom 15 were male and 19 were female.

No normal individuals were used as controls but Schönmehl [8] made the statement that in normal individuals, the convulsive dose is 10 cubic centimeters of a 10 per cent metrazol solution intravenously, while in schizophrenia the dosage was given as five to seven cubic centimeters.

Results.

The control group, which consisted of female patients only, reacted to the subcutaneous injections as follows: of the nine patients given eight cubic centimeters of metrazol, two developed grand mal seizures. In nine others, a dosage of six cubic centimeters was used and no seizures were produced. On five cubic centimeters, one of 84 patients developed a grand mal seizure. On examining

the history of this patient, it was found that she had shown retarded mental development and finished six grades of school at the age of 17. The history and course of the disease were not characteristic of schizophrenia.

The following results were noted in the epileptic group when the procedure described above was carried out. Of the 19 female patients, 10 developed one or more grand mal seizures and of the 15 male patients, six responded with epileptic discharges of a major type. A table of relative statistics might clarify the results of the study.

Number of patients.	Diagnosis.	Metrazol doses per 100 lb. (45.5 kilos).	Number of patients having seizures.	Per cent patients having seizures.
84	Functional psychosis	5 cc.	1	1.19
34	Epilepsy	5 cc.	16	47.05

When the relative percentages of convulsions produced by a standardized dose of metrazol given subcutaneously, in epileptic and non-epileptic patients are compared, it becomes evident that the method forms a rather effective means of determining the presence of epileptic tendency in a patient. Using five cubic centimeters per 100-pound body weight (45.5 kilograms) of a 10 per cent solution of metrazol subcutaneously as a standard dose, the authors feel that the production of a seizure is presumptive evidence of a convulsive tendency. The presence of organic brain disease should be ruled out before deciding that epilepsy is present. Failure to produce a seizure does not exclude the presence of epilepsy.

Conclusion.

Differences of convulsive reactivity are manifested when a stimulating agent to the central nervous system is given to a group of individuals.

In 84 patients suffering from functional psychoses and with a standard dose of metrazol used as a stimulant, 1.19% developed a seizure. The one patient having a convulsion was quite possibly incorrectly diagnosed. Of 34 epileptic patients, 47.05 per cent developed seizures on a dose equal to that for schizophrenic and other patients.

Patients developing grand mal seizures with a dose of five cubic centimeters or less per 100-pound body weight (45.5 kilograms) have an increased irritability of the nervous system and epilepsy is to be suspected in the absence of any evidence of organic disease of the brain.

BIBLIOGRAPHY.

1. Yearbook Neurology and Psychiatry. 1937.
2. Kruger, L.: Treatment of schizophrenia by Meduna's method. Psych. Neurol. Wchnschr., 38: 135, 1936.
3. Schönmehl: Induced epileptic convulsions—experiments and possibilities. Muench. Med. Wchnschr., 83: 721, 1936.
4. Duensing, F.: Is the diagnostic use of metrazol convulsion permissible? Muench. Med. Wchnschr., 84: 1011 (June 25) 1937.
5. Grubel, R.: The value of metrazol convulsions in hereditary social hygiene. Muench. Med. Wchnschr., 84: 469 (March 19) 1937.
6. Hoyer, C.: Metrazol test. Psych. Neurolog. Wschr., 39: 541 (Nov. 29) 1937.
7. Langeluddeke, A.: The diagnostic importance of experimentally effected convulsions. Deutsche Med. Wchnschr., 62: 1588, 1936.
8. Muyle, G.: Metrazol as a diagnostic agent in epilepsy. J. Belge de Neur. et de Psych., 7: 525, 1938.

SOME OBSERVATIONS ON ANANCASM.*

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The term, "anancasm," communicates a number of concepts which deal with compulsive, obsessive and phobic ways of experiencing. Compulsion stresses recurrent, repetitious, meticulous performance, a manner of acting which to the individual is irresistible, inevitable, which usually is against his desire and better judgment. Obsession restricts qualitatively similar processes to the realm of ideas and emotion. Phobia may be defined as the exaggerated, unwarranted dread of some real or fancied situation. Many persons experience phobias in a way that may be obsessive, compulsive or both. Since phobias of all intensities and varieties may drive individuals to "seek protection" through compulsions and obsessions, we believe that they are anancasms.

These definitions make it possible to include, descriptively, the following under the collective term "anancasm": excessive orderliness; meticulousness; exaggerated need for cleanliness; precision; pedantry; perfectionistic strivings of all manner of content and intensity; perseverative tendencies; ruminations; recurrent, stereotyped fantasies; harping on subjects; "sticking" overawareness of bodily functions; hair-splitting argumentativeness; and adherence to timetable-like routine in living, extreme dependence on rituals.

One must at once distinguish anancasms from other forms of repetitious, recurrent, orderly, stereotyped behavior and thinking. We exclude conditioned reflexes because of their automaticity, and the usually temporary and limited repetitions and care exercised in the trial and error method of learning. Whenever habits are accompanied by a "must" element, an "I can't stop it" attitude, or when they are partially or completely blocked, then increasing anxiety and tension result. Habits then are no longer habits but have passed into anancasms. Some habits, verging on anancasm, are still habits if they occur at a certain age and are later easily discarded as the individual enters another phase of development.

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Thumb-sucking is an example—this is commonly seen in infants. Later, persistent thumb-sucking is usually no longer a habit but rather an anancastic way of experiencing emotional or situational stresses. We shall not include under anancasm repeated fulfillment of recurrent physiologically determined needs, such as hunger, sleep, sexual desire, although even here we must recognize individual differences and rhythms.

Individual anancasms practiced by the group may become socially so accepted that they no longer carry the emotional charge of anancasm. In our culture, dependence on three meals a day and the daily bath as a part of "normal" cleanliness would hardly be criticized as anancastic. In another culture, such particular eating and washing routines, if practiced only by an occasional person, would be considered odd and, being different from the conventional patterns, would very likely be performed in a compulsive fashion. Anancasms lose their strength when such behavior or ways of experiencing become an integral part of the culture. One might say that certain cultures are "anancastic cultures." Individuals are forced to participate in varied, highly routinized, ritualistic or religious group-activities. However, individuals, unless they are anancasts, will take part frequently against their own wishes, in a matter-of-fact manner: such participation may hold little significance. The anancast, however, will not relinquish his anancastic tendencies. He will superimpose his own fastidiousness, rituals or what you will, upon the culturally accepted pattern.

For the present discussion, the term anancasm must be delimited to include only two main groups: (1) constructive anancasms and (2) non-constructive anancasms.

Most human beings seek to satisfy an inner need for arrangement, control or management of the various facets of their personality. Human beings strive to come to some sort of adjustment with themselves and simultaneously to adjust to fellow-men in the cultural group. We find that for some these adjustments are easily accomplished; for others it is a painful, perpetual struggle; for still others it is an almost insurmountable task. For many insecure, anxious, tense, ambitendent, hostile, dependent people, anancasms are "wonderful" stabilizers. Here is a way to have and to hold order in one's inner economy, a way to protect oneself against a hostile world. This type of adjustment demands a highly-developed

sense of routine, temporal and spacial order, meticulousness, a constant check and recheck. Other personality traits as impulse, temperament, intelligence, influence the expression and the experience of this adjustment. The ability to feel and act this way is a constructive force: it keeps the initially not too fortunately harmonized personality traits in working order; it placates anxieties and allays fears. It enables these people to function to some extent in the cultural group. In certain persons, anancasms are efficient stabilizers: they give direction, help to achieve goals and purpose and keep the inner man, so to say, tolerably integrated, constructed. Such anancasms are definite assets. Our culture provides niches for such people; they may work and render peculiar kinds of service which not infrequently are exploited by society.

In contrast, we sometimes see non-constructive anancasms. Under certain conditions, as we shall discuss later, the individual's anxieties and inner turmoil will break through the stabilizing anancastic patterns. We then still observe compulsions, obsessions and phobias, but now of such intensity, of such bizarre content and irrational nature that they become noxious to the individual. They heighten the person's basic insecurities, accentuate personality disharmony, they now superimpose fears and tensions of a new kind. The individual flounders hopelessly, feels torn asunder, as if without means to assemble himself. With alleviation of stressful conditions, with reduction of the basic anxiety through understanding and toleration, we may expect gradual improvement; the non-constructive phase may pass to a constructive, anancastic arrangement. Often individuals cannot achieve such reconstruction; they continue through life tormented, unhappy, ineffective and rejected by the cultural group.

We assume a constitutional "anlage" for anancasm. Anancasms are not observed in any one particular "type" of personality. People with anancastic tendencies frequently have other personality traits and ways of experiencing which cause inner emotional turmoil or conflict. These inner tensions, as well as situational tensions, may enhance anancasms. Indeed the anancast by his very anancasms, often creates his own situational stresses. It is often impossible to separate personality-determined from environmentality-created difficulties.

Anancastic tendencies frequently can be traced through many generations in members of a family group. Homes in which one or more anancasts dominate the scene provide fertile soil for the growth of anancasms of a younger generation.

The scope of this paper does not allow a comprehensive listing of the conditions which enhance the already existing anancastic tendencies, or which change constructive anancasms to non-constructive ones or vice versa.

In general, emotional distresses or situational strains, which raise anxiety and tension, augment anancastic tendencies. More specifically, physical disorders of many kinds, various psychotic states, particularly depressive episodes, may enhance or change types of anancasms. Temporary increases in anancasm frequently accompany stressful periods in the natural course of biological development. The adolescent often seeks to stabilize himself through newly established orders, routines, preoccupations. Groups in late middle age or approaching old age may find increasing need to cling to what has been; their waning faculties grasp or tolerate poorly changes and demands for readjustment. To such people, anancasm may be the way par excellence to hold on to the "has-beens" of another age period. Still another group, comprised of all ages, struggles with so much basic anxiety and insecurity that the only way of life is living through thousands of anancasms, grasping footholds here and there for fleeting moments. Their insecurities again and again demand the protective armament or effective weapons of anancastic behavior and ways of experiencing.

Perhaps short histories of three patients will illustrate best some of the above mentioned points.

CASE I.—Mrs. O is an attractive, well-mannered, outwardly courteous, 29-year-old married woman, the mother of two children. Her ancestors hailed from old New England stock. Many members were "nervous," fussy, "individualists," exceedingly conscientious and rigid. Their sense of duty and honor, above all, has weathered marital incompatibilities, bereavements, financial strains. The family motto has been: superficial conventionality, no matter what inner turmoil; lie in the bed you have made.

The patient was an unhappy child; she felt rejected by her parents, particularly by her mother. She always harbored, but rarely expressed, feelings that she was the least liked of the three daughters, of whom she was the oldest. As a child, she was willful, fussy, demanding, critical, embarrassingly honest and ineptly candid—she made overtures for love and affection, but

always felt unsatisfied. She was dependent on the mother, still is, but since the teen age she rebelled against authority and direction; at the same time she yearned for solicitude and approval. She was a good student, graduated from a well-known woman's college and taught school.

Various phases of sexual development were experienced with feelings of misgiving, shame, disgust, fear and anxiety. In all relationships she was markedly ambitendent, quick to criticize, frequently openly hostile and always had the feeling of being left emotionally unsatisfied, even when relief of sexual tension had been achieved. At 23 she married a man a few years her senior. He has been known to us as a patient: his problems are nuclear in his obsessive thoughts, compulsive behavior and his many phobias. He is a man of high intelligence and training, given to hair-splitting argumentation. He is hostile, vindictive and projects on others his feelings of inadequacy. A previous marriage had been terminated in divorce, and his present marriage has survived through fear-driven dependence on his wife and his attachment for his children. Mrs. O has been most unhappy, has not obtained sexual satisfaction. For many years she submitted to his anancasms, compelled to tolerate the almost intolerable through her intense guilt feelings about pre-marital sexual experiences. Her husband repeatedly held these up to her and rationalized these as the cause of his shortcomings.

In June 1939, the second child was born. The husband felt he had now securely anchored his wife; he neglected her or became overtly hostile and mean. Patient became afraid of her husband, felt increasingly tense and anxious, and following a state of panic was admitted to the clinic. Patient had noticed that her usual fussy, orderly, perfectionistic, domineering, carefully anticipatory behavior had suddenly, in the course of her increasingly difficult home situation, become markedly exaggerated. During the summer she had spent hours arranging bureau drawers. She had feelings of impending disaster unless she knew where every last possession was located. She needed more carefully studied and worked-out routines; she was annoyed by her excessive fastidiousness. She remarked spontaneously that her only sense of security lay in knowing just where everything was and how everything would proceed during the day. She felt too preoccupied to handle any new or unforeseen situation.

With intensive therapy her hate for her husband, her ambitendence to her family, her dependency on her mother, her anxiety, various methods of placating these anxieties, were brought to her attention. After a more realistic appreciation of the situation she improved considerably. Following discharge from the hospital, patient went home for a month and then decided to return to her husband. Later she remarked, "When I got home I realized I was better—I was able to let the suitcases stand in the room for several days before I got around to unpacking the last things. I could not have tolerated such untidiness last summer."

CASE II.—Mrs. L. This 30-year-old, married woman was admitted to the metabolism service of the New Haven Hospital presenting signs and symptoms of severe hyperthyroidism of at least six months duration. Patient's

chief complaint was "mental signs" of about two months duration. She was acutely and distressingly aware of certain new "habits." Each night she would get out of bed, two to three times, to place her bedroom slippers in symmetrical order. The smallest scrap of paper in the yard attracted her attention and had to be picked up and removed. The arrangement of clothes and furniture took up many hours during the day. Suddenly she developed a tremendous fear of dogs; this in marked contrast to a former fondness of these animals. Walking over pavement squares in two steps without stepping on the separating ridges marred all enjoyment of outdoor activity. Staircase climbing presented a problem of stair-counting. She developed many rituals about eating. The three teaspoonfuls of sugar which she had to put into her morning coffee were most annoying—she detested sweetened coffee. Patient was alarmed by this behavior and increasingly feared insanity. No other pathological mental trends could be elicited on examination.

With bedrest and on Lugol's solution, patient improved. Pre-operatively, she could give a clear account of her difficulties; she had full insight that the anancasms had receded with improvement of her general condition. Following subtotal thyroidectomy, only minimal anancasms could be detected. The patient felt free of compulsive and obsessive tendencies.

She was the oldest of five children who with their parents always lived in marginal economic circumstances. At the age of 13 schooling was terminated. For the ensuing 17 years patient took upon herself in a conscientious and oversolicitous fashion the care of the bed-ridden, paralyzed mother. She married at 18. Her husband was irresponsible and left her with two children, the younger afflicted with rheumatic heart disease. A few years later the father suddenly deserted the sick mother. Financially contributing brothers married or died with pneumonia.

Through all this emotional and situational turmoil patient was able to arrange her life rather satisfactorily; she remained orderly, neat, energetic and usually cheerful. She has always had a strong sense of responsibility. Her attachment to her mother appears excessive. If patient had any anancastic tendencies prior to her illness, they were very minimal and were probably assets rather than liabilities. She seemed to remain stable in the face of real difficulties. It was only when illness overtook her that her anancasms came into full expression. With physical improvement her anancasms receded.

Case III.—Mr. C. F.: 51 years old. Mr. F died in February 1939 with lobar pneumonia. From November 1937 to April 1938, patient was studied and treated in the psychiatric out-patient clinic of the New Haven Hospital. He presented symptoms typical of endogenous depression which had become progressively more prominent and disturbing in the course of 1½ years. With the development of the cardinal features of depression, patient noticed that he was acquiring a number of "nervous habits." These might be divided thus: (1) Repetitious movements, repeated in 4 or multiples of 4, such as drumming on table with fingers, teeth-picking, foot-wiggling. (2) Repe-

titious movements, repeated any number of times, to initiate some purposefull performance, *e.g.*, lifting glass repeatedly to mouth before drinking, marching around table before kissing wife, checking up on doors before going out, stair-counting. (3) Certain bizarre rituals to offset fear "that something terrible but ill-defined might happen" if certain mannerisms were neglected, *e.g.*, walking over holes in the streets in a certain way, walking in a circle around every tree on the street. (4) Frequent obsessive thoughts that he might kill his wife or child. Patient interpreted these anancasms as precursors of insanity—stressed this intense fear rather than harping on other depressive features.

Most of the members of this old Rhode Island family were considered stable. The patient's father went through at least one depression during which he made three suicidal attempts; the patient's mother was considered somewhat nervous and meticulous.

As a child the patient was serious, very conscientious, already given to "having to get at the bottom of things." He interrupted his formal high school education to help meet financial reverses. Patient became a draftsman, machine designer and amateur mathematician. Here his love and need for precision, his constant effort to avoid even the least error, his conscientious and meticulous way stood him in good stead. With the economic depression he lost his job. Finally he became dependent on W.P.A. where once more his particular anancasms made him a valued member among specialized technical workers.

Patient never felt completely at ease with other people. He married for a home, not for love. Emotionally he was never very close to his wife; sexual desires were perfunctorily satisfied.

Patient seemed critical, ambitendent, sensitive, set in his ways; he was hemmed in by feelings of duty and responsibility. He had marked ambitions for "education" and proceeded systematically to acquire this. His reading was mostly along technical lines. For culture's sake, he read the King James version of the Bible, five chapters every night, followed this with a Catholic version; then the plays of Shakespeare, so many acts in an evening. When he first came to the clinic, he was well through the Encyclopedia Britannica, having started with A, proceeding without skipping even one page.

Until the depressive illness overtook this man, his anancasms were assets in his work and recreation. His wife could adapt with ease to his routines and knew exactly what to expect from her husband. With his illness, his anancastic tendencies not only increased in intensity along the old lines, but began to invade new territories. His heightened compulsions and obsessions now produced so much new bizarre content, were so loaded with feelings of anxiety and fear that they became alarming to him. His wife was at her wit's end how to adjust to her husband. Friends and employers became intolerant of these "habits," and even the children in the neighborhood stopped to point their fingers at that "funny guy."

As the depressive features lifted, the anancasms receded. By April 1938 he was discharged very much improved. By September 1938 he reported

that his "mental condition had almost entirely gone." He was again the anancast of predepression days, experiencing his mannerisms once more as assets rather than as liabilities.

It is hoped that these histories have not only illustrated a few circumstances and settings in which anancasms are observed, but have also given a sampling of types of people who need and use and experience with anancasms. There is no one "anancastic type." People of very varied physique, of differing impulse, temperament and intelligence, may have anancastic tendencies. The anxieties and tensions of the anancast are usually present early in life and seem to be intimately linked with conflicts between dependence and independence, ambitendence, overt or covert hostility. These people seem to want an inordinate amount of affection, approval, prestige, solicitude. They are critical, rebellious, unyielding, defiant of the very sources that might give them at least a measure of the understanding and approval they seek. These people seem almost afraid or are unable to make attachments in which give and take is expected. They are usually more inclined to hate. Some take such one-sided, demanding, domineering, octopus-like possession of another person that they provoke counter-hostility of no mean magnitude. Often they are contemptuously tolerated as oddities in interpersonal relations. Anancasts usually do things with intensity; they are given to an "either-or" attitude. It has been repeatedly shown that anancasts have little insight into their personality organization. They seem unable to face themselves, they fear to admit those very personality traits through which they try to adjust. They make peculiarly inaccurate self-evaluations. These people frequently pride themselves on their anancasms. They magnify as virtues their compulsions, their primness, their conventionality or their super-methodical approach to all manner of problems. They often are most self-righteous. They are usually sensitive. They are readily hurt by the least disapproval or criticism, in spite of protection of their feelings, supposedly achieved through anancasms. Other peoples' preference for another way of living, another ethical code, another set of values seems to hurt or offend. Insecurity, mostly a fear of themselves, makes anancasts choose very set, well-defined, approved systems of values, codes, religions. They guard jealously what they have; only with the greatest reluctance will they finally submit to minor modifications. This is rigid-

ity. It would seem that the more rigid they are, the more anancasms they need to maintain the *status quo*, and the more anancasms they practice, the more rigid they tend to become.

In conclusion, we should like to stress once more the way and the diversity of content of anancasms, their constructive or non-constructive nature, the varied settings in which they may thrive, the personality traits most commonly observed. Anancasms are not isolated phenomena; for certain persons they are the ways and means of experiencing; they, too, make their bid for cultural acceptance, tolerance or disapproval.

Comment.

DR. GEORGE ALDER BLUMER.

Eight years ago the JOURNAL published from the pen of one of the Editors Emeriti, Dr. Edward N. Brush, some biographical notes and personal impressions of another Editor Emeritus, his lifelong friend Dr. G. Alder Blumer. Today it becomes our sad duty to record the passing of Dr. Blumer, who for a number of years had been the surviving Editor Emeritus. Dr. Blumer died after a brief illness on the morning of April 25 at his home at Providence, R. I.

Although for several years Dr. Blumer had not attended the annual meetings of the Association he retained always the keenest interest in its doings, and from time to time up to the last year would bring to the attention of the Editor items of news or other matters which he felt might be of interest to readers of the JOURNAL. A memorial of Dr. Blumer will be published in a forthcoming issue.

A gentleman of the old school whose urbanity and charm made an indelible impression upon those who were associated with him, Dr. Blumer was one of the outstanding pioneers who developed modern hospital methods for the treatment of the mentally ill and inaugurated the new era in American psychiatry. His ideals were of the highest and his wholesome influence has survived in the career of the JOURNAL during the years since he laid down his editorial pen.

We salute his memory with profound respect and affection.

DR. EDWARD MAPOTHER.

We regret to record the death on March 20, 1940, of Dr. Edward Mapother, professor of psychiatry in the University of London, and medical superintendent of the Maudsley Hospital.

This hospital was opened by the Ministry of Pensions in 1919 as a treatment center for war neuroses with Dr. Mapother as medical superintendent. This was an interim arrangement. The

hospital had been endowed by Henry Maudsley as an institution for treatment, teaching and research in psychiatry, and when it was opened by the London County Council in 1923 to carry out the provisions set forth by the founder, Dr. Mapother continued as medical superintendent and retained this position until his death.

To the organization and development of this great institution and the expansion of its work Dr. Mapother gave his best energies and his best years, with the result that his name is identified with Maudsley Hospital as the great English teaching and research center, in the minds of a long procession of physicians and students on both sides of the Atlantic who at one time or another worked under his guidance. His sound clinical procedure, evidential thoroughness and balanced judgment supplied a wholesome and stablizing influence amongst the diverse winds which blow across the psychiatric arena.

Maudsley Hospital was still in the process of development when Dr. Mapother was cut off at the early age of 58, his strength overtaxed by his devotion to duty. Only last year a new outpatient division, children's department and private wards had been added to the growing institution.

His memory will survive in the traditions of the Maudsley and in the impetus and direction he gave to psychiatric education and research.

News and Notes.

DR. ADOLF MEYER NEW PRESIDENT OF THE NATIONAL COMMITTEE FOR MENTAL HYGIENE.—The Board of Directors of the National Committee for Mental Hygiene announce the election of Dr. Adolf Meyer of Baltimore as president of the National Committee, to succeed Dr. Arthur H. Ruggles of Providence, R. I.

Dr. Meyer is professor of psychiatry of Johns Hopkins University and director of Henry Phipps Psychiatric Clinic, Baltimore, which he founded in 1913. Often referred to as the dean of American psychiatry, Dr. Meyer has long been the acknowledged leader of his profession and has exerted a wide and deep influence on the growth and development of psychiatric thought and practice both in this country and abroad. His former pupils occupy leading posts as teachers and practitioners of psychiatry throughout the United States and in many foreign countries. He has been president of The American Psychiatric Association, American Neurological Association and the American Psychopathological Association. He is a member of numerous scientific societies and has received academic honors from various universities here and abroad.

Dr. Meyer collaborated with Clifford Beers in the founding of The National Committee for Mental Hygiene in 1909, and gave the mental hygiene movement its name. He is chairman of the National Committee's Advisory Committee on Psychiatric Education, and was one of the organizers of the American Board of Psychiatry and Neurology, established in 1934. He was the first Salmon Memorial lecturer in psychiatry and mental hygiene at the New York Academy of Medicine.

MENTAL HOSPITAL SURVEY COMMITTEE TAKEN OVER BY U. S. PUBLIC HEALTH SERVICE.—On July 1, 1939, the United States Public Health Service took over the work of the Mental Hospital Survey Committee and incorporated it in a new Section on Mental

Health Methods of its Division of Mental Hygiene. The Mental Hospital Survey Committee, organized in 1936, had as its main purpose the survey of mental hospitals throughout the United States and Canada. It was made up of representatives of The American Psychiatric Association, the American Neurological Association, the American Medical Association, the American Board of Psychiatry and Neurology, the National Committee for Mental Hygiene, the United States Public Health Service, the Canadian National Committee for Mental Hygiene and the Canadian Medical Association.

It became apparent early in 1939 that the Committee could not continue its activities as originally planned because of the withdrawal of financial support from private sources. After reviewing its work the Committee strongly recommended that it be continued and it was decided to ask the Public Health Service to assume responsibility for it. The Surgeon General, in response to the request to do this, said:

> . . . . The Service feels with the Committee that this work is not completed and that it should go on, with the end in view of discovering the defects as well as the good points in the various hospital systems, so that by the application of the information gained the standards of care and treatment of the mentally ill, as well as the studies of mental diseases in various jurisdictions may be raised to a level commensurate with the importance of this problem.
>
> Sensing the need, the Public Health Service will take this work over and carry it on to the extent made possible by appropriations, and in doing so we will be pleased to have the continued cooperation and assistance of the groups and individuals who have done so much to make the work of the Committee a success in the past.

Dr. Samuel W. Hamilton, medical director of the Mental Hospital Survey Committee, accepted an invitation to go with the Public Health Service and the old Committee agreed to function in an advisory capacity as representatives of the various organizations of which it was originally formed.

NEW PERIODICAL IN PSYCHIATRY AND NEUROLOGY.—In January, 1940, appeared the first issue of the new journal *Diseases of the Nervous System*. This monthly periodical, which is under the editorial direction of Dr. Titus H. Harris of Galveston, Texas,

sets for itself the purpose of bringing the problems of psychiatry and neurology before the physician in the general practice of medicine to whom it is addressed rather than primarily to the specialist. For the most part the articles are brief and readable, and each issue comprises about 30 pages.

The editorial staff includes Dr. Harris as editor-in-chief, and a board of 22 associate editors representing different parts of the country. The editorial office is in the University of Texas School of Medicine, 800 Avenue B., Galveston, Texas. Mr. Leonard Kornfeld is business manager, the publication office being the Physicians Postgraduate Press, Alamogordo Rd. (P. O. Box 1069), El Paso, Texas.

THE HOGG FOUNDATION FOR MENTAL HYGIENE IN TEXAS.—President Homer Price Rainey of the University of Texas announced in February that a grant of $2,500,000 had been made by the Will C. Hogg estate, one object of which is the furtherance of mental hygiene in Texas. A tentative program for the use of the grant was outlined in January at a meeting of an advisory committee composed of Dr. Adolf Meyer, Dr. George Stevenson and Mr. Daniel Prescott, and assisted by state leaders in mental hygiene.

Objectives agreed upon by the committee include (1) the formulation of a program essentially educational; (2) the training in the principles of mental hygiene of selected groups who would form a personnel in selected educational institutions for the dessimination of facts related to mental health; (3) the organization of a group of social service workers trained in the promotion and maintenance of mental health to be sent into selected communities over the state for lecture and case work; (4) the holding of postgraduate clinics for mental hygiene workers, together with the establishment of traveling clinics to provide in-training service for this group; (5) the establishment of a center for investigation and research in mental hygiene among professional groups.

NEW TREATMENT BUILDINGS AT COLORADO STATE HOSPITAL.—Announcement is made of the completion and opening at the Colo-

rado State Hospital of a special hydrotherapy building and the rebuilding of the isolation unit for patients suffering from contagious diseases. These improvements have been made possible as a part of the state-WPA two million dollar expansion program. In the hydrotherapy building which provides twenty-four hour service the most modern construction, heating and lighting methods have been utilized. The rooms are sound-proof and air-conditioned and the building is fire-proof. Day and observation rooms and an adjoining kitchen service are provided for patients undergoing continuous hydrotherapy or treatment in the isolation section.

First National Italian Congress on Mental Hygiene.—On the occasion of the World's Fair to be held in Rome in 1942 the Executive of the Italian League of Mental Hygiene and Prophylaxis proposes to hold the first National Congress on Mental Hygiene in Italy.

The Congress, which it is anticipated will be convened under the presidency of Professor Eugenio Medea, will mark the completion of the first decade of the Italian League. A three day Congress is proposed with program to be announced later.

Southern Psychiatric Association Yearbook.—In connection with the fifth annual meeting of the Southern Psychiatric Association held in 1939 at Louisville, Kentucky, an innovation was introduced in initiating a Yearbook wherein are printed either in full or in abstract the outstanding papers of the scientific sessions. This first issue of the Yearbook is dedicated to Dr. Newdigate Moreland Owensby, secretary and founder of the Southern Psychiatric Association.

Medicine and Public Health at the New York World's Fair.—One of the most fascinating and strikingly informative exhibits of "The World of Tomorrow," which was the New York World's Fair of 1939, was that of modern medicine housed in the Medicine and Public Health Building on Constitution Mall.

This exhibit will be continued and enlarged in the New York World's Fair of 1940 which is dedicated to Peace and Freedom.

The theme center of this exhibit is the Hall of Man. Here visitors are first startled by a realistic reproduction of the greatly amplified systole and diastole of the human heart-beat which emanates from the Transparent Man. This giant primate, twenty-two feet high, speaks for himself, and by means of lights synchronized with the lecture demonstrates the essentials of human anatomy and physiology. For those who love the macabre a moving talking skeleton describes himself and the bones of which he is composed.

With amazing cleverness most of the major things which man knows about himself are presented to the visitor pictorially or by mechanical devices and demonstrated with objective clearness far beyond the possibility of any text.

The catalogue of the medical exhibits is a long one and includes the most recent developments in the healing art, and in preventive medicine and public health. Here one can visualize the story of the endocrine glands, of allergy, of the treatment of cancer, of the campaign against tuberculosis, of the action of the heart and the fluctuation of blood pressure, of the phenomenal effects of insulin and sulfapyridine, of the virus diseases, of the protean mischief of Spirochaeta Pallida, of the most modern methods for the control and prevention of disease.

The splendid medical exhibit has been made possible through the cooperation of the American Museum of Health Inc., the Carnegie Corporation of New York, the Oberlaender Trust, several prominent insurance companies, the American Medical Association, numerous pharmaceutical houses and makers of food products and various medical and public health organizations and social agencies.

Book Reviews.

INTELLIGENCE AND CRIME. By *Simon H. Tulchin.* (Chicago: University of Chicago Press (planograph) 1939.)

Much has already been written concerning the intelligence of convicted offenders, and unfortunately much of it has been found to be unreliable. We have been assured that as many as 95 per cent of prisoners were feebleminded, and with equal solemnity have been told by other students that prisoners, far from being defective, were of approximately average intellect and indeed brighter than the guards! It is a welcome relief, therefore, to find an objective study by a competent psychologist which deals with over 10,000 prisoners, representing *all* the inmates of three Illinois penal institutions over a period of 7 years (1920-1927). (The data find further corroboration in the study of 13,454 male prisoners admitted during the period 1930-1936, as reported by Brown and Hartman, *Journ. Crim. Law* 28:707; No. 5, Jan. 1938).

In order that data comparable with the Army draft findings might be secured, the Army Alpha and Beta tests were employed, supplemented by the short Army Performance and the Stanford Binet tests. About 20 per cent of the men were given individual examinations. Data were also available in the prison records on such topics as race, offense for which sentenced, number of previous convictions, age, height and weight, education, marital status, employment status, religion, and birthplace of prisoner and parents. The findings for the penitentiary (5,512 cases) and reformatory (4,748 cases) are given separately, for obvious reasons. There is also a short chapter on the 153 inmates of the women's prison.

The general agreement of the figures, especially of the penitentiary, with the Army draft figures for Illinois, is rather striking (the inferiors are fewer in the reformatory largely because of a higher proportion of native-born whites). Whether or not the Army draft was a fair cross section of the population, it was the largest group ever studied psychometrically, and certainly significant. For 25.9 per cent inferiors in the draft the penitentiary shows 23.4 per cent; superiors are respectively 10.6 per cent and 11.8 per cent.

The fact that crime is not a unitary phenomenon, but that it is influenced by many factors, including nativity, intelligence, race and age, is made abundantly clear by Tulchin's data.

"The men in the several nativity and race groups show marked percentage variation in the type of crime for which they are serving sentence. Native whites show more fraud cases than are found among the foreign-born and Negroes. In the penitentiary the smallest percentages of robbery,

larceny and burglary cases are found among the foreign-born, while in the reformatory the smallest percentage of robbery cases is found among the native-born whites, the smallest percentage of larceny cases among the Negroes, and the smallest percentage of burglary cases is again found among the foreign-born.

"In both institutions the foreign-born and the Negroes show nearly three times as many men committed for murder as do the native-born groups, with the percentage among the Southern-born Negroes higher than among the Northern-born Negroes. There is a greater proportion of sex crimes cases among the foreign-born than among the native whites, with the lowest percentages found among the Negroes."

In general the highest scores are found in men convicted of fraud, and the lowest in those guilty of sex crimes; however, with only one exception "individuals of all grades of intelligence are found in all six crime groups" (no Southern-born Negro of superior intelligence is found in the sex crime group).

The volume is a significant contribution to the fields of psychiatry and criminology, and should be read by all who are interested in securing reliable information on a subject which has hitherto been dealt with rather impressionistically. Dr. Tulchin has thrown a bright light into some dark recesses and all students of the crime problem are in his debt.

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PSYCHOBIOLOGY AND PSYCHIATRY. A TEXTBOOK OF NORMAL AND ABNORMAL HUMAN BEHAVIOR. By *Wendell Muncie, M.D.* (St. Louis: C. V. Mosby Company, 1939.)

After a preliminary discussion of the Mind-Body problem and of the history of psychiatric thought, Muncie defines psychobiology as follows:

"Man emerges as a special and a higher biological product, including within him all the data of physics and chemistry and zoology, but distinguished by the greater development of delayed reflexes as adaptive reactions, by the vast development and use of symbols, especially those of language, and by the hanging together of all these sensory, motor, and associative performances in a special manner called 'conscious.'

"In such a system the old contrast of mental and physical with its prejudicial barriers and insolubilia is replaced by consideration of degrees or levels of integration: Are the facts understood as non-mental, that is, as the qualities and activities of essentially detachable parts, organs, or systems (as treated in anatomy and physiology), or are they only intelligible in terms of mentally integrated performances—of a sort indissolubly a function, overt or implicit, of the total organism and having *meaning* as adaptive reactions in a total situation?

"Psychobiology then is the study of those functions distinctively human, the things man is best known for, the mentally integrated performances. The study demands knowledge of the physical sciences and of anatomy and

physiology, but these in no wise explain the phenomena under observation. Their explanation must be in terms appropriate to the complexity of their level of integration: biological, but of a type operating with more or less consciousness, a hanging together in a flow with symbolization."

It thus appears that the essential and underlying quality which gives especial value to psychobiology is *meaning* or sense and direction, which becomes clear when viewed in the light of the personal biographical record. It is therefore clear that psychobiology and psychiatry are not in a complete sense synonymous, since psychiatry is quite as concerned with the human functions for which no definite purpose or meaning can be found. The psychiatrist must, first of all, be a physician and be in constant touch with those "detachable" organs for which meaning cannot be found in any personal sense. As a neurologist he must be familiar with those automatic and reflex activities which are only vaguely part of personality development, and as a psychiatrist he is definitely interested in disease processes, such as those of neurosyphilis, which are again devoid of any interpretation in terms of meaning or even in terms of the biography of the individual since general paresis, for example, may be entirely accidental in the sense that the acquirement of syphilis may bear not the slightest relationship to the total personality of the individual. Psychiatry is just as much interested in the vitamin deficiency diseases of the nervous system as it is in the neuroses, although the vitamin deficiency diseases may be, at least for the time, adequately explained on the basis of physical, chemical, or physiological processes, whereas the neuroses at the present time may not be so explained.

I think that psychobiology has much less importance than it merits when thus restricted by the author of this book. Too zealous a search for meaning definitely leads to vague theorizing and mere ingenuity rather than to an assemblage of facts. Too much emphasis on meaning is against the best interests of science, since adventure into meaning usually has all the pleasures and disabilities of armchair philosophy.

Any emphasis on the total individual and insistence on the value of intensive biography, as well as a consideration of the personality from the standpoint of his social setting, are necessary parts of psychiatric technique and productive of great good. The consideration of the individual as a whole has great value, and it is to the credit of Adolf Meyer that this has now become a commonplace in psychiatry, and it is his most significant contribution that the study of the total individual is a *fait accompli*. Man, as a purposive striving creature, obsessed by his symbols and enmeshed by the results of the tools of his own creation, is as real, although not so measurable, as the biological man, living in a reciprocal relation with all the physical and chemical forces of the universe.

At the same time, the injunction to study the total individual may degenerate into a counsel of perfection, since it is really impossible to do a good job if one becomes too literal an accepter of this commandment. A study of the hormones and of the physical reactions of any patient in a hospital for mental disease might well occupy a dozen people for months,

and the accumulation and evaluation of all the facts in the social history would be even more arduous and time-consuming. More is learned by the lumbar puncture needle and a few chemicals in general paresis than by the most elaborate personality study, and it is a sad commentary on our efforts to find a rational direction for therapeutics that the only treatment that really does much good in the depressive states is that non-rational approach "shock," where no study of the whole individual is at all essential. We must not, it seems to me, make a fetish of even the best of slogans, such as "study of the whole individual." It is just as well that there are people who devote themselves to narrow and very specific studies of isolated functions and reactions. What is lost in breadth is gained in factual aquirement.

For one, I see nothing gained in unifying under the heading "holergasia" the major syndromes, such as depression, excitement, delusional and hallucinatory developments, confusion, stupor, deterioration or dementia. Nor do I see any point whatever in using the term "dysergasia" to represent a wide range of conditions of toxic, infectious, metabolic, circulatory and traumatic origin, in which it is stated that the symptoms are "directly due to disorder of the brain through poor support of its functioning from toxic, infectious (with fever) metabolic, and traumatic causes, *i. e.*, from extrinsic factors acting on the brain." (Just what the word support means in this connection is utterly beyond the comprehension of the reviewer.) For all we know, schizophrenia may be dysergasia, if by this is meant that a brain function is organically disturbed. Nor can much be said for the term "anergasia" to designate such diverse processes as cerebral arteriosclerosis, brain tumor and Pick's disease. And it remains a mystery to me why the substitution of such words as "merergasia" or "kakergasia" are believed to throw any light on the conditions over-unified by the term psychoneuroses. What psychiatry needs is not new names but new facts, new procedures and new and fruitful hypotheses.

Despite all these criticisms, the book is heartily recommended to the reader. It has historical significance in that it puts together a point of view which hitherto has been vaguely scattered in many diverse papers and journals. It has very worth-while clinical features. The section entitled "Student's Personality Study" is an excellent presentation of personality analysis and recording. The historical summary at the end should be part of the required reading and training of every psychiatrist, since the history of any discipline lays the foundation for critical evaluation of new work.

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THE MECHANISM OF THOUGHT, IMAGERY, AND HALLUCINATION. By *Joshua Rosett*. (New York: Columbia University Press, 1939.)

The author has achieved a much needed synthesis of many heretofore isolated or poorly related neuro-psychological phenomena such as states of attention, thought, imagery, sleep, startle, dreams, hallucinations and the epileptic seizure. He draws on a wealth of clinical and experimental data

collected by himself as well as others. By an integration of these data a general principle or neural mechanism is disclosed which is shown to be the common denominator in all of the above mentioned neuro-psychological phenomena.

This basic mechanism is used throughout the book to show the correspondence of neural events to series of psychological states. Thus, a state of strong concentration with a fixed and restricted field of attention is shown to be associated with an inhibition or reduction in function of the greater part of the cerebral cortex. This condition amounts to a partial functional decerebration and serves to release the essential activity of the succeeding links in the nerve-chain. In the case of the muscular phenomena of attention, for example, the outward evidence of the operation of this principal is seen in the enhanced subcortical motor activity manifested in the increased postural tone manifested by a state of "at-tension."

Fatigue, toxins and other injurious agencies may produce different degrees of disintegration of the organization of the cerebral nerve conduction circuits. The wave of disintegration may advance for a greater or lesser distance along the nerve pathway, but only in the direction of transmission of normal nerve impulses. Each of the successive links in the affector-effector nerve-chain is involved in turn. Thus, an injury or a functional suppression of one link first releases for hyperactivity and then suppresses the function of the next link in the circuit, etc. As an example of this, the author describes the normal suspension of sensory reception during sleep or its abnormal suspension as the result of an injury causing partial blindness or deafness. In either case, the essential activity of the association nerve systems which form the next link in the affector-effector conduction chain is released. Since their hyperfunction is not guided by the activity of the sensory receptors which keep the person "in touch" with the reality of the outer world, it is characterized by fantastic imaginings, dreams or hallucinations.

Following the greater or less disintegration of cerebral nerve function, recovery or reintegration takes place, link by link, in the reverse, or *effector-affector*, order. This is characteristically seen in the step by step recovery from an epileptic seizure as well as in the stages of awakening from normal sleep.

Finally, this orderly sequential pattern of neuro-psychological events associated with disintegration and reintegration of the nerve conduction circuit, is shown, in a number of its details, to follow Hughlings Jackson's law of evolution and dissolution of the nervous system, but to deviate from it in its main features.

This work is a pioneering contribution and will stand or fall on the validity and fundamental importance of the central theme as briefly outlined above, rather than on the presence of any minor issues of a possibly controversial nature which are not essential for the main thesis.

The author uses a minimum of technical terms for such a work, and enhances interest and clarifies his argument by a wealth of examples and illustrations. The book should be of value not only to neurologists, psy-

chiatrists, psychologists and educators, but also to those educated laymen who are interested in the unification and correlation of mental phenomena with neural structure and function.

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THE CONCEPTUAL REPRESENTATION AND THE MEASUREMENT OF PSYCHOLOGICAL FORCES. Contributions to Psychological Theory. Vol. 1, No. 4, Serial No. 4. By *Kurt Lewin*. (Durham, N. C.: Duke University Press, 1938.)

It seems to be most appropriate to reprint the author's summary which gives a sufficient idea of his aims.

"We have attempted to describe the position of the concept force in psychology and to discuss major methods of measuring psychological forces. One of our objectives is to bring into the open and to apply scientific standards to those concepts which actually underlie certain recognized research methods and to eliminate those unnecessary accessories which create much discussion but little progress.

"One of the outstanding properties of force is its directedness. Direction in psychology cannot be defined as physical direction and cannot be determined by Euclidian geometry. A geometry applicable in psychology is that of hodological space. The geometrical properties of this space are described, and examples of its application in determining directions and distances in the life space are offered.

"The conceptual properties of the construct force are given and its coordinating definition to observable processes. The conceptual and dynamic relation between psychological forces, valences, and tension are discussed and certain basic theories concerning the relation between need, environment, and the 'mechanics' of locomotion.

"Various methods of measuring forces and valences are surveyed, especially those related to opposing forces and to velocity of restlessness, consumption, translocation, and learning. The problem of the structure of the force field and of overlapping force fields is discussed, including several choice and conflict situation with stable and labile equilibria. We have limited our discussion mainly to relatively simple problems of driving and restraining forces.

"One of the main objectives of both the geometrical and dynamical concepts is to develop a frame of reference useful as much in social as in quasi-physical situations. An adequate treatment of social problems, especially social conflicts, however, makes certain distinctions necessary, particularly that between 'own' and 'foreign' forces, which we have merely mentioned."

An attempt to improve our methods of dealing with psychological problems should always be considered with great interest. That the concepts are of help in grasping the fundamentals of psychological experience cannot be doubted. In studying his book one often had the feeling that a conceptual

framework of this type will remain barren if it avoids the more concrete situations of the life of an organism. It might be doubted, for instance, whether the psychological space can be defined without giving specific attention to the problem of the human body and our attitudes toward it. However, it might be necessary to approach the problems at first in a schematic way and we may hope that the further elaboration of the measurement of psychological forces will yield a conceptual apparatus in which the specific human problems will find an expression. One would also like to know what the relation of direction in psychology is to the direction in the physical sense, and one would also like to know more about the relation of the hodological space to the space of physiology and physics.

However, I consider Lewin's efforts as of fundamental importance.

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MENTAL HEALTH THROUGH EDUCATION. By *W. Carson Ryan.* (New York: The Commonwealth Fund, 1938.)

This book contains the results of a survey of educational practices in the United States "in respect to what has been called, for want of a better name, mental hygiene." The survey was made during 1935-1936 and was financed by the Commonwealth Fund of New York. Dr. Ryan, who is on the staff of the Carnegie Foundation for the Advancement of Teaching, president of the Progressive Education Association and an educator of wide experience, was enabled to visit many schools and clinics in order to see at first hand what is being done in the way of mental hygiene in nursery schools, elementary and secondary schools, universities and teacher training institutions in many parts of the country.

More than a summary of his findings, this book is a statement of much that is desirable from a mental health standpoint in teacher selection and training, classroom practices, curriculum school administration, special services and the school's relations to the family and the community. The view of education in these pages is familiar to persons interested in progressive education. In this view education and mental hygiene have much in common. The author admits that there were "good mental attitudes in school and elsewhere long before any science of human behavior or mental hygiene movement had developed." His plea is for a definite attempt to apply scientific knowledge in the field of mental health to the educational practices. The objection to this plea that is most commonly met with in educators is that teachers would have to be psychologists or psychiatrists to do this. As the author points out, "teachers can be of most service not by trying to be specialists in this field in any highly technical sense," for it is not the exceptional child that is being aimed at, "but by having some insight into a few fundamental principles." It is decidedly heartening to find an author giving concrete suggestions as to the changes such an attempt would involve. Many of these suggestions are familiar, but their relationship to the mental health of teacher and pupil is presented here with exceptional clarity.

The teacher's attention should be directed more to developing serviceable habits and attitudes in every child and to the fact that this is accomplished during the acquisition of the usual skills and knowledge. Affection, a sense of security and a sense of accomplishment are needs of every child that must be met both in the school setting and outside of it. Two outstanding factors making for more effective work by the teacher are the appreciation of the fact that each child is an individual in rate of progress and interests, and that all behavior has causal factors back of it. Examinations, pupils' records, "discipline," the curriculum are discussed in this book always in relation to the individual pupil's health and habits, and progressive practices are described.

Appropriately enough, one-fourth of the book is devoted to the teacher's personality and training. Little has been done in practice about more satisfactory selection of prospective teachers although there has been much discussion concerning it. Scholastic standing as the basis for selection is rejected by Dr. Ryan as even less satisfactory than indiscriminate admission, as "the proportion of students academically selected tends to bring out candidates with a high percentage of personality difficulties." The existing personality tests are of little help in this field. In the recent general increase in the teacher's training period, only a few colleges have seized the opportunity to encourage those students who showed interest and ability in understanding and managing children. Such students have been given the opportunity for close contact with children. A system suggestive of apprenticeship is being tried out in one state, in which the student serves as assistant to a successful teacher and participates in the teaching. The book contains very useful descriptions in more or less detail, of a number of attempts to introduce mental hygiene into teacher training during both preservice and in-service years.

The chapter on "Special Services for the School Child" will be of much interest to visiting teachers and to psychological, psychiatric and social workers who function in more or less close cooperation with schools. It should help these workers to see their work in relation to the whole field. It will serve as a stimulus to many who have not conceived their function in as broad terms as it is here stated.

There is little to quarrel with in the book. Much of the philosophy in it is familiar. The results of the surveys and the clear statements of the relationship of the suggested practices to mental hygiene are useful and stimulating. The book can be commended to educators as putting a challenge that has been accepted by increasing numbers of teachers in the past ten years, and that will be faced by the teachers generally in the next decade.

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Explorations in Personality. By *H. A. Murray, M. D., Ph. D.* (New York: Oxford University Press, 1938.)

This volume is both interesting and challenging. It describes a three-year study of 51 men of college age (unemployed and college students) by 28

physicians, psychoanalysts and psychologists associated with the Harvard Psychological Clinic. All experimenters studied the same individuals and reported their findings in periodic assemblies and discussions. Observations were directed to emotional and behavioral reactions under a variety of partially-controlled conditions; but the conditions resembled those of everyday life rather than the artifacts of traditional laboratory experimentation. The outcome at the present time is a description of a proposed theory of personality, and of experimental techniques for systematic study of personal behaviour—both theory and method of investigation suggesting a broad eclecticism that should (in the opinions of these scientists) reconcile and stimulate the ideation of workers with widely divergent points of view. Most of the reporting is done by Dr. Murray, but specific procedures are described by some of the collaborators.

Basic to the theory of personality is the concept of *need*. This concept goes beyond the simple S-R formula of behaviourism, or the cause-effect relationship of mechanistic physics and includes the typical biological and psychological drives, with a still more significant addition. Korzybski's view of man as a *time-binding* organism is accepted, even though its interpretation is less clear than one could desire, and appears to be lost sight of at many points in the report. The survey of human needs is extensive, and a few novel and expressive concepts are introduced. There is no very systematic arrangement in the presentation, and relatively little attention to the dynamic interrelationships among the needs. Developmental concepts are implied, but not clarified; and no definite criterion of desirability of needs is furnished.

The notes on psychological techniques employed are interesting, although the reader will not find much concrete reporting as to experimental results and conclusions. The most significant feature of this section of the book would seem to be the impressive degree of freedom in investigation that the enterprise encouraged. No one particular or stereotyped approach was emphasized. The studies obviously challenged the greatest versatility in attack and subtlety of qualitative insight in interpretation. How successful the outcomes were, however, we are unable to judge.

From one point of view, the project may appear to be a mere elaboration of the usual clinical procedure, where a group of investigators examine a patient independently and discuss their findings in conference. Instead of being unduly concerned, however, with recommendations on therapeutic procedures in connection with the subject examined, as is usually the case in clinical work, the object of this project was apparently quite otherwise, although it is difficult to see how that object can be defined more clearly than in terms of general understanding of human personality. In a sense, this may be said to be a weakness of the project. One has the feeling that Dr. Murray's statement of a theory of personality is a common denominator to which all the workers would, in general, subscribe; but beyond that, each specialist would go to work from his own particular point of view. In another sense, however, it is possibly a strong point; for it creates a

situation with great opportunities for following original insights and clarifying some challenging leads. Subsequent reports of the project or its continuation may indicate more clearly the degree to which it has justified its advantages as an investigatory setting. At present one feels that, for instance, the elaborate description of the "Case of Earnst" involved a very detailed enquiry into all sorts of experiences and events pertaining to the life of this individual, but that the case study as reported was decidedly lacking in purpose and bewildering in outcome.

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Man Against Himself. By *Karl A. Menninger.* (New York: Harcourt, Brace & Co., 1938.)

The author of the *The Human Mind* has followed up that popular success by another book dealing with what appears at first sight to be a much narrower field, the subject of suicide. Actually the treatment is not nearly so narrow as might be expected, as under the general head of suicide, complete or partial, he is able to include discussions of such subjects as alcohol-addiction, anti-social behaviour, self mutilations, asceticism, chronic invalidism and other such conditions. The book makes fascinating reading, taking as it does its illustrative material from history, clinical studies, newspaper reports and contemporary literature. The general thesis is the Freudian one:

"And so our final conclusion must be that a consideration of war and crime, no less than of sickness and suicide, leads us back to a reiteration and reaffirmation of the hypothesis of Freud, that man is a creature dominated by an instinct in the direction of death, but blessed with an opposing instinct which battles heroically with varying success against its ultimate conqueror." (p. 470)

More specifically concerned with suicide, the following statement defines Dr. Menninger's position very concisely:

"The theory of suicide is that the wish to kill, unexpectedly robbed of certain external occasions or objects of unconscious gratification, may be turned back upon the person of the 'wisher' and carried into effect as suicide." (p. 32)

To the establishing of this proposition the author brings a wealth of supporting material and a very convincing method of presentation. But in this case, as in so much of Freudian literature, the usual doubts arise. He will readily convince the already converted, he will almost certainly fail to convince those scientists who do not think within the framework of Freudian theory. This happens so often that one is forced to wonder why non-Freudians find so unsatisfactory all these elaborate demonstrations of the essential truth of the master's dogmas. To a social anthropologist the reason seems to reside in the type of language that Freudians habitually adopt in explaining their theory. Dr. Menninger's final explanation of suicide, expressed in the first of the above quotations, proves on close in-

spection to be of an anthropomorphic nature. Even if we know what instincts are, to say that two instincts "battle heroically" with each other, is about as good an argument as saying for example, that a woman's eyes are like stars, or that the sun battles its way through the clouds. Such metaphors have their place no doubt, in a volume of poems or in elementary school textbooks, but hardly in the scientific laboratory. John Stuart Mill pointed out years ago that "there is a spontaneous tendency of the intellect to account to itself for all cases of causation by attributing them to the intentional acts of voluntary agents like itself. The strength of this tendency does not lie in argument but in an obstinate tendency of the infancy of the human mind." Instincts which "battle heroically" with each other are not very different from "voluntary agents like ourselves," especially when those instincts become, as they readily become in Dr. Menninger's writing, "imps" that "lurk" half-hidden in the human mind. The religions of primitive people are full of similar imps. The whole medieval theory of spirit-possession was based upon unpredictable voluntary agents, more or less personalised, which resided "inside" the personality somewhere, somehow, and capriciously caused the individual to commit suicide, become paralysed, become a chronic invalid, take to drink, etc. Catholic children even in the present day are taught that good acts and bad acts are due to good and bad angels who "lurk" on the right and left shoulders and whisper good and bad suggestions into the right and left ears of individuals. It would seem to a neutral observer that it is because of the similarity between these primitive "explanations" of human behaviour and the Freudian "explanations" that many scientists, used to mechanical and not personal causation, remain unconvinced by, or at least suspicious of, the Freudian system; and the only remedy seems to lie with the Freudians themselves, namely to attempt to express their discoveries in language that is a little less poetical and a little nearer to the language spoken by other sciences.

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Directory of Medical Specialists Certified by American Boards. (1939.) By *Paul Titus, M. D.*, Directing Editor, *J. Stewart Rodman, M. D.*, Associate Editor. (New York: Columbia University Press, 1940.)

This volume of 1572 pages is the first official directory of approximately 14,400 diplomates certified by the American Boards representing the various branches of medicine. It is published under the auspices of the Advisory Board for Medical Specialists, which is composed of representatives of the special American Boards and certain other groups concerned with graduate medical education.

The several Boards are presented in alphabetical order—Anesthesiology, Dermatology and Syphililogy, Internal Medicine, Obstetrics and Gynæcology, Ophthalmology, Orthopædic Surgery, Otolaryngology, Pathology, Pediatrics, Plastic Surgery, Psychiatry and Neurology, Radiology, Surgery, Urology.

Under each Board the organization and examination and certification requirements are set forth together with the list of officers. Then follows the list of diplomates by states, and under each state by cities, both in alphabetical order. A brief biographical outline is included under each name.

In a second section of the book is a complete alphabetical list of diplomates with the address and specialty after each name.

The value of an authoritative reference work of this kind is obvious, and the profession, hospital officials, social agencies, medical societies and libraries and the inquiring public are in the debt of the Advisory Board and the Board of Editors of this Directory.

C. B. F.

In Memoriam.

HENRY JOHNS BERKLEY.

1860-1940.

Dr. Henry Johns Berkley, emeritus clinical professor of psychiatry at Johns Hopkins University, died April 5, 1940, at his home in Baltimore, Md. The cause of death was believed to have been cerebral concussion occasioned by a fall, but there was evidence that he might have had a stroke just previous to the accident. It is of interest that he had been taking vitamin B_1 for the relief of rheumatic pains and had said that he noted certain head and leg symptoms accompanying this treatment.

Dr. Berkley was born July 17, 1860, the son of Edris and Virginia (Enders) Berkley of Virginia. His parents had moved to Baltimore shortly before the outbreak of the Civil War and Dr. Berkley was born in Maryland. He was descended from Sir William Berkeley, Colonial Governor of Virginia.

After preliminary education at private schools Dr. Berkley graduated in medicine in 1881 at the University of Maryland. Somewhat later he spent several years studying abroad, including two years in Vienna. He received the degree of Doctor of Science from the University of Austria in 1904. He returned to Baltimore in 1888 to private practice in nervous and mental diseases and was appointed associate in psychiatry at Johns Hopkins University March 5, 1896; associate in neuropathology April 25, 1896; clinical professor of psychiatry October 1, 1898; and emeritus clinical professor of psychiatry April 30, 1920. He was also visiting physician to Bay View, the City Detention Hospital, for a number of years.

He was the author of *A Treatise on Mental Diseases*, "based upon the lecture course at the Johns Hopkins University, 1899, and designed for the use of practitioners and students of medicine," which was published in 1900, a volume of 602 pages. He also wrote numerous articles, notably one with Richard H. Follis, M. D., "An Investigation into the Merits of Thyroidectomy and

Thyrolecithin in the Treatment of Catatonia" published in THE AMERICAN JOURNAL OF INSANITY, January, 1909 (65: 415-469). This brought him a certain amount of notoriety which was distasteful to him. His research activities were curtailed about 35 years ago when he suffered an enucleation of his left eye, it is said, for melano-sarcoma.

Dr. Berkley's civic interests were centered in the State Board of Mental Hygiene of which he was a member from 1915 to 1930. During the World War he was a member of a medical advisory board. He held memberships in The American Psychiatric Association, The Philadelphia Neurological Society and The Maryland Psychiatric Society.

Dr. Berkley had several avocations and early began to collect engravings of General Washington, finally having over 254, forming the largest private collection in this country. He presented this collection to The Maryland Historical Society as a memorial to his parents. He had joined the Historical Society in 1906 and was an active member until his death. From 1928 to 1935 he was a member of its library committee and council. He contributed a number of papers on historical subjects, some of them relating to colonial medical history. In his younger days he was an enthusiastic mountain climber.

Dr. Berkley was married in 1886 to Ella Linthicum, daughter of Thales A. and Emma (Alexander) Linthicum of Annapolis and Baltimore, who died June 1920. Their only child Margaret Harwood Stockett, died October 12, 1925. His second wife was Miss Mary Miles Jordan of South Carolina who died July 6, 1936. A nephew and two nieces survive him.

W. R. D.

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